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Effects of single bout of very high-intensity exercise on metabolic health biomarkers in overweight/obese sedentary men

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ABSTRACT

Purpose. This study aimed to investigate the effects of a single session of sprint interval training (SIT) and a single extended sprint (ES), matched for total work, on metabolic health biomarkers.

Methods. Ten overweight/obese men aged 26.9 ± 6.2 years participated. Following a pre-trial incremental exercise test and SIT familiarization, each participant undertook three 2-day trials in randomized order. On Day 1 participants either undertook no exercise (CON), four maximal 30-s sprints, with 4.5 min recovery between each (SIT), or a single maximal extended sprint (ES) matched with SIT for work done. On Day 2, participants had a fasting blood sample taken, undertook an oral glucose tolerance test to determine insulin sensitivity index (ISI), and had blood pressure measured.

Results. Total work done during exercise did not differ between SIT and ES (61.7 ± 2.9 vs. 61.3 ± 2.8 kJ; $p = 0.741$). Mean power was higher in SIT than ES (518 ± 21 vs. 306 ± 16 W, $p < 0.0005$), resulting in a shorter high-intensity exercise duration in SIT (120 ± 0 vs. 198 ± 10 s, $p < 0.0005$). ISI was 44.6% higher following ES than CON (9.4 ± 2.1 vs. 6.5 ± 1.3 ; $p = 0.022$), but did not differ significantly between SIT and CON (6.6 ± 0.9 vs. 6.5 ± 1.3 ; $p = 0.208$). However, on the day following exercise fat oxidation in the fasted state was increased by 63% and 38%, compared to CON, in SIT and ES, respectively ($p < 0.05$ for both), with a concomitant reduction in carbohydrate oxidation ($p < 0.05$).

Conclusion. A single ES, which may represent a more time-efficient alternative to SIT, can increase insulin sensitivity and increase fat oxidation in overweight/obese sedentary men.

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Abbreviations: BMI, Body mass index; CON, Control trial; ELISA, Enzyme-linked immunoassay; HOMA_{IR}, Homeostasis Model Assessment estimated insulin resistance; ISI, Insulin sensitivity index; OGTT, Oral glucose tolerance test; RER, Respiratory Exchange Ratio; SIT, Sprint interval training; ES, Single extended sprint.

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1. Introduction

The benefits of physical activity in reducing the risk of cardiovascular disease [1,2] and type 2 diabetes [3] have been clearly established. Nevertheless, physical activity levels remain low [4,5], with lack of time often cited as a key barrier to participation [4–6]. As such, interest has grown in time-efficient exercise strategies which reduce the exercise duration required to provide health benefits [7,8].

A number of recent reports have demonstrated that very low volumes of high-intensity exercise in the form of sprint interval training (SIT) – which typically comprises 4–6 30-second ergometer sprints – can induce substantial improvements in both performance [9–11] and health-related [12–15] outcomes. Indices of aerobic (e.g. maximal oxygen uptake, muscle oxidative enzyme activity, time-trial performance and time to fatigue) as well as sprint performance (e.g. peak sprinting power) [9–11,16] have been shown to improve with 2–6 weeks of this type of training. In addition, we [14] and others [12,13,15] have demonstrated that SIT can induce a number of health benefits, including increased insulin sensitivity [12–14], reduced blood pressure [14], and improved vascular function [17]. However, as the beneficial effects of the SIT regime on insulin sensitivity and blood pressure were evident for 24, but not 72, h post-exercise in our study [14], it is unclear whether the observed improvements represented an acute response to the final SIT session or a cumulative, but short-lived, training response to the six session SIT training programme. The first aim of the present study was therefore to address this issue by determining the effects of a single session of SIT on vascular and metabolic risk factors.

It has been proposed that SIT represents a time-efficient approach to obtaining health benefits from exercise, as sessions involve a total of only 2–3 min of high-intensity exercise [18,19]. However, the exercise model used in most SIT studies involve 4 to 6 maximal 30-s sprints on a cycle ergometer, interspersed with 4 to 4.5 min of active recovery between sprints. Thus, SIT sessions still require a total time commitment of 25–30 min, a similar duration to that advocated in conventional physical activity guidelines [20,21]. To determine whether the SIT principle could be adapted into a more time-efficient approach, we sought to investigate whether undertaking a single extended maximal sprint effort (which was matched for the same volume of work done in the 4 × 30-s sprints of an SIT session) could have a similar effect as the SIT protocol on vascular and metabolic risk factors.

2. Methods

2.1. Subjects

Ten men volunteered to participate in this study (age 26.9 ± 6.2 years, height 1.78 ± 0.09 m, body mass 94.2 ± 9.1 kg, BMI 29.9 ± 9.1 kg m⁻², $\dot{V}O_{2peak}$ 42.0 ± 2.4 ml kg⁻¹ min⁻¹) (Mean ± SD). All participants were aged between 18 and 40 years, were overweight or obese (BMI: 25–35 kg m⁻²), and all but one subject were participating in less than two hours per week of structured exercise. This group was chosen for study as they

represent a population who would benefit from increased activity and who might be particularly targeted for a high-intensity exercise intervention of this nature. Exclusion criteria included uncontrolled hypertension (blood pressure >160/90 mm Hg), previous history of coronary heart disease or family history of early cardiac death (<40 years), and diabetes. All participants provided written informed consent prior to commencing the study which was approved by the University of Glasgow Faculty of Biomedical and Life Sciences Ethics Committee.

2.2. Study design

Following preliminary tests (anthropometry, a Wingate test, a maximal ramp-incremental exercise test, and a preliminary SIT session; all described below), participants performed three main experimental trials in a randomized order, each conducted over two days: a control (CON) trial, an SIT trial and a single extended sprint (ES) trial. Trials were conducted approximately one week apart. On Day 1 participants either: (1) completed four 30s “all-out” sprint efforts (i.e. repeated Wingate tests) on a cycle ergometer with a 4.5 min active recovery between efforts (SIT); (2) performed a single extended maximal cycle ergometer sprint matched for total work with the familiarization SIT (ES); or (3) performed no exercise (CON). On Day 2, 18–22 h after exercise in the SIT and ES trials, participants attended the laboratory for metabolic testing, comprising assessment of resting metabolic rate and substrate utilization by indirect calorimetry, an oral glucose tolerance test (OGTT) and measurement of blood pressure. Participants were asked not to undertake planned exercise outwith the lab sessions and maintain their normal lifestyle for the duration of the study. Participants completed a food diary and refrained from alcohol ingestion for 48 h prior to the first OGTT and replicated this diet before all subsequent trials. There were no differences in energy or macronutrient intake between trials on these days. Energy intake on the day prior to the OGTT (i.e. on the exercise days in the SIT and ES trials) was 2291 ± 792 kcal in CON, 2177 ± 842 kcal in SIT, and 2253 ± 742 kcal in ES.

2.3. Anthropometric Assessment

Height, body mass, and skinfold thickness at four sites (biceps, triceps, subscapular and suprailiac) were measured in accordance with the International Standards for Anthropometric Assessment [22]. Percentage fat was calculated by estimating body density from skinfold measures [23] and then by applying the Siri equation [24]. Fat free mass was then determined and used to calculate the appropriate resistance for the SIT sessions.

2.4. Metabolic Testing

Participants arrived at the lab after a 12 h overnight fast. They lay in a supine position for 10 min prior to 25 min of continuous pulmonary gas exchange measurements using a ventilated hood (Oxycon Pro, Jaeger, Germany) to assess resting metabolic rate and rates of fat and carbohydrate oxidation [25]. An OGTT was then performed. A cannula

(Vasofix, Braun, Germany) was inserted into an antecubital vein and was kept patent using sterile saline solution. A baseline blood sample (0min) was taken 10min after cannulation. Subjects then consumed a drink containing 82.5g of glucose monohydrate (equivalent to 75g anhydrous glucose) in 300ml of water, with blood samples taken at 30min intervals thereafter for 120min. Blood samples were collected into potassium EDTA tubes (BD Vacutainer, UK), immediately placed on ice and then centrifuged for 15min at 3000rpm to provide plasma samples. Plasma was then dispensed into 0.5ml aliquots and stored at -80°C until analysis. Blood pressure was measured using an automated blood pressure monitor (Omron HEM705 CP, Omron Healthcare UK Limited, Milton Keynes, UK) which has been validated according to the European Society of Hypertension International Protocol [26]. Three measurements of blood pressure were taken whilst participants had been resting in a supine position for over an hour and the lowest of these values was used for analysis.

2.5. Plasma Analyses

Plasma samples were analyzed with commercially available kits to determine glucose, triglycerides, total and HDL cholesterol (all ABX Pentra, Montpellier, France) using a semi-automatic analyzer (Cobas Mira Plus, ABX Diagnostics, France). Each sample was analyzed in duplicate and a single analyzer run was used for each subject. Insulin concentration was determined using a commercially available enzyme-linked immunoassay (ELISA) (Mercodia AB, Uppsala, Sweden). Each individual's samples were analyzed in duplicate for insulin concentration on a single plate. Insulin sensitivity was calculated using the insulin sensitivity index (ISI), as described by Matsuda and DeFronzo [27]. This calculation uses the fasting plasma glucose (in mg dl^{-1}) and plasma insulin (in mU l^{-1}) and the average plasma glucose and insulin values over the 30, 60, 90 and 120min from an OGTT i.e. $10,000 / \sqrt{[(\text{fasting glucose} \times \text{fasting insulin}) \times (\text{mean glucose during OGTT} \times \text{mean insulin during OGTT})]}$. In addition, Homeostasis Model Assessment estimated insulin resistance (HOMA_{IR}) was calculated using fasting insulin (mU l^{-1}) $\times$ fasting glucose (mmol l^{-1}) / 22.5 [28].

2.6. Exercise Tests

All exercise tests were conducted on a computer-controlled cycle ergometer (Excalibur Sport, Lode, Gronigen, Netherlands). Verbal encouragement was provided throughout all exercise tests to promote maximal efforts from participants.

2.6.1. Wingate Test

The Wingate test involved the subject sprinting “all-out” against a fixed braking force (0.065kg per kg of fat free mass) for 30s. Braking forces were assigned according to fat free mass rather than body mass as it has been shown that this leads to greater peak power output generation in overweight and obese groups [29]. One second prior to the load being applied (via Wingate Software V1, Lode, Groningen, The Netherlands), participants were instructed to start sprinting and began accelerating to overcome the inertia that would be required to pedal maximally when the loading was

applied. They continued to pedal maximally for the 30s duration of the sprint. Participants warmed-up and warmed-down for 4min before and after the sprint at a constant work rate of 30W.

2.6.2. Maximal Ramp-Incremental Exercise Test

The maximal ramp-incremental exercise test involved a progressive increase in work rate of 25W min^{-1} (ramp-rate) until the limit of tolerance, defined as the point where participants could not maintain a pedaling cadence of $>50\text{ rev min}^{-1}$ regardless of verbal encouragement. Participants cycled at 20W for 4min before and after the ramp incremental phase of the test. Breath-by-breath pulmonary gas exchange was measured using a metabolic cart (Oxycon Pro, Jaeger, Germany) as described in detail previously [14]. Peak oxygen uptake ($\text{VO}_{2\text{peak}}$) was determined as the average VO_2 from the final 20s of the incremental test. Peak power (P_{peak}) was recorded as the power output achieved at the end of the test.

2.6.3. Sprint interval training (SIT) protocol

The SIT required completion of repeated Wingate tests. The protocol has been described in detail previously [14]. Briefly, after a 4-min warm-up at a work rate of 30W, participants completed four 30s “all-out” sprint efforts (i.e. Wingate Tests) against a constant braking force of 0.065kg per kg of fat free mass. There was a fixed active recovery period of 4.5min (at 30W) between each sprint and a 4-min cool-down at 30W was performed on completion of the 4 sprints. Arterialized capillary blood samples were taken from the thumb following the warm-up and then 30s and 4min after each sprint in order to determine lactate concentrations using an Analox GM7 analyzer (Analox Instruments, Hammersmith, U.K.).

A preliminary SIT session was undertaken ~7 days prior to the main experiment trials for two reasons: (1) to familiarize participants with the SIT protocol and (2) to determine the volume of work done by each individual in the SIT session. The latter was required to enable matching of the work done in the ES and SIT main trials as trials were conducted in a random order and therefore some participants undertook the ES trial before the SIT trial. There was no significant differences in work done between the preliminary SIT and the SIT main trial (62.1 ± 3.1 vs. $62.2 \pm 2.9\text{kJ}$; $p=0.344$).

2.6.4. Extended sprint (ES) protocol

The ES involved the performance of the same volume of work (kJ) as that completed in the SIT protocol, but in a single bout. As the load applied in the SIT protocol (0.065kg per kg of fat free mass) would be unsustainable for a prolonged period (resulting in an inappropriately low cadence, $<50\text{ RPM}$), a lower braking force was determined for the ES, based on P_{peak} from the maximal ramp-incremental exercise test. The cycle ergometer was set in cadence-dependent mode (i.e. where $\text{Power} = \text{Linear factor} \times \text{cadence}^2$), and the linear factor (flywheel resistance) was calculated as: $\text{Linear factor} = P_{\text{peak}} - (2 \times \text{ramp-rate}) / 80^2$ (Murgatroyd, Ferguson, Cannon, Bowen, Wylde, Porszasz, Rossiter, unpublished data). We considered that this method of calculating the braking force would allow these participants to provide a maximum effort in the ES by allowing the maintenance of an appropriate cadence ($>50\text{rpm}$) throughout. Similar to the SIT protocol, participants

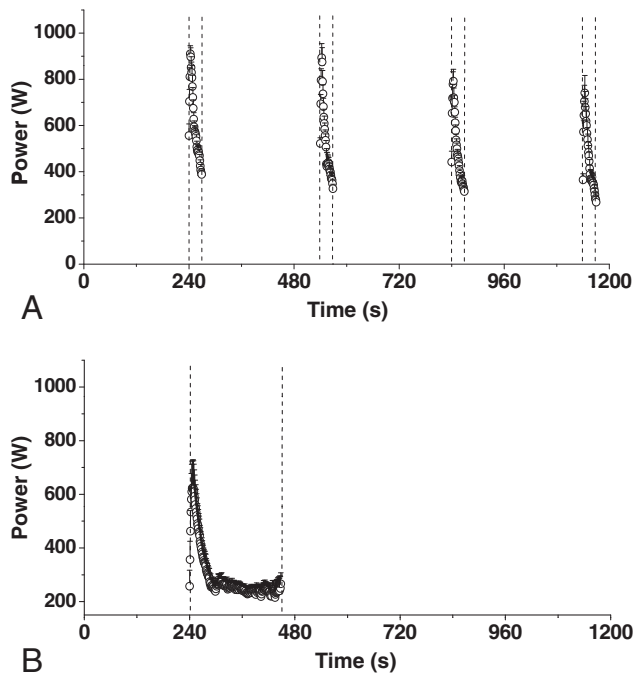


Fig. 1 – Power output profiles for sprint interval training (SIT) (Panel a) and extended sprint (ES) (Panel b) sessions. Data presented as mean±SEM. Peak power, mean power and work done during the sprints in the SIT and ES sessions are shown in Table 1.

were encouraged to pedal in an “all-out” manner throughout the ES until they completed the required work (i.e. the same work as that accomplished in the four 30-s sprints of the preliminary SIT session). Capillary blood samples for lactate analysis were taken following the warm-up and 30s and 4min after the ES. Participants warmed-up and cooled-down for 4min before and after the sprint at a constant work rate of 30W.

2.7. Statistical Analysis

Statistical analysis was performed using Statistica (version 6.0; StatSoft, Tulsa, OK, USA) and Minitab (version 13.1; Minitab, State College, PA, USA). A priori power calculations based on our previous data [14], indicated that, with 10

subjects, the study had 80% power to detect a 25% difference in ISI between trials at $p < 0.05$.

Prior to analysis, all data were tested for normality, using the Ryan Joiner test. No data required transformation: raw data values were used in all analyses. Differences between CON, SIT and ES measurements were determined using one-way repeated measures ANOVA with post-hoc Tukey tests. Data are presented as means±SEM, unless otherwise stated and statistical significance was set at $p < 0.05$ level.

3. Results

3.1. Exercise measurements

All participants successfully completed the ES protocol and 9 of the 10 participants successfully completed 4 sprints in the SIT protocol. One participant felt nauseous during the SIT session and only completed 3 of the 4 sprints. His data have been retained in the analyses. Fig. 1 shows power output profiles and Table 1 shows power output, work done, and lactate concentrations for each of the 4 sprints in the SIT and for the ES. Peak power (average peak power for 4 sprints in SIT trial) was higher in SIT compared with ES (888 ± 51 vs. 754 ± 47 W, $p = 0.001$). Total work done in both the SIT and ES trials was the same (61.7 ± 2.9 vs. 61.3 ± 2.8 kJ; $p = 0.741$). However, as mean power in SIT was higher than in ES (518 ± 21 vs. 306 ± 16 W, $p < 0.0005$) high-intensity exercise duration was 65% shorter in the SIT session (120 ± 0 vs. 198 ± 10 s, $p < 0.0005$), although the total time commitment required for SIT was considerably longer (i.e. 20min vs. ~3.3min, excluding warm-up and cool-down) due to the active recovery required between sprints. Peak blood lactate concentrations were higher in SIT than in ES (10.93 ± 0.64 vs. 7.38 ± 0.66 mmol l^{-1} ; $p < 0.005$).

3.2. Blood variables

Insulin and glucose responses, and ISI values in the control, SIT and ES are shown in Fig. 2, with fasting insulin and glucose concentrations and areas under the glucose and insulin curves shown in Table 2. Compared to CON, ISI was significantly higher (by 44.6%, $p = 0.022$) and HOMA_{IR} was significantly lower (by 31.8%, $p = 0.043$) in ES. In contrast, neither ISI (1.5% higher, $p = 0.208$) nor HOMA_{IR} (18% lower, $p = 0.556$) was significantly different from CON in the SIT trial. There were no significant

Table 1 – Performance data including sprint durations, power outputs and lactate concentrations for the four individual sprints and the “time-efficient” extended sprint (ES).

	SIT				ES
	Sprint 1	Sprint 2	Sprint 3	Sprint 4	
Duration of sprint (s)	30	30	30	30	198±10
Peak power (W)	1004±44	977±65	868±68	767±51	754±47
Mean power (W)	600±25	527±25	490±19	458±21	306±16
Work done (kJ)	17.9±0.8	15.8±0.8	14.3±0.6	13.7±0.6	61.3±2.9
Pre-lactate (mmol l^{-1}) ^a	1.08±0.10	7.54±0.37	10.23±0.36	10.73±0.49	0.94±0.11
Post-lactate (mmol l^{-1}) ^a	5.09±0.35	9.39±0.29	10.37±0.43	10.93±0.64	7.38±0.66

Data are presented as mean±SEM. N=10 except for lactate measurements^a, which are for n=8.

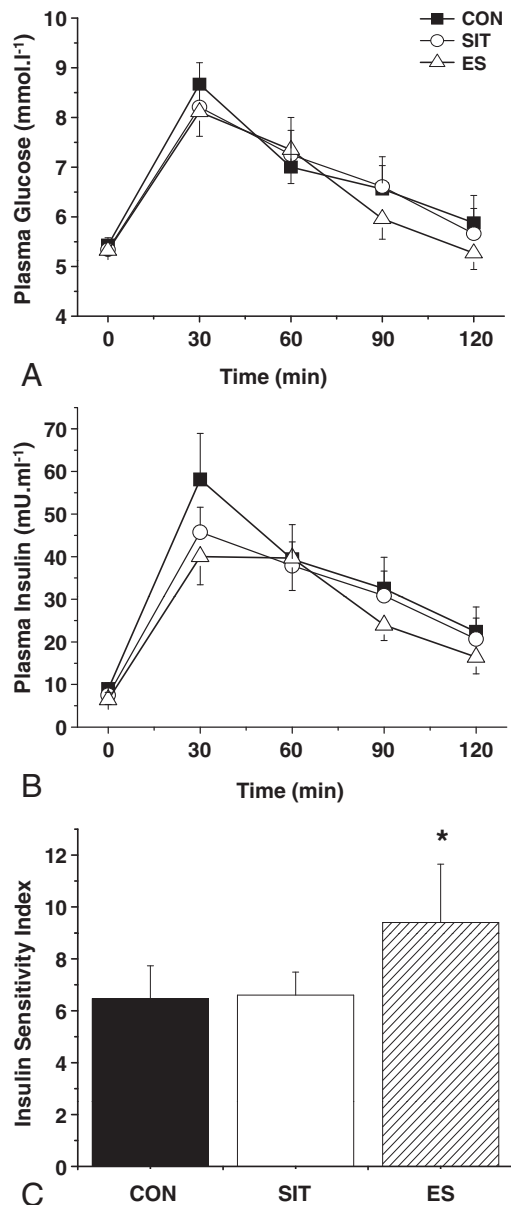


Fig. 2 – Plasma glucose (Panel a) and insulin (Panel b) responses to a 75 g OGTT, and insulin sensitivity index (Panel c), in Control (CON), sprint interval training (SIT) and extended sprint (ES) trials. Data presented as mean $\pm$ SEM. Area under curve (AUC) values for plasma glucose and insulin during the oral glucose tolerance test (OGTT) are shown in Table 2.

differences between trials in fasting glucose, insulin, triglyceride, total cholesterol or HDL cholesterol concentrations (Table 2). Similarly, glucose AUC and insulin AUC values did not differ significantly between trials (Table 2).

3.3. Resting Energy Expenditure and Substrate Utilization

Resting energy expenditure and substrate utilization data in the three trials are shown in Table 2. Resting energy expenditure did not differ between trials ($p > 0.05$), but RER values were significantly lower than CON in both SIT and ES

($p < 0.05$ for both). Consequently, compared to CON, resting fat oxidation was 38% higher in ES ($p = 0.028$) and 63% higher in SIT ($p = 0.003$). Reciprocally, carbohydrate oxidation was 50% lower in ES ($p = 0.023$) and 75% lower in SIT ($p = 0.004$) than CON.

3.4. Blood Pressure

Blood pressure values are shown in Table 2. There were no significant differences in systolic or diastolic blood pressure between trials.

4. Discussion

The main findings from this study were that the day after a single extended cycle ergometer sprint of ~200s duration, the insulin sensitivity index had increased by almost half and HOMA_{IR} was reduced by almost a third in overweight and obese men. However, an SIT session of four 30s cycle ergometer sprints, matched for total work with the extended sprint, did not have a significant effect. However, both ES and SIT altered substrate metabolism on the day following exercise, increasing fat oxidation and reducing carbohydrate oxidation compared to CON. Thus, the effect of two weeks of SIT on insulin sensitivity observed previously [12–15] does not seem to be replicated in response to a single SIT session, suggesting that the accumulation of several SIT sessions may be necessary to elicit this effect. In contrast, ~200s of continuous maximal exercise, which was well tolerated by participants, was shown to significantly increase insulin sensitivity. Thus this study provides an exciting first step in determining the minimum duration of an exercise session required to benefit metabolic health and indicates that time-efficient high-intensity exercise may be sufficient to elicit improvement in health-related outcomes.

There is a growing body of evidence to suggest that short-term SIT interventions can be used as a mode of training to induce health benefits in both normal weight, moderately fit individuals [12] and in overweight to obese, unfit sedentary individuals [13–15]. It has consistently been reported that two weeks of SIT enhances insulin sensitivity [12–15] with some reports showing these improvements persisting 2–3 days post intervention [12,13]. However, data from Whyte et al. (2010) suggested that these improvements were transient, with increased insulin sensitivity at 24h post, but not at 72h post intervention [14]. Similarly, Richards et al. (2010) found no increase in insulin sensitivity 72h after completing a single SIT session of 4 $\times$ 30s “all-out” sprints [13]. The present study extends these findings by showing that insulin sensitivity is not improved 18–22h following a single SIT session. Thus, taken together, the available evidence suggests that the improvement to insulin sensitivity in response to SIT represents a training adaptation, albeit one which may be short-lived, rather than an acute response to a single exercise session.

However, in contrast, performing the work of 4 $\times$ 30s maximal sprints in a single extended sprint of ~200s did substantially improve insulin sensitivity. The underlying mechanisms responsible for the improvement in insulin sensitivity after ES but not SIT are not clear. Speculatively, this may be a consequence of differing energy fluxes and

Table 2 – Blood biomarkers, energy expenditure, substrate utilization, and blood pressure measurements for Control (CON), Sprint Interval Training (SIT) and the “time-efficient” extended sprint (ES).

Variable	CON	SIT	ES	CON vs. SIT P-value	CON vs. ES P-value
Insulin/glucose variables					
Fasting glucose (mmol l ⁻¹)	5.43±0.15	5.34±0.16	5.32±0.10	0.791	0.738
Fasting insulin (mU l ⁻¹)	8.92±1.43	7.49±0.69	6.46±1.16	0.926	0.138
Glucose AUC (mmol l ⁻¹ min)	462.3±27.6	456.2±27.4	440.2±22.4	0.831	0.325
Insulin AUC (mU l ⁻¹ min)	2353±355	2084±252	1850±278	0.736	0.185
Insulin sensitivity index	6.5±1.3	6.6±0.9	9.4±2.1	0.480	0.022
HOMA _{IR}	2.2±0.4	1.8±0.2	1.5±0.3	0.279	0.043
Lipid variables					
Fasting triglycerides (mmol l ⁻¹)	1.47±0.31	1.28±0.14	1.16±0.11	0.660	0.345
Fasting HDL cholesterol (mmol l ⁻¹)	1.12±0.08	1.08±0.08	1.10±0.08	0.544	0.786
Fasting total cholesterol (mmol l ⁻¹)	4.83±0.54	4.73±0.50	4.87±0.51	0.845	0.958
Metabolic rate and substrate utilisation^a					
RER	0.81±0.01	0.73±0.01	0.75±0.01	0.002	0.015
Energy expenditure (kcal min ⁻¹)	1.47±0.05	1.53±0.04	1.48±0.04	0.348	0.936
CHO oxidation (g min ⁻¹)	0.12±0.02	0.03±0.01	0.06±0.01	0.004	0.023
Fat oxidation (g min ⁻¹)	0.08±0.03	0.13±0.01	0.11±0.01	0.003	0.028
Blood pressure variables					
Systolic BP (mmHg)	127±4	125±4	122±3	0.410	0.107
Diastolic BP (mmHg)	70±4	67±3	64±4	0.146	0.172

Data are presented as mean±SEM. N=10 except for metabolic rate and substrate utilization measurements^a, which are for n=7. P-values are for one-way repeated measures ANOVA with post-hoc Tukey tests. AUC: area under curve; BP: blood pressure; CHO: carbohydrate; HOMA_{IR}: Homeostasis model estimated insulin resistance; RER: respiratory exchange ratio.

substrate utilization during the two exercise bouts. The metabolic nature of SIT is such that there are oscillations in energy requirements, whereby during the sprint there is a high demand for ATP, whilst during the recovery periods ATP demand is lessened. During the first 30s bout, ATP demand is primarily met via phosphocreatine breakdown and glycolysis, with oxidative metabolism contributing about ~30% to the overall of energy requirements [30]. In later bouts, the proportional contribution from oxidative phosphorylation is progressively increased, with less reliance on phosphocreatine hydrolysis or anaerobic glycolysis [31–33]. In the ES trial, without these recovery periods between bouts, it is likely that the shift towards oxidative metabolism energy supply would have been more pronounced. Indeed it has been shown that the aerobic contribution to maximal exercise increases with the duration of the activity (e.g. [31–35]): for athletics events such as 800m and 1500m which have durations which lie between those of the ES in the current study, aerobic contributions are ~60% and ~80% respectively [35]. As such, it is reasonable to assume that there would be greater energy flux through the mitochondria during the ES compared with SIT, and as high rates of energy flux through mitochondria have been associated with greater insulin sensitizing effects [36]: this may contribute to the differential effects of SIT and ES on insulin sensitivity. In addition, during the ES, there are greater sustained contraction cycles relative to SIT (as the sprinting period is longer), which could feasibly cause greater concentrations in both intracellular/myoplasmic Ca²⁺ and AMP concentrations [37]. In turn these could act on Ca²⁺/calmodulin-dependent protein kinase and AMP activated protein kinase respectively, mediating activation of GLUT4 and therefore indirectly increasing insulin sensitivity [38]. These are speculative suggestions which warrant further investigation.

Whole body fat oxidation was increased the day after exercise in both the SIT and ES trials by 63% and 38% respectively. This shift in substrate utilization is likely to reflect the high glycogen use occurring during maximal exercise and the increase in post-exercise fat oxidation necessary to facilitate glycogen repletion [39]. Interestingly, while both SIT and ES increased fat oxidation, only the latter improved insulin sensitivity, suggesting dissociation between the mechanisms by which exercise induces these two effects, at least over the short term. We previously reported that resting fat oxidation rate was increased for ~24h following completion of an SIT training programme [14]. However, it is of interest that the increase in fat oxidation observed in response to 2 weeks of SIT training, at 18% [14], was lower than that observed in the present acute study. This may indicate that the increase in resting fat oxidation seen in response to a single very high-intensity exercise session in an untrained person is attenuated in individuals habituated to this type of exercise. Further study investigating post-exercise fat oxidation responses in the same individuals after a single session and in response to a period of very high intensity exercise training is needed to confirm this suggestion. However, this observation does highlight the caution required when attempting to extrapolate findings from responses to single exercise sessions to effects over the long term.

One of the major claims made in support of SIT is that it is time-efficient [18,19]. However, the most common SIT format – four to six 30s sprints with 4 to 4.5min recovery – does not require appreciably less time to complete than a conventional moderate-intensity physical activity programme undertaken in line with present guidelines [20,21]. The present data indicate that an exercise session involving ~200s of high-intensity exercise, requiring a total time commitment of only ~10–12min (including warm-up and

warm-down) can induce improvements in insulin sensitivity and increases in fat oxidation comparable to 60–90 min of moderate intensity exercise [40,41]. This could potentially have important implications for physical activity prescription in individuals who lack the time for traditional exercise regimes, providing a viable alternative to those prepared to undertake sustained short-term high-intensity all-out exercise. However, it is important to recognize that while the present study provides ‘proof-of-principle’ that time-efficient high intensity exercise is beneficial, trials are still needed to evaluate the effectiveness (i.e. taking into account both efficacy and compliance/drop-out) of such a regime over the long-term in ‘real-world’ settings.

Thus, a key challenge remains to translate this research from a controlled laboratory setting, to a practical model which can be recommended to the public. Safety is a key concern as the risk of an acute cardiovascular event during exercise, especially in older individuals, or those with pre-existing cardiovascular disease, is greater with increasing intensity of exercise [42–44]. However, several studies have indicated that “at risk” groups can both tolerate and successfully improve health-related variables whilst using high-intensity interval training [14,15,45–48]. Nevertheless, it is likely that low-volume, high-intensity exercise may be unsuitable for certain population groups. However, the population studied in the present report – overweight and obese inactive individuals under 40 years – represent a large portion of the population and would seem an ideal group to target for this type of exercise approach. These individuals are at increased risk of future cardiovascular and metabolic disease, but are at relatively low risk of an acute vascular event.

In addition, cycle ergometer protocols used in the present study require the use of specialized equipment (e.g. Lode ergometer and Wingate software) to allow an almost instantaneous switch from unloaded pedaling to applying a high braking force. It is the application of this high braking force which ultimately provides the characteristic Wingate power profile of high peak power within the first few seconds and a progressive decline in power thereafter. It is unclear whether this sprint power profile is needed to elicit the metabolic benefits seen or whether general leisure facility stationary bikes could perform this as effectively.

Furthermore, all trials were supervised and subjects were given considerable verbal encouragement. As such, all but one subject was able to complete the 4 sprinting bouts (one subject completed 3 sprints) during the SIT trial and everyone completed the ES in an “all-out” fashion. However, in order to do so, it required a high degree of motivation and without both motivation and encouragement, the magnitude of change in insulin sensitivity and fat oxidation found in the present study may not have been as large.

In summary, the present report has shown that a single extended sprint of ~200s substantially improved insulin sensitivity and increased fat oxidation on the day following exercise in inactive overweight/obese men. A session of 4 × 30s maximal sprints increased fat oxidation but did not improve insulin sensitivity. Further research is needed to take this form of exercise from a controlled-lab setting and translating this concept of low-volume, high-intensity exercise into the public domain. Incorporating such concepts into

exercise classes and establishing whether assigning an unsupervised low-volume, high-intensity training intervention can safely provide the same benefit to that of traditional moderate intensity training are of key importance to establish the “real-world” efficacy of this type of training. It is therefore premature to advocate to the general public that ~3 min of exercise can provide health benefits. Nevertheless, this study provides proof-of-principle, that a very-short duration, high-intensity exercise session can induce substantial metabolic benefits in a population of inactive, overweight/obese men.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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