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ORIGINAL ARTICLE

Effect of acute exercise on glucose tolerance following post-exercise feeding

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Abstract It is well documented that a single bout of endurance exercise (EE) can improve insulin sensitivity, whereas relatively little is known about the acute effects of resistance exercise (RE) in humans. The objective of this study is to investigate the insulin and glucose responses to an oral glucose tolerance test (OGTT) following a high intensity bout of either EE or RE followed by post-exercise carbohydrate–protein hydrolysate ingestion. Eighteen participants were divided into two groups: a group in which nine participants completed 1 h of EE (cycle ergometry at 75% W_{max}) and a RE group in which nine participants completed a RE circuit (3 sets of 10 repetitions). Participants ingested 1.5 l of a carbohydrate (200 g)–protein hydrolysate (50 g) beverage within 1 h of exercise completion. An OGTT was performed 6 h post-exercise. On the control day the endurance and resistance groups performed the above protocol without the prior exercise (CEE or CRE). The control and exercise days were counterbalanced. RE reduced plasma glucose AUC (822 ± 68 vs. 694 ± 23 mmol l^{-1}·120 min; CRE vs. RE, respectively; $P < 0.05$) but EE did not lead to a change (784 ± 40 vs. 835 ± 59 mmol l^{-1}·120 min; CEE vs. EE, respectively). Plasma insulin AUC remained unchanged compared to the control in both the RE and EE groups. The results suggest that the benefit of RE on glucose tolerance following CHO intake remains for 6 h even when a carbohydrate–protein hydrolysate beverage was ingested within 1 h after exercise, while the well documented benefit of EE was not observed.

Keywords Resistance exercise · Endurance exercise · Muscle glycogen content · Glucose–protein hydrolysate drink · OGTT

Introduction

Regular exercise is of key importance in regulating glycemic control and preventing the development of hyperinsulinemia. Cross-sectional studies have observed higher insulin stimulated glucose uptake and a blunted insulin response to a glucose load in endurance-trained individuals compared to their untrained counterparts (King et al. 1987; Lohmann et al. 1978; Rodnick et al. 1987). Intervention studies have demonstrated that endurance exercise (EE) training can increase both insulin stimulated glucose clearance and insulin sensitivity in healthy (Dela et al. 1992; Houmard et al. 1999), obese (Bruce et al. 2006; Houmard et al. 2004) and diabetic groups (Dela et al. 1995; Reitman et al. 1984) (see Ivy et al. 1999 for a review of the pre 1999 literature). In addition to training induced enhancements in insulin sensitivity, an acute bout of EE has been shown to improve insulin stimulated glucose clearance (Richter et al. 1989; Wojtaszewski et al. 1997, 2000) and these effects can persist for up to 48 h post exercise (Mikines et al. 1988; Perseghin et al. 1996).

There are relatively few studies that have investigated the effects of resistance exercise (RE) training on glucose handling and insulin sensitivity. Those that have, observe

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that resistance training can decrease fasting blood glucose and improve glucose tolerance in humans (Craig et al. 1989; Fenicchia et al. 2004; Holten et al. 2004; Ibanez et al. 2005; Miller et al. 1984, 1994). Studies have reported that both aerobic and RE training in rats increased GLUT4 protein content and insulin stimulated glucose uptake (Holten et al. 2004; Yaspelkis et al. 2002). Furthermore, Krisan et al. (2004) observed that increased insulin stimulated glucose uptake post resistance training was associated with enhanced insulin signalling demonstrated by an increase in PI3kinase activity.

To date there has been relatively little investigation into the acute effects of RE on muscle glucose uptake. Hernandez et al. (2000) observed an increased rate of glucose uptake 6–12 h after an acute bout of RE in rats. Human studies have also observed that acute RE can improve glucose tolerance and insulin sensitivity for at least 24 h (Fenicchia et al. 2004; Fluckey et al. 1994; Koopman et al. 2005b).

Evidence suggests that the mechanisms leading to improvements in glucose tolerance and insulin sensitivity post exercise differ between EE and RE. It has been shown that EE-associated increases in glucose uptake do not involve the up-regulation of any components in the insulin signalling cascade (Dela et al. 1992, 1995). One regulatory factor appears to be the low muscle glycogen content post exercise that activates glycogen synthase (Derave et al. 2000; Nielsen et al. 2001), reducing the intracellular glucose concentration and promoting glucose uptake. However with RE there is evidence suggesting that up-regulation of some of the components within the insulin signalling cascade occurs post-exercise and such up-regulation is maintained if not enhanced with high muscle glycogen (Creer et al. 2005; Koopman et al. 2006). We, therefore, hypothesized that the improvement in glucose uptake and insulin sensitivity would remain after post-exercise glycogen re-synthesis in the case of RE, and would not be observed in the case of EE.

The aim of the present study was therefore to investigate the effects of a prior bout of either endurance or RE on glucose tolerance measured 6 h post exercise by means of an OGTT. Participants were fed a carbohydrate–protein hydrolysate beverage containing ~1,000 kcal, immediately post-exercise to induce high rates of muscle glycogen re-synthesis (van Loon et al. 2000; Zawadzki et al. 1992) in an attempt to restore muscle glycogen stores.

Methods

Participants

Eighteen healthy, recreationally active students, the characteristics of whom are shown in Table 1, were recruited for the purposes of this study. Participants were excluded from the study if they had any known metabolic impairment or were being medically treated for heart or blood pressure irregularities. Before enrolment in the study, each of the participants was fully informed of the purpose and potential risks associated with the procedures, and a written informed consent was obtained. The study was approved by the Ethics Committee of the School of Sport and Exercise Sciences at the University of Birmingham, UK.

Table 1 Subject characteristics

	Group	
	Endurance	Resistance
Age (year)	21 ± 2	23 ± 1
Body mass (kg)	71.7 ± 8.3	74.1 ± 6.7
Height (m)	1.77 ± 0.09	1.76 ± 0.08
BMI (kg m^{-2})	22.8 ± 1.9	23.9 ± 0.8
W_{max} (W)	258 ± 38	NA
$\dot{V}O_2$ max (ml kg^{-1} min^{-1})	51.1 ± 6.6	NA
1 RM leg extension (kg)	NA	142 ± 13
1 RM leg curl (kg)	NA	111 ± 6

Values are mean ± SE, $n = 9$ for each group

BMI body mass index, W_{max} maximal power output, $\dot{V}O_2$ max maximal oxygen uptake, *1 RM* one repetition maximum

General design

The 18 participants were randomly assigned into two groups; an EE group and a RE group. Following the preliminary testing described below, the participants participated in two randomly assigned trial days, separated by at least 5 days. The EE trial day and the RE trial day consisted of 1 h of the respective exercise, followed by ingestion of a carbohydrate–protein hydrolysate beverage. The participants then fasted (water ad libitum) and rested for 5 h after which the OGTT was started. On the control days for both the endurance exercise group (CEE) and the resistance exercise group (CRE) the above routine was similar, however without the prior exercise bout.

Preliminary testing

Approximately 1 week before the start of the study all participants visited the laboratory on two separate occasions. On the first visit an oral glucose tolerance test (OGTT) was performed to check whether the participants had normal glucose tolerance and on the second visit the EE group completed an incremental exercise test to exhaustion on a cycle ergometer, and the RE group had their ten repetition maximum (10 RM) determined on six pieces of resistance equipment.

Oral glucose tolerance test

For the first preliminary visit participants reported to the laboratory in the morning (8 a.m.) after an overnight (10 h) fast. On arrival, standard measures of height and body mass (Seca Alpha, Hamburg, Germany) were taken. Participants were then placed in a reclined position and a flexible 20-gauge Teflon catheter (Quickcath, Becton Dickinson, Plymouth, UK) was inserted into an antecubital vein. A three-way stopcock (PVB Medizintechnik, Kirchseean, Germany) was attached to this to allow for repeated blood sampling. A resting blood sample (5 ml; $t = 0$) was taken, immediately after which participants ingested a 25% glucose beverage [75 g glucose (Meritose-200; Amylum UK Ltd, London, UK) made up with water to a volume of 300 ml]. Further blood samples (5 ml) were collected with participants seated at 15, 30, 45, 60, 90 and 120 min. The catheter was kept patent by flushing with 2–3 ml of isotonic saline (0.9%, Baxter, Norfolk, UK) after each blood sample collection and at 75 and 105 min.

The same procedure was used during the OGTT on the trial days.

Endurance exercise group—$\dot{V}O_2$ max On their second preliminary visit the EE group carried out an incremental exercise test to volitional exhaustion on a cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands).

W_{max} values were used to determine the workload (75% W_{max}) used in the later experimental trial. Breath-by-breath measurements were taken throughout exercise using an Oxycon Pro automated gas-analysis system (Jaeger, Wuerzburg, Germany). Heart rate (HR) was measured continuously by telemetry using a Polar Vantage HR monitor (Polar Electro Oy, Kempele, Finland). $\dot{V}O_2$ was considered to be maximal if the following conditions were met (1) a levelling off of $\dot{V}O_2$ with further increasing workloads (an increase of no more than 2 ml kg^{-1} min^{-1}), and (2) a respiratory exchange ratio (RER) of greater than 1.05.

Resistance exercise group—10 RM On their second preliminary visit the RE group underwent a gym session during which their 10 RM was determined on each of six pieces of equipment (Stairmaster, USA). The ten repetition max was determined as follows. The desired movement and positioning of the machine was fully explained and each participant was allowed to perform several unloaded repetitions to familiarise themselves with the movement of the exercise and allow correct positioning. An initial weight of 50% 10 RM was estimated by the instructor as a warm up, weight was then added incrementally on the basis of the relative effort of the warm up initially and each previous set thereafter. If it was clear that the participant could perform ten repetitions the set was ended prior to completion of the ten repetitions to prevent fatigue. Weight was then added until the participants could perform no more than ten repetitions for a given weight, this weight was selected as their 10 RM. Participants were allowed 5–10 min rest between successive attempts and the exercise stations were alternated between different muscle groups to prevent premature fatigue.

The exercises were chosen in an attempt to recruit most major muscle groups during the exercise session and included; bicep curl, seated shoulder press, seated leg curl, seated leg extension, assisted triceps dips, assisted pull ups. In addition participants were asked to perform as many repetitions as possible of press ups and sit ups in 30 s.

All exercise sessions were supervised by a qualified YMCA gym instructor.

Exercise protocol

Endurance exercise (EE)

Endurance exercise consisted of a 5 min warm up followed by cycling at 75% W_{max} for 55 min.

Resistance exercise (RE)

RE consisted of a 15 min warm up [participants performed an initial 5 min warm up on the treadmill at a self-selected pace (<11 km/h). This was followed by a resistance circuit at ~50–60% of the 10 RM], followed by three sets of 10 RM (as listed above). Participants performed each set at a rate such that the ten repetitions were completed in 30 s. Each exercise was separated by 30 s rest with a 2 min rest interval between each full circuit.

Drinks

The beverage ingested on trial days contained ~1,000 kcal, the major constituents of which were 200 g of maltodextrin (Glucidex-19, Roquette, Corby, UK) and 50 g of whey protein hydrolysate (LE80GF, DMV International The Netherlands). In order to make the drink palatable 6 g citric acid, 3 g guar, 2 g sodium citrate (all from Sigma-Aldrich, Dorset, UK), and 2 g raspberry flavouring (Givaudan, OH, USA) were added. The final beverage was made up with water to a volume of 1.5 l.

Diet and exercise

Participants were asked to record their food intake the evening before the first trial day so that the meal consumed could be repeated the evening before the second trial day. Participants were asked to refrain from alcohol and

caffeine consumption and any vigorous exercise 24 h before each trial day.

Biochemical analyses

Blood samples (5 ml) were collected into pre-chilled EDTA containing tubes (Becton Dickinson, Plymouth, UK) and centrifuged at 3,500 rev min^{-1} at 4°C for 10 min. Aliquots of plasma were immediately frozen in liquid nitrogen and stored at –75°C until further analyses. Glucose (HK125, ABX diagnostics, Montpellier, France) was analyzed on a COBAS MIRA S plus BIO semi automatic analyzer (la Roche, Basel, Switzerland). Insulin was analyzed by enzyme-linked immunosorbent assay (ELISA DX EIA-2935, IDS limited, Bolden, UK).

Calculations

Plasma glucose and insulin concentrations during the OGTT were used to determine the whole body insulin sensitivity index (ISI) according to the following equation of Matsuda (Matsuda and DeFronzo 1999):

$$\mathrm{ISI} = \frac{10,000}{\sqrt{(\mathrm{FPG} \times \mathrm{FPI})\ (\text{mean OGTT insulin concentration})\ (\text{mean OGTT glucose concentration})}},$$

where FPG is fasting plasma glucose concentration, FPI fasting insulin concentration and 10,000 represents a constant that allows numbers ranging between 1 and 12 to be obtained. The square root conversion is used to correct the non-linear distribution of values.

Statistical analysis

Two-way analysis of variance for repeated measures was used to compare differences between the groups in all biochemical parameters. In the case of a significant F ratio, paired t tests were carried out to locate differences within participants, and unpaired t tests were carried out to locate the differences between exercise types. All statistical tests were carried out using SPSS for windows version 10.0 software package (Chicago, IL, USA). All data are reported as means ± SE. Statistical significance was set at $P < 0.05$.

Results

Oral glucose tolerance test

All participants showed normal fasting plasma glucose (4.68 ± 0.92 mmol l^{-1}) and insulin (17.07 ± 5.69 μU ml^{-1}) concentrations, with no differences observed between the EE and RE groups (Table 2).

Glucose response

There was no difference in resting plasma glucose concentrations between the control and exercise trials for either EE or RE groups (Table 2). However over the course of the 2-h OGTT it was observed that the participants in the RE trial had a significantly lower glucose area under the curve (AUC) when compared to both the CRE trial (694 ± 29 vs. 823 ± 45 mmol l^{-1}·120 min RE vs. CRE, respectively; $P < 0.05$) and the EE trial (694 ± 29 vs. 835 ± 59 mmol l^{-1}·120 min RE vs. EE, respectively; $P < 0.05$; Fig. 1a). No difference was observed between the EE and CEE trials.

Insulin response

There was no difference in resting plasma insulin concentrations between the control and exercise trials for either endurance or RE groups (Table 2). Although there is a 10% reduction in insulin AUC between the respective control and exercise trials in both the endurance (4,118 ± 590 vs. 3,759 ± 324 μU ml^{-1}·120 min CEE and EE, respectively) and resistance groups (4,978 ± 706 vs. 4,458 ± 598 μU ml^{-1}·120 min CRE and RE, respectively; Fig. 2), this did not reach significance.

Insulin sensitivity index

The ISI was not different between the EE and CEE trials and although there was an 18% increase in insulin sensitivity in the RE trial compared to the CRE trial this did not reach significance (Table 2).

Discussion

The major finding of the present study is that RE immediately followed by ingestion of a carbohydrate–protein hydrolysate beverage can reduce plasma glucose AUC by 15% following a 75 g glucose load when measured 6 h post exercise. In addition an 18% increase in insulin sensitivity was observed following RE although this failed to reach statistical significance. In contrast we found no changes in either glucose AUC or insulin sensitivity 6 h post EE.

Table 2 Plasma glucose, insulin and insulin sensitivity indices pre- and post-exercise for both endurance and resistance groups

	Group			
	CEE	EE	CRE	RE
Plasma glucose (mmol l^{-1})	4.48 ± 0.27	4.78 ± 0.43	4.76 ± 0.39	4.51 ± 0.24
Plasma insulin (μU ml^{-1})	18.85 ± 1.20	18.65 ± 1.01	15.30 ± 2.33	13.70 ± 1.60
Insulin sensitivity index	4.58 ± 0.46	4.57 ± 0.61	4.78 ± 0.65	5.63 ± 0.60

Values are mean ± SE, $n = 9$. Plasma glucose and insulin values are for time zero during the OGTT

CEE and *EE* control and exercise trial, respectively, for endurance group; *CRE* and *RE* control and exercise trial, respectively, for resistance group

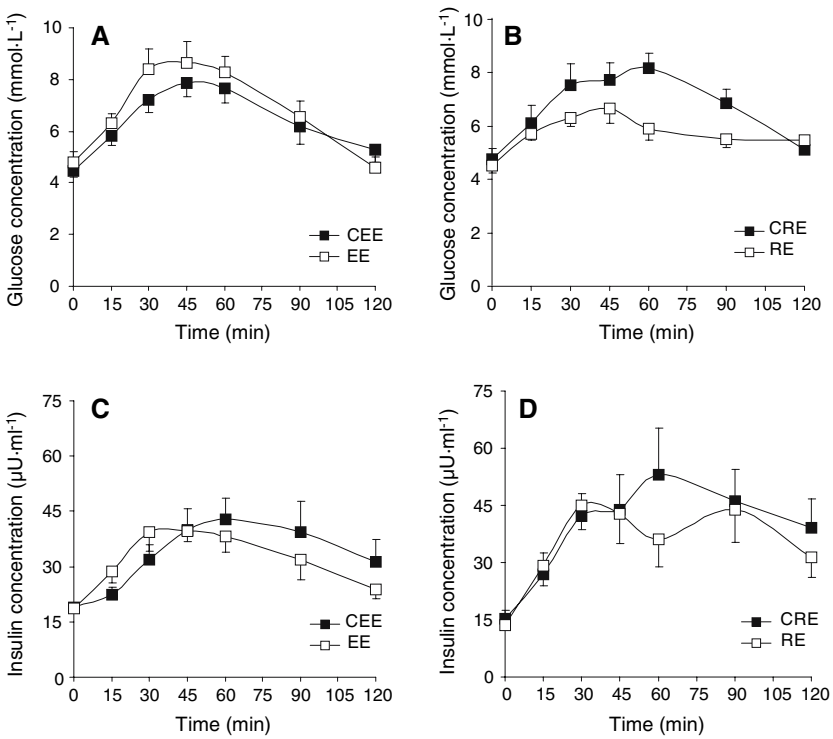

Fig. 1 Glucose (**a**, **b**) and insulin (**c**, **d**) concentrations during a 2 h OGTT

Although previous studies have shown that acute EE can lead to enhanced skeletal muscle glucose uptake when compared to non-exercise (Wojtaszewski et al. 1997, 2000), this increase in insulin stimulated glucose uptake following EE is not thought to be due to an up-regulation of any of the proximal components of the insulin signalling pathway such as insulin receptor tyrosine kinase (IRTK) activity, insulin receptor substrate 1 (IRS-1), PI3 kinase activity or glycogen synthase kinase 3 (GSK-3) (Wojtaszewski et al. 1997, 2000). It is important to note that 4 h following one leg knee extensor kicking exercise, Wojtaszewski et al. did observe a 40% reduction in muscle glycogen content in the exercised leg that showed enhanced glucose uptake (Wojtaszewski et al. 1997, 2000). In subsequent studies it has been postulated that the decrease in muscle glycogen content post exercise drives the activation of glycogen synthase, which will lower intracellular glucose concentrations and ultimately increase glucose uptake (Derave et al. 2000; Nielsen et al. 2001; Yan et al. 1992; Zachwieja et al. 1991). Several studies have demonstrated a negative correlation between muscle glycogen concentration and the activity of glycogen synthase and glucose uptake (Richter et al. 1989, 2001). In addition it has recently been demonstrated that muscle glycogen content is a more potent regulator of glycogen synthase activity and glucose uptake than either insulin or muscle contraction (Nielsen et al. 2001).

The observations of the present study in which no change occurs in glucose tolerance following EE are not at odds with the literature as the participants were fed immediately post exercise with a carbohydrate–protein hydrolysate beverage (~1,000 kcal). Such beverages have previously been shown to induce a large plasma insulin response and enhance glycogen synthesis rates (van Loon et al. 2000) thus we assume that this feeding schedule will

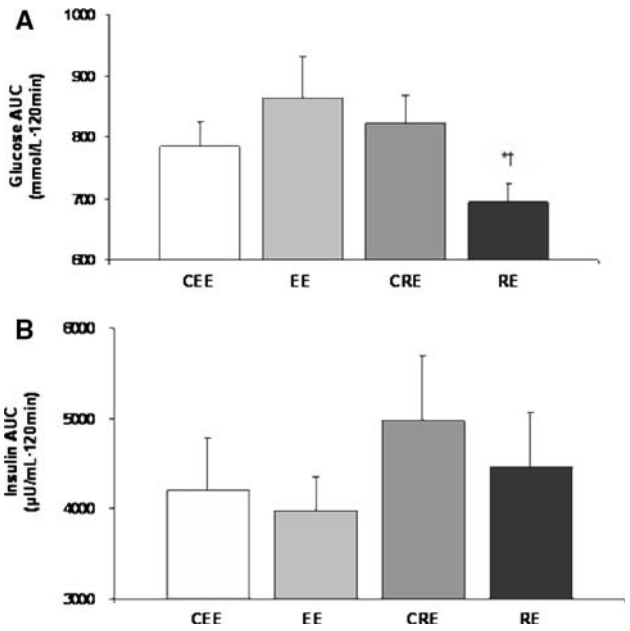

Fig. 2 Glucose (**a**) and insulin (**b**) AUC for endurance and resistance exercise groups pre-and post an acute bout of exercise; *AUC* area under the curve, *CEE* endurance exercise control trial, *EE* endurance exercise trial, *CRE* resistance exercise control trial, *RE* resistance exercise trial; values are mean ± SE, $n = 9$ for each group; * significant difference from respective control trial, † significant difference from endurance exercise trial

have resulted in at least partial restoration of the muscle glycogen stores. If muscle glycogen content is the primary regulatory factor controlling glucose uptake following EE, we would therefore not expect to find a change in glucose tolerance following EE and post-exercise replenishment of muscle glycogen stores.

Although quantitatively there are fewer studies investigating the effects of RE on glucose clearance and insulin sensitivity than there are investigating EE, the evidence suggests that RE can be just as effective as EE in bringing about such improvements (Krisan et al. 2004; Perseghin et al. 1996; Reitman et al. 1984). Koopman et al. (2005a) have recently observed that acute RE in man can lead to a decrease in muscle glycogen content. In contrast to EE however, initial animal studies suggest that RE up-regulates components of the insulin signalling cascade. Hernandez et al. (2000) observed that acute RE followed by 1 h of feeding in rats significantly increased glucose uptake when measured 6 h post exercise and in contrast to EE this increase was accompanied with an almost fourfold increase in PI3 kinase activity. The observation that acute RE can significantly lower blood glucose AUC during an OGTT 6 h later is in agreement with the observations of Hernandez et al. (2000) in rats.

In addition recent human studies have demonstrated that acute RE can also activate downstream insulin signalling steps such as Akt phosphorylation (Creer et al. 2005) and p70/p85-S6 protein kinase phosphorylation (Koopman et al. 2006). Interestingly Creer et al. (2005) demonstrated that the up-regulation of Akt phosphorylation following RE is only observed with high muscle glycogen content, indicating that glycogen replenishment is important for up-regulation of the insulin signalling cascade. The evidence from these studies (Creer et al. 2005; Hernandez et al. 2000) and that from the present study appears to indicate that RE followed by carbohydrate feeding to enhance glycogen re-synthesis is beneficial if not crucial to the enhancement of glucose tolerance in the post-exercise period.

In conclusion the present study has shown that an acute bout of RE followed by the ingestion of a carbohydrate–protein hydrolysate beverage can bring about improvements in glucose tolerance. However this is not observed when EE is followed by ingestion of a carbohydrate–protein hydrolysate beverage. We suggest this could be due to the different regulatory mechanisms involved in glucose uptake following acute RE and EE.

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