

The effects of two weeks high-intensity interval training on fasting glucose, glucose tolerance and insulin resistance in adolescent boys; A pilot study

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Abstract

Background This study examined whether improvements in fasting and postprandial [insulin], [glucose] and aerobic fitness are possible after two weeks of high-intensity interval training (HIIT) in adolescent boys. **Methods** Seven boys (14.3 ± 0.3 y) completed 6 sessions of HIIT over two weeks. Homeostatic model assessment of insulin resistance (HOMA-IR), quantitative insulin sensitivity check index (QUICKI), fasting glucose:insulin ratio (FGIR) and blood [glucose] and [insulin] responses to a Mixed Meal Tolerance Test (MMTT) were assessed before (PRE), and 20 h and 70 h after (POST) the final HIIT session. Maximal oxygen uptake (VO₂ max) was assessed PRE and 70 h POST. **Results** Compared to PRE, two weeks of HIIT had no effect on fasting plasma [glucose] or [insulin] or HOMA-IR at 20 h and 70 h POST. However, a strong negative correlation was observed between PRE training HOMA-IR, QUICKI and FGIR, and change in HOMA-IR, FGIR and QUICKI at 20 h POST ($r = -0.96, 0.969$ and 0.826 for HOMA-IR, QUICKI and FGIR respectively all $P < 0.05$). Plasma [insulin] and [glucose] area under the curve in the postprandial period following the MMTT were unchanged 20 h and 70 h POST compared to PRE. **Conclusion** Two weeks of HIIT did not elicit improvements to fasting or postprandial glucose or insulin health outcomes or aerobic fitness in a group of adolescent boys. Interventions of this type may, however, be effective in adolescents with raised baseline IR.

Background

Insulin resistance (IR), impaired beta cell function (%) and glucose tolerance are all implicated in the development of type two diabetes (T2D) and cardiovascular disease (CVD) [1]. Such risk factors are known to be prevalent in youth [2] and can predict future risk of CVD and T2D [3]. The early development of IR begins 10-20 y before onset of T2D and is thought to be one of the best predictors of future diabetic risk [4]. This makes the pubertal years a prime target for interventions to prevent the onset of T2D and CVD, as well as associated co-morbidities.

Physical activity (PA) is an effective intervention to improve risk factors associated with T2D and CVD in youth. A meta-analysis revealed a small to moderate effect of exercise training to improve fasting insulin and IR in youth, especially for those who are overweight or obese [5]. However, despite the known importance of PA in youth, less than one third of school aged children and adolescents meet the minimum UK government recommendation of 60 min of moderate to vigorous physical activity (MVPA) per day [6]. Furthermore, a meta-analysis of school-based interventions designed to increase levels of PA in adolescents showed a small but non-significant increase in moderate-to-vigorous physical activity equating to approximately two additional minutes of MVPA per day [7]. Adolescence is also associated with declining levels of PA [8] and represents a period in time when PA has the most profound effect on IR [9], highlighting the importance of exploring alternative “time efficient” forms of PA to improve cardiometabolic health outcomes in this group.

Recent observational data in youth have shown that small amounts (< 7 min) of vigorous intensity PA are associated with favourable temporal changes in cardiometabolic risk factors, including blood pressure,

waist circumference and cardiorespiratory fitness in youth [10]. This suggests that promoting high-intensity PA in this group may help in modifying disease risk. In healthy adolescents, just two weeks high-intensity interval training (HIIT), consisting of short duration sprint intervals, has been shown to improve aerobic fitness [11], indicating that short duration HIIT may have health benefits in youth. However evidence for the metabolic health benefits of HIIT in youth is currently limited to longer (7-12 weeks) training periods that often target adolescents who are overweight or have low fitness [12-14]. In contrast, it has recently been shown that improvements in insulin sensitivity (IS) and glucose tolerance in adolescent boys are possible after just a single bout of high-intensity interval exercise (HIIE) [15], suggesting that repeated bouts of HIIE performed over just two weeks may be a feasible way to improve glucose tolerance and IS in youth.

The increased IS following a single session of HIIE has been shown to persist for ~ 48 h in adults [16, 17], and up to 24 h in adolescents [18], meaning that any improvements in health outcomes beyond this time frame may be considered a chronic adaptation to training. Studies with both healthy adult participants and patients with T2D have shown an increase in the expression of skeletal muscle glucose transporters (e.g. GLUT-4) and the activity of mitochondrial enzymes after just 1-2 weeks of HIIT [19, 20], suggesting chronic adaptations are possible in this timeframe. However, a recent study has shown that two weeks of HIIT in a mixed-sex group of adolescents had no effect on fasting and postprandial plasma [insulin] and [glucose] outcomes when measured 24 and 72 h after the last training session [21]. This finding was surprising given previous work showing an acute bout of HIIE improved postprandial insulin and glucose outcomes in adolescent boys [15]. This lack of an effect may, in part, be due to the combined analysis of the adolescent boys and girls in previous work [21] and the use of the HOMA method to estimate IR which is known to have poorer measurement reliability [22] compared to other indices such as the quantitative insulin sensitivity check index (QUICKI) [23] and fasting glucose:insulin ratio (FGIR) [24]. Additionally, establishing the effects of exercise training in boys is important since boys are at an increased risk of developing IR and impaired fasting glucose compared to their female peers [25].

Therefore, the aim of this paper was to examine the effect of two weeks of HIIT on acute (1 day after final training session) and chronic (3 days after last training session) changes in glucose and insulin outcomes in adolescent boys. It was hypothesised that two weeks of HIIT would: 1) improve glucose and insulin outcomes the day after the last training session, representing an acute adaptation to HIIT); and 2) improve glucose, insulin and fitness outcomes up to 72 h post the final training session, representing a chronic adaptation to HIIT. A sub-set of data included in a previous report [21] was used to test this hypothesis.

Methods

Participants

Nine boys were recruited from a local secondary school. Following an explanation of the study procedures and the associated risks and benefits, parental consent and participant assent were obtained.

Participants completed an initial health questionnaire and were free from any metabolic or medical conditions. Ethical approval was granted by the university of Exeter sport and health sciences ethics committee. One boy failed to complete the HIIT due to an unrelated illness, and one boy could not complete the training due to an unrelated injury. This left a sample of seven participants (14.3 ± 0.3 y) for analysis.

Study design

This study consisted of four laboratory visits, and 6 training sessions in the school setting, which took place over a three week period. Visits included an initial familiarisation visit and three experimental visits. Visits 1 and 2 consisted of baseline measures of fitness and the [glucose] and [insulin] response to a mixed meal tolerance test (MMTT) prior to undertaking the HIIT intervention (PRE). Visits 1 and 2 were separated by 3-5 days. Participants then completed 6 supervised HIIT sessions over a two week period, after which post training measures were assessed 20 h (visit 3; 20 h POST) and 70 h post-intervention (visit 4; 70 h POST).

Visit 1: Familiarisation and baseline fitness assessment

Stature and body mass were measured to the nearest 0.01 m and 0.1 kg, and used to calculate body mass index (BMI). BMI was used to classify participants as normal weight, overweight and obese, using validated age-specific percentile cut points [26]. Pubertal status was determined by self-assessment of the five stages of pubic hair development described by Tanner [27].

Participants were familiarised with the cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) and completed a combined ramp-incremental and supramaximal test to exhaustion to determine maximal oxygen uptake ($\dot{V}O_2$ max) and the gas exchange threshold (GET) [28]. Pulmonary gas exchange and heart rate were measured (Cortex Metalyzer III B, Germany) and $\dot{V}O_2$ max was accepted as the highest 10 s average $\dot{V}O_2$ during the ramp or supra-maximal test. Peak power (PP) was taken as the highest power output during the ramp test whilst maintaining a cadence > 60 revolutions \cdot min $^{-1}$. The GET was estimated at the point where the first disproportionate increase in $\dot{V}CO_2$ production compared to $\dot{V}O_2$ and verified using the ventilatory equivalents for $\dot{V}O_2$ and $\dot{V}CO_2$.

Visits 2: Baseline metabolic assessment

Participants were driven to the laboratory and arrived at $\sim 07:45$ following a 12 h overnight fast. After 15 min of seated rest, participants provided a capillary blood sample for plasma [glucose] and [insulin]. At $\sim 08:30$ a MMTT was conducted which consisted of a commercially available fruit smoothie with 50 ml of double cream added, chocolate croissant with chocolate spread and a chocolate muffin (80 g of glucose, 68 g of fat, 7134 kJ). The meal was consumed over a 15 min period, after which capillary blood samples were taken at 30, 60, 120 min for assessment of plasma [glucose] and [insulin]. No other food was consumed and water was available *ad libitum* during visit 2 (PRE). This was recorded and subsequently

replicated for the POST measures. Participants remained in the laboratory throughout the visit, completing sedentary activities such as reading, watching DVDs or playing computer games. Participants left the laboratory at ~ 15:00.

HIIT intervention

Participants performed a two week HIIT programme on a cycle ergometer (Monark 827e, Monark exercise AB, Sweden) with adjustments made to the handle bar and seat height for each participant. Training took place within a local secondary school and consisted of 3 supervised HIIT sessions per week. Each session started with a 3 min warm up of unloaded pedalling, followed by 8-10 one min intervals at 90 % of the PP achieved during the incremental ramp test performed during visit 1. Each interval was interspersed with 75 s of unloaded pedalling. This HIIT protocol was selected to mimic previous studies from our laboratory [15, 29, 30]. Sessions one and two consisted of 8 x 1 minute bouts, sessions three and four 9 x 1 minute bouts and sessions five and six 10 x 1 minute bouts. Participants were asked to maintain a self-selected cadence (70-95 revolutions·min⁻¹) and were reminded of this during each session.

Visit 3 and 4: Post-training

The protocol outlined above for visit 2 was replicated the day after (20-POST) and three days (70-POST) after the last training session. One hour after completion of the MMTT during the 70- POST visit, participants completed a post intervention $\dot{V}O_2$ max assessment as described in visit 1.

Standardisation of physical activity and diet

Physical activity was measured during the 48 h period prior to each experimental visit using a wrist worn accelerometer (GENEActiv, Activinsights, UK). Time spent performing, light, moderate and vigorous PA was determined using cut points previously validated in a paediatric population [31]. Participants were asked to avoid any structured physical activity outside of the training intervention.

With supervision from their parents/guardians, a food diary was completed by each participant during the 48 h period preceding each experimental visit. Food diaries were assessed to estimate total energy and macronutrient content using commercially available software (CompEat Pro, Nutrition systems, UK). Participants were asked to replicate their diet during the 48 h preceding each experimental visit and if appropriate, to document any discrepancies.

Blood analyses

Fingertip capillary blood samples (~ 600 μ L) were taken from a pre-warmed hand into a fluoride heparin coated and lithium heparin coated microvette (CB 300 tubes, Sarstedt Ltd, Leicester, UK) for plasma

[glucose] and [insulin] determination, respectively. Both microvettes were centrifuged at 6000 revolutions.min⁻¹ for 10 min. Plasma was separated for immediate analysis of [glucose] (YSI 2300 Stat Plus Glucose analyser, Yellow Springs, OH, USA) or stored at -80°C for later analysis of plasma [insulin] using an ELISA enzyme immunoassay kit (DRG Diagnostics, Germany). Haematocrit and haemoglobin content of samples were assessed from the fasted sample of each visit in order to account for any change in plasma volume following training. In our laboratory, the within batch coefficients of variation for the plasma [insulin] and [glucose] analyses were < 5%.

Data handling

Changes in plasma [glucose] and [insulin] during the postprandial period following the MMTT were quantified using total and incremental area under the curve (tAUC, iAUC) analysed employing the trapezium rule (GraphPad Prism, GraphPad, SanDiego, CA). Fasting plasma [glucose] and [insulin] were used to calculate IR, IS and % using using HOMA-IR [32], QUICKI [23] and FGIR [24], which have been validated for use in adolescents [33].

Statistical analysis

Descriptive statistics were calculated using SPSS (version 19.0, Chicago, USA) and presented as mean ± SD. Analysis of the HOMA, QUICKI, FGIR, fasting [glucose] and [insulin] and AUC calculations across visits was performed using a one-way repeated-measures ANOVA. Changes in fitness parameters were assessed by a paired sample t-test. All results are presented as *P* values, unless stated otherwise. Pearson correlations were performed between HOMA-IR, QUICKI, FGIR, $\dot{V}O_2$ max and BMI at baseline and change in HOMA-IR after the 2 week training period. A significant correlation was accepted if *P*<0.05.

Results

The participants' descriptive characteristics are shown in Table 1. Tanner stage was provided by 6 participants and ranged between 3 and 4 (stage 4: *n*=4, stage 3: *n*=2). The BMI of participants ranged from 17.8 to 24.0 kg·m⁻², with 3 participants classified as overweight²⁶. Time spent in moderate and vigorous PA in the 48 h preceding each visit highlighted no differences between visits (*P*>0.05). No differences in estimated energy intake or macronutrient contribution to diet were evident prior to each visit (all *P*>0.05). The PA and diet data are shown in Table 2.

All participants completed the six HIIE training sessions, with 100% adherence to the protocol, with no adverse effects recorded.

Fasting and postprandial outcomes and cardiorespiratory fitness data are shown in Table 3. Fasting plasma [glucose], [insulin], QUICKI, FGIR, HOMA-IR, HOMA S% and HOMA % were unchanged at both 20-POST and 70-POST intervention compared to PRE (*P*>0.05). The plasma [glucose] and [insulin] response

during the postprandial period following the MMTT are shown in Figure 1. The tAUC and iAUC analyses for [glucose] and [insulin] were unchanged 20 h and 70- POST intervention when compared to PRE ($P > 0.05$). Absolute and relative $\dot{V}O_2$ max and PP output were unchanged POST compared to PRE ($P > 0.05$). Significant strong negative correlations were found between change in HOMA-IR, QUICKI and FGIR 20-POST and PRE HOMA-IR, QUICKI and FGIR ($r = -0.96$, $P = 0.001$; $r = -0.97$, $P = 0.001$; $r = -0.83$, $P = 0.022$ for HOMA-IR, QUICKI and FGIR respectively, Figure 2). The changes in HOMA-IR, QUICKI and FGIR post intervention were not related to $\dot{V}O_2$ max or BMI (both $P > 0.05$). There was no correlation between changes in postprandial outcomes at 20-POST and PRE training values ($P > 0.05$ for all).

Discussion

The key finding of this preliminary pilot study was that two weeks of HIIT did not elicit any acute or chronic changes to fasting and postprandial markers of metabolic health in a group of adolescent boys. However, a strong negative correlation was found between baseline IR (HOMA-IR, QUICKI and FGIR) and the change at 20-POST HIIT, suggesting a beneficial effect in participants with the greatest IR at baseline. Short duration HIIT protocols may therefore be a useful exercise strategy for youth with poorer metabolic health profile at baseline and should be a target for future intervention work.

In the present study two weeks of HIIT (8-10 1 min intervals at $\sim 90\%$ of PP, interspersed with 75 s of unloaded pedalling) was not sufficient to improve IR or fasting and postprandial measures of metabolic health when measured 20 h or 70 h after the final training session. Interestingly, our findings corroborate those of earlier studies conducted on healthy, asymptomatic adolescents. In two separate studies [34, 35], Buchan and colleagues reported no change to either fasting [insulin] or [glucose] after a 7 week school-based HIIT program (4-6 repeats of 30 s maximal sprints with 20-30 s recovery 3 x per week), but did not report HOMA index of IR, QUICKI or FGIR. However, in these studies moderate intensity PA did improve fasting [insulin] suggesting this intensity of exercise may be superior to HIIT. In contrast, studies investigating the effectiveness of HIIT in overweight or obese participants over 12 weeks [12-14] have shown improvements to fasting [glucose], [insulin] and HOMA-IR. These findings may show that the duration of the HIIT programme is important as HIIT programmes lasting >12 weeks have yet to be conducted in normal weight adolescents to our knowledge. However, it is pertinent to note that in these HIIT studies in overweight and obese youths (15, 27, 36) the participants had a baseline HOMA-IR of ~ 4 -5 arbitrary units (AU), which is notably higher than the present study (2.5 ± 1.0 AU) and suggests a limited window to improve IR after HIIT in participants with low baseline IR. Published reference values for HOMA-IR in Caucasian youth suggest a 75th percentile cut-off point for cardiometabolic risk at 3.02 AU [36]. In our study, analysis of the individual data found three participants appeared to respond positively to two weeks of HIIT and were characterized by an IR between the 90th and 97th centile. These participants recorded an improvement in IR 20- POST ranging from 59-219%, with the largest improvement occurring in the participant with the highest baseline HOMA-IR. This is reflected by the significant negative correlation between the change in IR 20- POST and PRE IR (Figure 2) which was evident in HOMA-IR, QUICKI and FGIR and suggests that two weeks of HIIT may be a feasible intervention

to improve metabolic health in adolescents with a high IR at baseline. Finally, it has recently been reported that the ability for physical activity to attenuate IR is diminished in 16 year-old adolescents [9]. The mean age of participants in the present study was 14.3 y with Tanner stages between 3 and 4, which may have influenced the effectiveness of the HIIT intervention to modify plasma [glucose] and [insulin]. Taken collectively, there may be a limit to improvements to IR through just two weeks of HIIT, especially in those who have a low IR at baseline, are or normal weight and in late adolescence.

In the current study, two weeks of HIIT had no effect on postprandial plasma [glucose] and [insulin] after a MMTT. The inclusion of postprandial measures is a strength of our study because it is known that postprandial hyperglycaemia is a contributor to glycaemic control (e.g. HbA_{1c}), which often precedes any increase in fasting glucose levels and is more harmful to skeletal muscle glucose homeostasis than chronically sustained hyperglycaemia [37]. In overweight/obese adolescents reductions in two hour postprandial [glucose] and [insulin] after an oral glucose tolerance test (OGTT) have been shown after 12 weeks of HIIT, but not after matched-duration moderate intensity exercise training [14]. In healthy young men (21.2 y), Babraj and colleagues [38] found two weeks of HIIT (6 sessions of 4-6 30 s sprints) reduced the plasma [glucose] and [insulin] AUC response to an OGTT by 12 % and 37 % respectively, 2 to 3 days after the last exercise session. In agreement with the present study, however, the authors found no changes to fasting [glucose] or [insulin] [38]. These findings suggest that the response to exercise training may differ for fasting and dynamic (postprandial) measures of [insulin] and [glucose], which we have also found in previous work [18]. Thus it is possible that the use of the MMTT to examine postprandial changes in glucose and insulin rather than an OGTT in the current study may account for the lack of effect when compared to the work by Babraj and colleagues. In particular, the MMTT will have a lower glycaemic index which will alter the glucose excursions [39] and is likely to have influenced the rate of glucose appearance in the circulation [40]. That said the MMTT holds better external validity as it is more representative of the habitual nutrient meal composition compared to an OGTT.

One of the aims of this study was to highlight any acute benefits from the HIIT by measuring the outcomes 20 h post the final training session. Contrary to our original hypothesis, no acute improvements in fasting or postprandial [glucose] and [insulin] were present at ~ 20-POST. We have previously shown that a single bout of HIIE can improve both glucose tolerance and IS in adolescent boys [15], and that these changes persist for up to 24 h after exercise [18]. It is therefore surprising that two weeks of HIIT did not improve metabolic outcomes the day after the last training session in the current study. However, the aforementioned acute exercise studies used an OGTT and not a MMTT, which may account for the discrepancies in findings. The lack of change to metabolic outcomes 20-POST in the current study may also indicate that improvements after HIIT in healthy adolescents do not persist into the next day.

Aerobic fitness, as measured using a validated cycle test to exhaustion, was unchanged in adolescent boys after the 2 week HIIT programme. This result differs from the outcome of a recent meta-analysis showing that ≥ 4 weeks of HIIT to have a large effect on improving cardiorespiratory fitness (ES= 1.05, mean difference = 2.6 mL.kg⁻¹.min⁻¹) in adolescents [41]. A 5 % improvement in $\dot{V}O_2$ max has been

shown after two weeks of HIIT, however this study incorporated 30 s “all out” sprint type HIIT [11], which may have provided a greater stimulus to augment $\dot{V}O_2$ max.

This study is the first to assess both fasting and postprandial measures of metabolic health in a healthy adolescent population after short duration HIIT programme. Previous studies in this area are largely limited to overweight/obese adolescents and longer duration HIIT programmes. The strengths of this study include the control of physical activity and diet prior to the experimental measures, which limits any confounding effects of these factors. Additionally we include multiple indices on IR, which in previous work is limited to HOMA-IR, this is important as we have recently shown HOMA-IR to have a large variability in this population, with other measures such as QUICKI and FGIR potentially better placed to use in this population [22]. Limitations include the lack of a control group, although this is consistent with other short duration HIIT studies in youth [11] and adults [42]. The small sample size is also a limitation; however this study is reported as a pilot study.

Conclusion

This preliminary study shows that fasting or postprandial measures of [insulin] and [glucose] in adolescents were not sensitive to change after two weeks of HIIT. However, a strong negative correlation between baseline IR and IR 20-hour POST across all measurement indices highlights the potential for this type of intervention to promote metabolic health in a more clinical population of individuals with elevated baseline IR and is worthy of future investigation.

List Of Abbreviations

CVD	Cardiovascular disease
FGIR	Fasting glucose:insulin ratio
GET	Gas exchange threshold
GLUT-4	Skeletal muscle glucose transporter 4
HIIE	High-intensity interval exercise
HIIT	High-intensity interval training
HOMA-IR	Homeostatic model assessment of insulin resistance
iAUC	Incremental area under the curve
IR	Insulin resistance
IS	Insulin sensitivity
MMTT	Mixed Meal Tolerance Test
MVPA	Moderate to vigorous physical activity
PA	Physical activity
PP	Peak power
QUICKI	Quantitative insulin sensitivity check index
VO ₂ max	Maximal oxygen uptake
T2D	Type two diabetes
tAUC	Total area under the curve

Declarations

Ethics approval and consent to participate

The study was conducted according to the ethical guidelines of the Declaration of Helsinki and was approved by the ethics committee of the University of Exeter Sport and Health Science department. All participants provided a written informed assent, and parental consent.

Consent to publish

Not applicable

Availability of data and material

The datasets generated and analysed during the current study are not publicly available due to ethical restrictions but are available from the corresponding author upon reasonable request..

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

E.J.C., B.B., C.A.W., N.A., and A.R.B. conception and design of research; E.J.C., B.B., S.H., and S.J. performed experiments; E.J.C., B.B., and S.J. analysed data; E.J.C., B.B., C.A.W., N.A., and A.R.B. interpreted results of experiments; E.J.C. and A.R.B. prepared figures; E.J.C. drafted manuscript; E.J.C., B.B., C.A.W., S.H., S.J., N.A., and A.R.B. edited and revised manuscript; E.J.C., B.B., C.A.W., S.H., S.J., N.A., and A.R.B. approved final version of manuscript.

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Tables

Table 1. Participant descriptive characteristics.

	Mean \pm SD	Range
Age, y	14.3 \pm 0.3	13.9 to 14.7
Body mass, kg	60.0 \pm 7.4	57.7 to 69.9
Stature, m	1.67 \pm 0.81	1.57 to 1.78
BMI, kg.m ²	21.6 \pm 2.6	17.8 to 24.6
Results expressed as mean \pm SD and range. BMI, body mass index.		

Table 2. Physical activity and dietary intake during the 48 h preceding each experimental visit.

	PRE	20 h POST	70 h POST	ANOVA <i>P</i> -value
Moderate-vigorous physical activity (min•day ⁻¹)	45 \pm 25	59 \pm 42	56 \pm 17	0.55
Total energy intake (kcal•day ⁻¹)	1971 \pm 280	1950 \pm 294	2052 \pm 293	0.71
Energy from carbohydrate (%)	43 \pm 7	47 \pm 5	47 \pm 9	0.67
Energy from fat (%)	40 \pm 10	36 \pm 4	38 \pm 5	0.54
Energy from protein (%)	18 \pm 4	17 \pm 4	14 \pm 4	0.43

Results shown as Mean \pm SD. 20 h POST includes the final training session of the HIT intervention (~27 min)

Table 3. Physical and biochemical characteristics at PRE, 20 h and 70 h post intervention.

	PRE	20 h post intervention	70 h post intervention	P-value
Fasting				
Glucose (mmo•L ⁻¹)	5.05 ± 0.3	5.00 ± 0.3	5.09 ± 0.2	0.86
Insulin (μU•ml.)	19.41 ± 8.4	18.63 ± 3.5	20.60 ± 8.2	0.84
HOMA-IR (arbitrary units)	2.47 ± 1.04	2.37 ± 0.45	2.61 ± 0.99	0.85
HOMA-S% (arbitrary units)	45.86 ± 15.44	43.51 ± 8.62	42.27 ± 13.11	0.87
HOMA- B% (arbitrary units)	170.93 ± 39.86	172.70 ± 18.10	177.26 ± 46.70	0.93
QUICKI (arbitrary units)	0.311 ± 0.017	0.311 ± 0.010	0.308 ± 0.015	0.89
FGIR (mg/10 ⁻⁴ U)	5.27 ± 1.64	4.96 ± 0.82	4.92 ± 1.43	0.86
IMTT				
iAUC Glucose (nmol•min•L ⁻¹)	91.08 ± 80.26	107.79 ± 77.26	81.06 ± 43.99	0.57
tAUC Glucose (nmol•min•L ⁻¹)	696.76 ± 74.11	707.67 ± 48.73	690.47 ± 36.96	0.56
iAUC Insulin (μU•ml•min ⁻¹)	4499.57 ± 1834.26	4538.14 ± 1882.24	4908.71 ± 1329.51	0.78
tAUC Insulin (μU•ml•min ⁻¹)	6807.00 ± 1415.10	6774.29 ± 1661.46	7380.29 ± 1906.95	0.60
Fitness				
O ₂ max (mL•kg ⁻¹ min ⁻¹)	40.71 ± 9.80	-	42.08 ± 10.75	0.25
O ₂ max(L•min ⁻¹)	2.44 ± 0.70	-	2.52 ± 0.76	0.27
HR max (b min ⁻¹)	192 ± 8	-	193 ± 9	0.65
GET (L•min ⁻¹)	1.33 ± 0.29	-	1.35 ± 0.28	0.85
GET (% VO ₂ max)	55.7 ± 7.1	-	54.9 ± 7.5	0.60
PP (W)	233 ± 58	-	244 ± 66	0.09

Results shown as mean ± SD. HOMA-IR, homeostatic assessment of insulin resistance; HOMA-S%, homeostatic assessment of insulin sensitivity; HOMA- B%, %, homeostatic assessment of beta-cell function; QUICKI, quantitative insulin sensitivity check index; FGIR, fasting glucose;insulin ratio, iAUC, incremental area under curve, tAUC, total area under curve; HR, heart rate; GET, gas exchange threshold; PP, peak power.

Figures

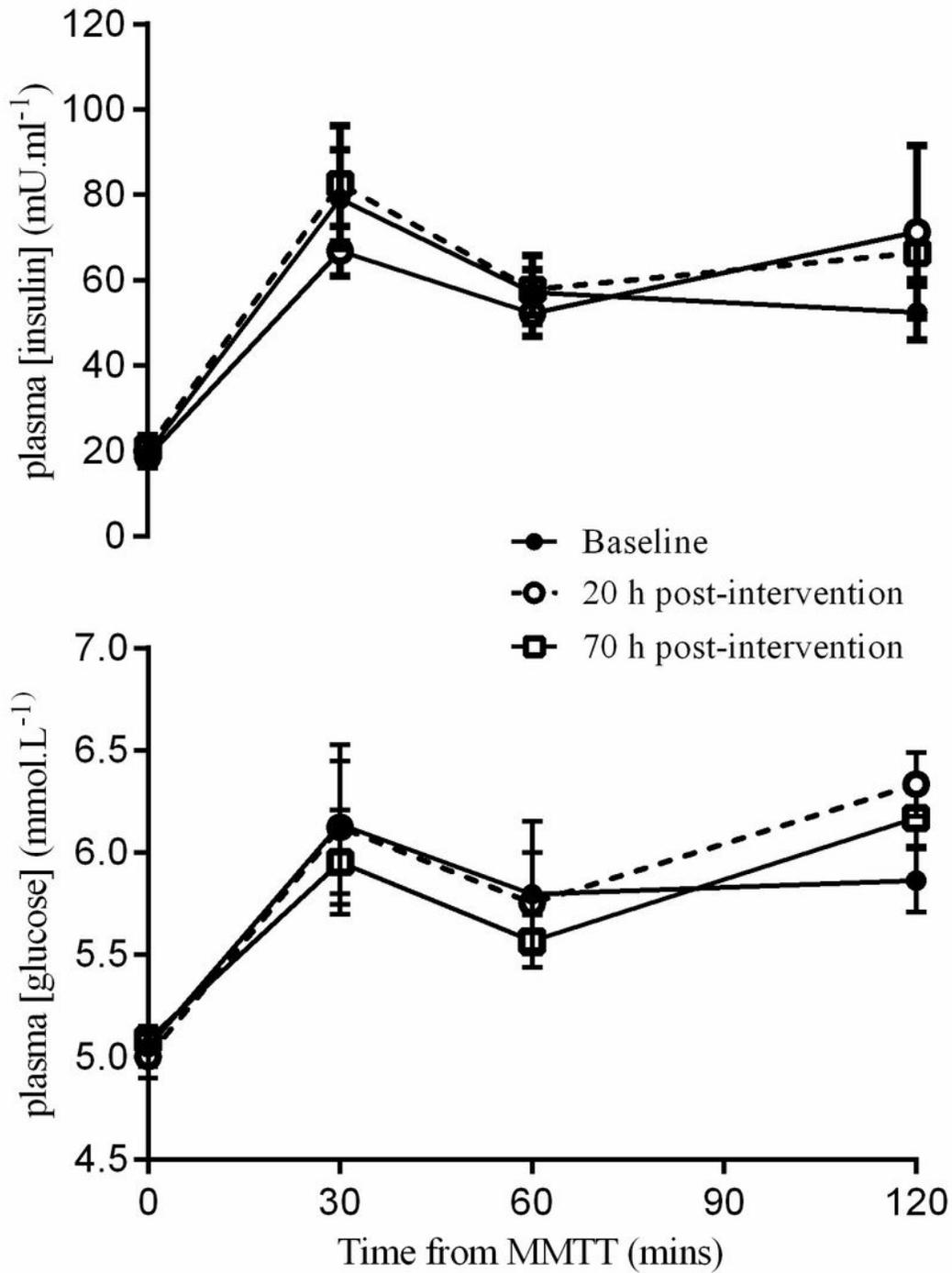


Figure 1

Mixed meal tolerance test: Postprandial plasma [glucose] and [insulin] response to the mixed meal tolerance test (MMTT) at baseline and at 20 h and 70 h after the HIIT intervention. Results shown as mean \pm SEM

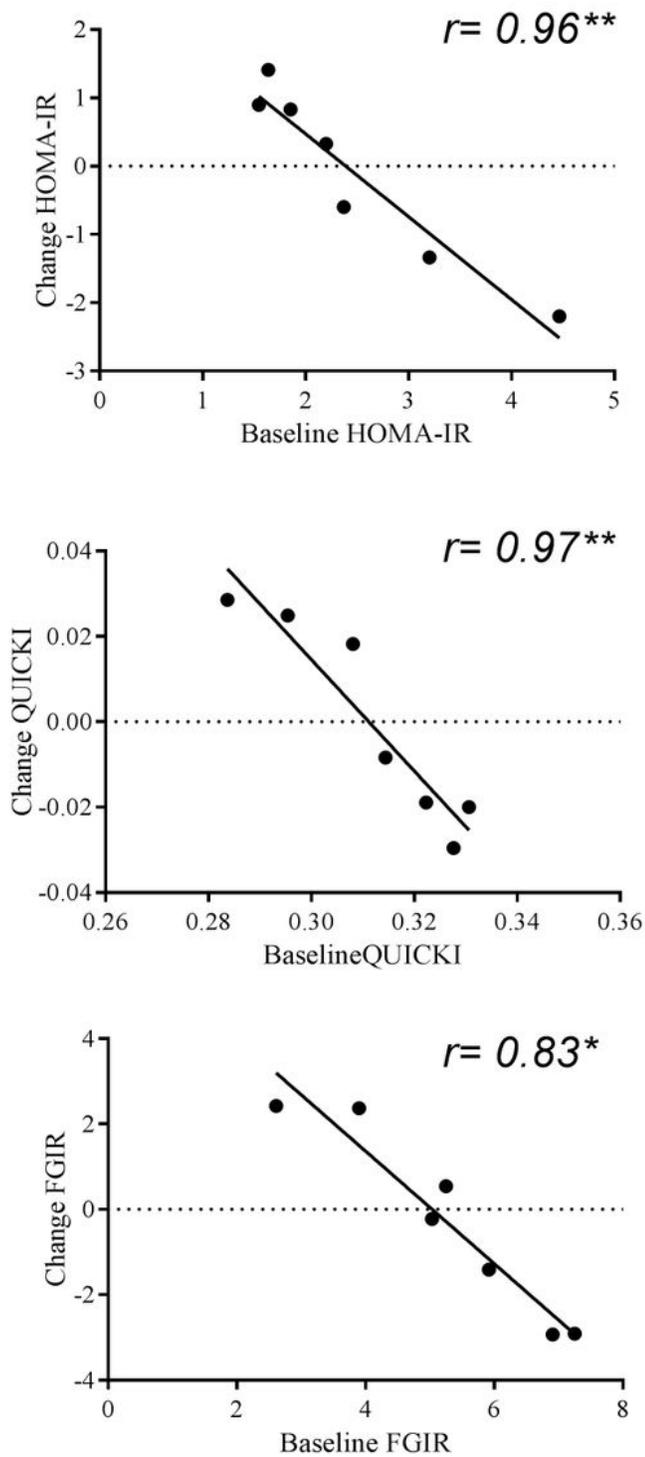


Figure 2

Correlations between changes in IS indices: Scatter plot showing correlation between change at 20 h POST HIIT and at baseline for HOMA-IR, QUICKI and FGIR. ****** $P < 0.01$ ***** $P < 0.05$