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Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men

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Abstract

The aim of this study was to investigate the effects of very high intensity sprint interval training (SIT) on metabolic and vascular risk factors in overweight/obese sedentary men. Ten men (age, 32.1 ± 8.7 years; body mass index, 31.0 ± 3.7 kg m^{-2}) participated. After baseline metabolic, anthropometric, and fitness measurements, participants completed a 2-week SIT intervention, comprising 6 sessions of 4 to 6 repeats of 30-second Wingate anaerobic sprints on an electromagnetically braked cycle ergometer, with 4.5-minute recovery between each repetition. Metabolic, anthropometric, and fitness assessments were repeated post-intervention. Both maximal oxygen uptake (2.98 ± 0.15 vs 3.23 ± 0.14 L min^{-1}, $P = .013$) and mean Wingate power (579 ± 24 vs 600 ± 19 W, $P = .040$) significantly increased after 2 weeks of SIT. Insulin sensitivity index (5.35 ± 0.72 vs 4.34 ± 0.72, $P = .027$) and resting fat oxidation rate in the fasted state (0.13 ± 0.01 vs 0.11 ± 0.01 g min^{-1}, $P = .019$) were significantly higher and systolic blood pressure (121 ± 3 vs 127 ± 3 mm Hg, $P = .020$) and resting carbohydrate oxidation in the fasted state (0.03 ± 0.01 vs 0.08 ± 0.02 g min^{-1}, $P = .037$) were significantly lower 24 hours post-intervention compared with baseline, but these changes were no longer significant 72 hours post-intervention. Significant decreases in waist (98.9 ± 3.1 vs 101.3 ± 2.7 cm, $P = .004$) and hip (109.8 ± 2.2 vs 110.9 ± 2.2 cm, $P = .017$) circumferences compared with baseline were also observed after the intervention. Thus, 2 weeks of SIT substantially improved a number of metabolic and vascular risk factors in overweight/obese sedentary men, highlighting the potential for this to provide an alternative exercise model for the improvement of vascular and metabolic health in this population.

1. Introduction

Despite the widespread acceptance that undertaking physical activity is associated with a reduced risk of many diseases, participation in physical activity remains low (eg, Morrow et al [1]). For some time now, the American College of Sports Medicine has advocated that adults should accumulate at least 30 minutes of moderate-intensity exercise on most days of the week to attain health benefits [2]. However, more recently, the American College of Sports Medicine guidelines have placed greater emphasis on shorter-duration (ie, a minimum of 20 minutes), higher-intensity exercise to be undertaken on a minimum of 3 times per week [3].

This may be an important first step in increasing physical activity levels, as lack of time has regularly been shown to be a major barrier to physical activity and has been associated with low physical activity levels (eg, Reichart et al [4], Trost et al [5], and Brownson et al [6]). However, there is still much debate surrounding the optimal intensity, duration, and volume of exercise that are required to provide the most favorable impact on health. Several studies have compared the effects of energy expenditure–matched low- and high-intensity exercise on indices of glucose control; and although not unequivocal [7,8], a number of reports have demonstrated greater improvements to insulin sensitivity [9-11] at the higher intensity. It has also been recently reported that higher-intensity exercise induces greater changes to body composition than energy-matched lower-intensity exercise [12].

Therefore, the available evidence suggests that higher-intensity exercise may offer a more time-efficient strategy for improving metabolic health than conventional moderate-intensity exercise programs. The next step is to identify exercise regimens with suitably low durations that will

This study was approved by the Faculty of Biomedical and Life Sciences Ethics Committee for Non-Clinical Research Involving Human Subjects.

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negate time from being a barrier to exercise and thus facilitate increases in physical activity levels. As such, there has recently been some speculation that a particular form of very high intensity, and thus potentially very low duration, exercise known as *sprint interval training* (SIT) may provide health benefits [13,14]. This form of training involves repeated 30-second "all-out" sprints against a fixed load on a cycle ergometer with a recovery period of 4 minutes between repeats. As little as 3 weeks of this training has been shown to improve maximum oxygen consumption ($\dot{V}O_{2max}$) [15] and endurance performance [16,17] in recreationally active individuals. Some of the mechanisms underlying these improvements have been demonstrated, with changes in both glycolytic and oxidative enzyme content and activity [18,19]. Intriguingly, several other factors that are related to health, as well as fitness, have been shown to improve after this form of training. For example, after only 1 week of SIT, skeletal muscle glucose transporter 4 (GLUT4) content significantly increased [20]. Similarly, β-hydroxyacyl coenzyme A dehydrogenase activity, which catalyzes a key rate-limiting step in fat oxidation, also significantly increased after 6 weeks of SIT [15]. Furthermore, Rakobowchuk and colleagues [21] found that 6 weeks of SIT improved peripheral vascular structure and function. In addition, a recent study reported that insulin sensitivity was increased in a group of young (age, 21 ± 2 years), fit ($\dot{V}O_{2max}$, 48 ± 9 mL kg^{-1} min^{-1}), normal-weight (body mass index [BMI], 23.7 ± 3.1 kg m^{-2}) men after a 2-week SIT intervention [22].

However, it is not known whether this form of exercise can be tolerated by overweight/obese sedentary individuals with low fitness levels and, moreover, whether the risk marker changes seen in young, fit men [22] also occur in this population as a result of SIT. Therefore, the purpose of this study was to investigate the effects of 2 weeks of SIT on a cluster of health-related physiologic markers in overweight/obese sedentary men. We hypothesized that 2 weeks of SIT would improve these vascular and metabolic risk markers.

2. Methods

2.1. Subjects

Ten men volunteered to participate in this study (age, 32.1 ± 8.7 years; height, 1.76 ± 0.07 m; body mass, 93.9 ± 12.8 kg; BMI, 31.0 ± 3.7 kg m^{-2}, $\dot{V}O_{2max}$, 2.98 ± 0.48 L min^{-1}) (mean ± SD). They were included on the basis that they were aged between 18 and 40 years, were overweight or obese (BMI, 25-35 kg m^{-2}), and were sedentary (participating in <1 h/wk of structured exercise, as assessed by the International Physical Activity Questionnaire [23]). Exclusion criteria included smoking, 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 before commencing the study as approved by the Faculty of Biomedical and Life Sciences Ethics Committee for Non-Clinical Research Involving Human Subjects.

2.2. Study design

All volunteers participated in a familiarization session, 3 experimental trials, and 6 training sessions. The first visit to the laboratory involved basic anthropometric measurements and familiarization with the exercise tests (Wingate anaerobic test and a ramp incremental exercise test on a cycle ergometer). One week after the familiarization session, participants returned to complete the battery of baseline tests in a single session. The baseline testing session incorporated resting metabolic and pulse wave velocity (PWV) measurements. Within 1 week of the baseline testing session, the participants commenced the 2 week SIT intervention. Post-intervention metabolic measurements and measurements of PWV were conducted approximately 24 and 72 hours after the final training session to obtain information on both the acute and chronic effects of SIT. Post-intervention anthropometric assessment was performed 24 hours after the final SIT session, and the post-intervention exercise tests were performed 72 hours after the final SIT session. Throughout the intervention, subjects were asked to refrain from consuming alcohol and were encouraged to continue consuming their normal diet and maintain their typically sedentary behavior outwith the training period. Subjects recorded a 48-hour food diary before baseline testing and replicated this before subsequent tests. There were no significant differences in energy, carbohydrate, protein, or fat intake between tests.

2.3. Anthropometric assessment

All anthropometric measurements were conducted in accordance with the International Standards for Anthropometric Assessment [24]. Body mass was measured to the nearest 0.05 kg using a beam balance scale (Avery, Royston, England). Height was measured to the nearest 0.1 cm with a stadiometer (Invicta Plastics, Leicester, England). Waist and hip circumferences were measured to the nearest 0.1 cm.

2.4. Metabolic testing

Subjects arrived at the laboratory after a 12-hour overnight fast. They lay in a supine position for 10 minutes before continuous measurement of pulmonary gas exchange for a 25-minute period using a ventilated hood (Oxycon Pro; Jaeger, Hoechberg, Germany) to allow assessment of metabolic rate and rate of fat and carbohydrate oxidation via indirect calorimetry [25]. A cannula (Vasofix; Braun, Melsungen, Germany) was inserted into an antecubital vein, and the baseline blood sample was taken 10 minutes after cannulation. Subjects subsequently underwent an oral glucose tolerance test (OGTT). Briefly, they consumed a drink containing 75 g of anhydrous glucose in 300 mL of water; and blood samples were taken at 30-minute intervals

for 120 minutes. Blood samples were collected into potassium EDTA tubes (Vacutainer; BD, Oxford, United Kingdom) and immediately placed on ice. Within 15 minutes of the samples being collected, they were centrifuged for 15 minutes at 3000 rpm. Plasma was then dispensed into 0.5-mL aliquots and stored at −80°C until analysis. Commercially available kits were used to determine glucose, triglycerides, total and high-density lipoprotein (HDL) cholesterol (all ABX Pentra, Montpellier, France), and nonesterified fatty acids (NEFA) (Wako Chemicals, Neuss, Germany) using a semiautomatic analyzer (Cobas Mira Plus; ABX Diagnostics, Montpellier, France). A single analyzer run was used for each subject, and each sample was analyzed in duplicate. Insulin was determined using a commercially available enzyme-linked immunoassay (Mercodia, Uppsala, Sweden). Each subject's samples were analyzed for insulin concentration on a single plate with each sample again analyzed in duplicate. Insulin sensitivity was calculated using the insulin sensitivity index, as described by Matsuda and DeFronzo [26]. This calculation uses the fasting plasma glucose (in milligrams per deciliter) and plasma insulin (in milliunits per liter) and the average plasma glucose and insulin values over the 30, 60, 90, and 120 minutes from an OGTT, that is, 10 000/ √[(fasting glucose × fasting insulin) × (mean glucose during OGTT × mean insulin during OGTT)].

2.5. PWV and blood pressure

Subjects lay supine for at least 30 minutes before all PWV and blood pressure measurements. Blood pressure was measured using an automated blood pressure monitor (Omron HEM705 CP; Omron Healthcare, Milton Keynes, United Kingdom) that has been validated according to the European Society of Hypertension International Protocol [27]. On each occasion, 3 measurements of blood pressure were taken; and the lowest of these values was used for analysis. Once blood pressure measurements were conducted, carotid-femoral PWV was performed using the Complior SP system (Artech Medical, Pantin, France) to provide an index of arterial stiffness. Pulse transit time was determined using pressure transducers placed over the carotid and femoral pulses with the Complior software (Artech Medical) establishing the propagation time from the carotid to femoral artery. The transit distance was measured as the superficial distance between the 2 pressure transducers. Thus, the PWV was calculated as the transit distance divided by the transit time. Measurements of PWV were repeated 6 times on each occasion, and the mean of these values was used for analysis.

2.6. Exercise tests

Subjects performed 2 exercise tests: a Wingate anaerobic test and a maximal incremental exercise test.

The Wingate anaerobic test involved the subject sprinting "all-out" against a fixed braking force (0.065 kg per kg of fat-free mass [FFM]) for 30 seconds on a computer controlled cycle ergometer (Excalibur Sport; Lode, Groningen, Netherlands). Subjects warmed up and warmed down for 4 minutes before and after the sprint at a constant work rate of 30 W.

The maximal incremental exercise test involved a ramp increase of 15 to 30 W min^{-1} on the cycle ergometer (Excalibur Sport) until volitional exhaustion (tests were terminated when participants could not maintain a pedaling cadence of >50 rpm). Subjects cycled at 20 W for 4 minutes before and after the ramp incremental phase of the test. Subjects were not told when the ramp increase began to avoid participants knowing the duration of the test and thus attempting to better that target in the post-test. Throughout the test, participants respired through a rubber facemask connected to a bidirectional turbine volume sensor (with the turbine having a resistance of <0.1 kPa L^{-1} s^{-1} at a flow rate of 15 L s^{-1}) for measurement of respiratory volume and flow that was calibrated using a fixed volume (3-L syringe; Hans Rudolph, Kansas City, MO) over a range of flow profiles. Respired CO_2 and O_2 concentrations were measured every 20 milliseconds by O_2 (chemical fuel cell) and CO_2 (infrared absorption) analyzers (Oxycon Pro), calibrated with one precision-analyzed gas mixture and room air to span the concentration range observed during exercise. The time delay between the volume and gas concentration signals was measured by abruptly switching between delivery of high-CO_2 low-O_2 calibration gas and room air to the system via a low–dead-space solenoid-operated valve. The measured volume and time-aligned concentration signals were processed online for breath-by-breath display of ventilatory and gas exchange variables. Verbal encouragement was given throughout the test.

2.7. Training protocol

The SIT intervention was modeled on recent studies led by Gibala at McMaster University, Canada (eg, Burgomaster et al [16]). The 6 training sessions consisted of repeated 30-second "all-out" sprint efforts (ie, Wingate anaerobic tests) on an electromagnetically braked cycle ergometer (Excalibur Sport) with a fixed recovery period of 4.5 minutes between each sprint. During each sprint, the braking force was kept constant at 0.065 kg per kg of FFM; and during the recovery period, subjects exercised at 30 W. Braking forces were assigned according to FFM rather than body mass because it has been shown that this leads to greater peak power output generation in overweight and obese groups [28]. Fat-free mass was estimated from skinfold thickness measurements (bicep, tricep, subscapular, and suprailiac) and applying Durnin and Womersley's [29] and Siri's [30] equations to calculate body density and total body fat, respectively. Total body fat was then subtracted from the total body mass to gain FFM. The 6 sessions were completed over a 2-week period, with 1 to 2 days of recovery between each session. Four repeated sprints were completed on sessions 1 and 2, 5

repeated sprints on sessions 3 and 4, and finally 6 sprints on sessions 5 and 6. Subjects were given verbal encouragement during each sprint.

2.8. Statistical analysis

Statistical analysis was performed using Statistica (version 6.0; StatSoft, Tulsa, OK) and Minitab (version 13.1; Minitab, State College, PA). Before analysis, all data were tested for normality (Ryan-Joiner). If the data differed substantially from a normal distribution, they were transformed using the appropriate factor determined from Box-Cox analysis. For parameters only measured once post-training, pre- vs post-intervention comparisons were made using paired Student *t* tests. Differences between baseline, 24-hour post, and 72-hour post measurements were determined using 1-way repeated-measures analysis of variance with post hoc Tukey tests. Statistical significance was accepted at $P < .05$ level. Data are presented as means ± SEM, unless otherwise stated.

3. Results

3.1. Performance measurements

These measurements are shown in Table 1. The $\dot{V}O_{2max}$ was significantly increased after the intervention in both absolute terms (by 8.4%, $P = .013$) and relative to body mass (by 9.5%, $P = .015$). Maximum heart rate achieved during the incremental tests did not differ between baseline and post-intervention measurements (183 ± 4 vs 184 ± 5 beats per minute, $P = .866$). Furthermore, mean power during the 30-second Wingate anaerobic test increased by 3.6% ($P = .040$); but peak power during the Wingate test did not change significantly ($P = .195$).

3.2. Anthropometric measurements

There was a tendency for body mass to be lower after the SIT intervention ($P = .055$); and waist (by 1.1%, $P = .004$) and hip circumferences (by 1.0%, $P = .017$) were modestly but significantly reduced post-intervention (Table 1).

3.3. Blood variables

Insulin and glucose responses, and insulin sensitivity index values at baseline, 24 hours post-intervention and 72 hours post-intervention are shown in Fig. 1, with fasting insulin and glucose concentrations and areas under the glucose and insulin curves (AUCs) shown in Table 1. Fasting insulin (by 24.6%, $P = .047$) and insulin AUC were significantly lower (by 15.0%, $P = .042$) and insulin sensitivity index was significantly higher (by 23.3%, $P = .027$) at 24 hours post-intervention compared with baseline. However, these values did not differ significantly from baseline at the 72-hour post-intervention measurement. No significant differences were observed between trials for fasting glucose or glucose AUC or for fasting triglycerides, NEFA, total cholesterol, or HDL cholesterol (Table 1).

3.4. Resting energy expenditure and substrate utilization

There was no significant difference in resting metabolic rate between measurements at baseline, 24 hours post-intervention, and 72 hours post-intervention. However, respiratory exchange ratio (RER) and resting carbohydrate

Table 1
Anthropometric, fitness, and metabolic measurements at baseline and at 24 and 72 hours post-intervention

Variable	Baseline	24 h post-intervention	72 h post-intervention	Baseline vs 24 h *P* value	Baseline vs 72 h *P* value
$\dot{V}O_{2max}$ (L min^{-1})	2.98 ± 0.15	–	3.23 ± 0.14	–	.013
$\dot{V}O_{2max}$ (mL kg^{-1} min^{-1})	32.8 ± 1.4	–	35.9 ± 1.6	–	.015
Peak power (W)	981 ± 62	–	1027 ± 69	–	.195
Mean power (W)	579 ± 24	–	600 ± 19	–	.040
Body mass (kg)	93.9 ± 4.0	92.9 ± 4.2	–	.055	–
Waist circumference (cm)	101.3 ± 2.7	98.9 ± 3.1	–	.004	–
Hip circumference (cm)	110.9 ± 2.2	109.8 ± 2.2	–	.017	–
Fasting glucose (mmol L^{-1})	5.51 ± 0.05	5.35 ± 0.11	5.23 ± 0.21	.685	.212
Fasting insulin (mU L^{-1})	10.42 ± 1.91	7.86 ± 1.38	10.24 ± 1.49	.047	.996
Fasting triglycerides (mmol L^{-1})	1.28 ± 0.17	1.13 ± 0.15	1.17 ± 0.09	.481	.935
Fasting NEFA (mmol L^{-1})	0.63 ± 0.11	0.67 ± 0.09	0.54 ± 0.11	.654	.778
Fasting HDL cholesterol (mmol L^{-1})	1.07 ± 0.08	1.02 ± 0.07	1.04 ± 0.07	.474	.423
Fasting total cholesterol (mmol L^{-1})	4.89 ± 0.29	4.66 ± 0.31	4.53 ± 0.30	.106	.147
Glucose AUC (mmol L^{-1} h)	15.6 ± 0.9	15.3 ± 0.9	14.9 ± 0.9	.773	.315
Insulin AUC (mU L^{-1} h)	144 ± 26	122 ± 21	134 ± 25	.042	.781
RER	0.78 ± 0.01	0.73 ± 0.01	0.75 ± 0.02	.013	.094
Resting metabolic rate (kJ min^{-1})	5.3 ± 0.2	5.5 ± 0.2	5.5 ± 0.2	.404	.223
Carbohydrate oxidation (g min^{-1})	0.08 ± 0.02	0.03 ± 0.01	0.05 ± 0.02	.037	.179
Fat oxidation (g min^{-1})	0.11 ± 0.01	0.13 ± 0.01	0.12 ± 0.01	.019	.113
Systolic blood pressure (mm Hg)	127 ± 3	121 ± 3	125 ± 3	.020	.197
Diastolic blood pressure (mm Hg)	80 ± 3	71 ± 2	73 ± 3	.066	.062
PWV (m s^{-1})	7.79 ± 0.48	7.64 ± 0.47	7.64 ± 0.50	.976	.976

Data are presented as mean ± SEM.

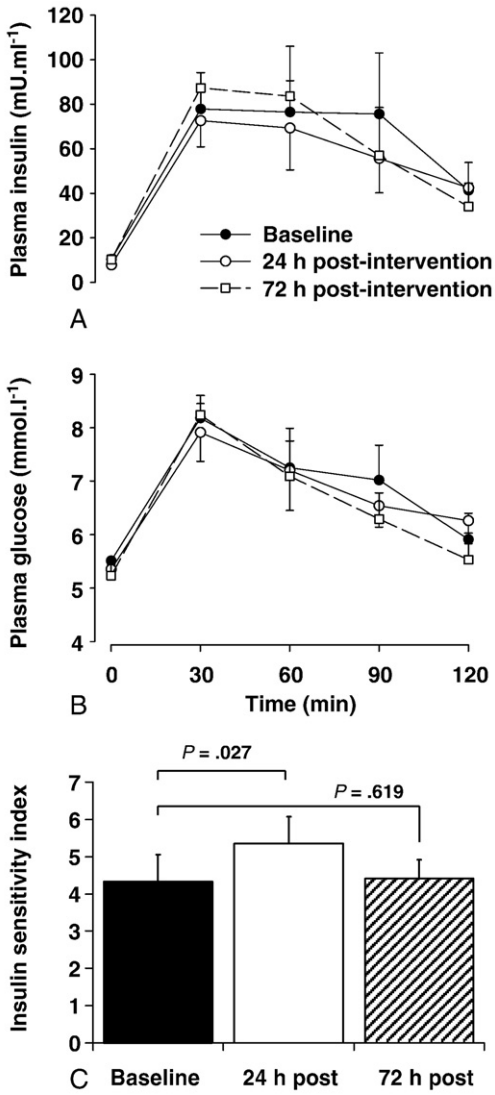

Fig. 1. Plasma glucose (A) and insulin (B) concentrations in response to a 75-g oral glucose load, and insulin sensitivity index (C) at baseline and then 24 and 72 hours post-intervention. The AUC values for plasma glucose and insulin during the OGTT are shown in Table 1.

oxidation were significantly lower (P = .013 and P = .037 respectively) and resting fat oxidation was higher (by 18.2%, P = .019) at the 24-hour post-intervention measurement compared with baseline. No differences were observed in resting metabolic rate or substrate utilization between baseline and 72-hour post-intervention measurements (Table 1).

3.5. Blood pressure and PWV

Systolic pressure was 4.7% lower at the 24-hour post-intervention assessment (P = .020) compared with baseline, but this effect was lost by 72 hours (P = .197). Diastolic blood pressure tended to be lower than baseline at 24-hour (P = .066) and 72-hour (P = .062) assessments (Table 1). However, PWV did not differ significantly from baseline at either 24- or 72-hour post time points (P = .976 and P = .976, respectively).

4. Discussion

The main finding of this study was that 6 sessions of SIT undertaken over 2 weeks increased $\dot{V}O_{2max}$ and mean power output during a Wingate test, improved insulin sensitivity, increased resting fat oxidation, and reduced systolic blood pressure in a group of overweight/obese men. This extends the findings of earlier studies which have demonstrated that exercise training of this nature can improve indices of cardiorespiratory fitness [15,19] and insulin sensitivity [22] in young, fit, normal-weight adults, as well as demonstrating for the first time that such a protocol markedly influences resting substrate utilization. The magnitude of the changes seen were comparable to those observed after 6 to 8 weeks of conventional endurance-type exercise training in untrained or moderately trained adults [21,31,32], and the training sessions were generally well tolerated by the participants. Thus, the findings of the present study provide preliminary evidence to suggest that SIT may provide an alternative exercise model for the improvement of vascular and metabolic health in sedentary overweight and obese men.

Maximal oxygen uptake was increased by 8.4% after 2 weeks of the SIT intervention. The magnitude of this improvement is similar to that reported by MacDougall et al [19] after 7 weeks of a similar SIT intervention and is greater than that found after 3 weeks of SIT in recreationally active individuals [15]. The rapid improvement in $\dot{V}O_{2max}$ found in our cohort of subjects in comparison with other SIT interventions is most likely due to their relatively low baseline level of fitness, as rapid increases in $\dot{V}O_{2max}$ are often seen when unfit, sedentary individuals initially start an exercise program [33]. In addition, consistent with our findings, it has been shown that as little as 2 weeks of SIT increases mitochondrial enzyme activities [16,18], with increases in mitochondrial enzyme levels [33] playing a small role in increasing $\dot{V}O_{2max}$ [33]. One limitation of the present study is the lack of a control group; and thus, it is conceivable that the observed change in $\dot{V}O_{2max}$ might be confounded by a "learning effect" or subjects not providing a true maximal effort during the baseline test. However, 2 pieces of evidence indicate that this is unlikely to have had a major effect on the findings. Firstly, all subjects completed a familiarization session before baseline testing; and no difference was observed between familiarization and baseline measurements of $\dot{V}O_{2max}$ (2.95 ± 0.17 vs 2.98 ± 0.21 L min^{-1}, respectively; P = .777). Secondly, the maximal heart rate achieved during incremental tests did not differ between the baseline and post-intervention measurement (183 ± 4 vs 184 ± 5 beats per minute, P = .866).

The change to insulin sensitivity observed in the present study was lost at the 72-hour post-intervention assessment. This is in common with many exercise interventions that have reported relatively transient improvements in insulin sensitivity [34,35]. From the available data, it is not possible to determine whether the changes observed in the present study were due to an acute effect of recent exercise or a short-lived

training adaptation. However, it seems likely that the "last bout" effect played an important role. It has recently been shown that activation of adenosine monophosphate–activated kinase (AMPK) is increased after a single session of four 30-second cycle ergometer sprints [36]; and activation of AMPK has been shown to increase glucose uptake into skeletal muscle, via increased translocation of GLUT4, and to affect insulin signaling directly via phosphatidylinositol 3-kinase [37]. Furthermore, it has been demonstrated that two to four cycle ergometer 30-second sprints reduce muscle glycogen concentrations by approximately 30% to 45% [36,38]. This reduction in glycogen concentration causes activation of glycogen synthase [39,40], possibly via AMPK [40], and plays a key role in mediating translocation of GLUT4 and glucose uptake into muscle [39,41]. Interestingly, the degree of glycogen depletion elicited by two to four 30-second sprints (expending ~ 40-80 kcal energy) is equivalent to that elicited by approximately 45 to 90 minutes of moderate-intensity endurance-type exercise [42]. This rapid glycogen-depleting effect may explain, at least in part, why SIT induces the observed metabolic changes with such low levels of total energy expenditure.

Systolic blood pressure was significantly reduced 24 hours, but not 72 hours, after the SIT intervention. It has been known for some time that a single bout of exercise can transiently lower blood pressure [43], with this effect persisting for up to about 24 hours post-exercise [44,45]. The exact mechanisms responsible have not been fully elucidated, but it is likely to be a combination of reduced sympathetic nervous activity [46] and increased nitric oxide–mediated vasodilatation [47]. In contrast, PWV—a noninvasive method of determining arterial distensibility, with a higher velocity indicating greater stiffness and thus a higher risk of cardiovascular disease [48,49]—did not change in response to the 2-week intervention in this study. However, Rakobowchuk et al [21] reported an increase in popliteal artery distensibility after a 6-week SIT program in healthy individuals. This difference may be a consequence of the greater duration of their intervention (2 vs 6 weeks): it is possible that 2 weeks of SIT is too short an intervention to develop these vascular changes. It is also possible that because our subjects had relatively low baseline PWV values (ie, indicating they had high arterial compliance), which were within the range found in a healthy population [50], the scope for exercise to improve this already high compliance was limited.

One intriguing finding from the present study was that the SIT intervention increased resting fat oxidation and reduced resting RER. This is likely to be a consequence of the effects of the exercise sessions on muscle glycogen concentrations [51] and could conceivably have implications for the long-term maintenance of body weight. The increase in fat oxidation observed is of a similar magnitude to that seen on the day after 90 minutes of moderate-intensity exercise in the absence of an energy deficit [52]. In addition, we have recently reported that the magnitude of increase in resting fat oxidation in response to an exercise training intervention is a significant predictor of the extent of exercise-induced fat loss, independent of exercise energy expenditure and change in resting metabolic rate [53]. However, the short-term nature of the present intervention precluded any obvious effects on body weight becoming apparent, although significant reductions in waist and hip circumferences were observed. A longer-term intervention is needed to ascertain whether an intervention of this nature can play a role in body weight management.

Although this study has demonstrated, in principle, that SIT can elicit a number of health-related benefits in overweight/obese men, it is premature to recommend SIT in the form used here as a physical activity strategy to the general population. The risk of an acute cardiovascular event during exercise increases with increasing exercise intensity, particularly in those who are older, are unaccustomed to exercise, or have existing cardiovascular disease [54], although high-intensity interval-type exercise programs have recently been successfully implemented, without incident, in patients with the metabolic syndrome [55] and with coronary artery disease [56]. However, SIT might be an appropriate physical activity option for younger individuals without pre-existing cardiovascular disease, particularly if they progress to this after an initial period of moderate-intensity activity. In addition, to complete the SIT program in its present form, high levels of motivation are essential; thus, it is not known whether individuals can attain the same benefits achieved during this study when unsupervised.

Furthermore, recent emphasis has been placed on finding modes of exercise training that provide a time-efficient strategy to improve health, as lack of time is an often-reported barrier to exercise (eg, Reichart et al [4], Trost et al [5], and Brownson et al [6]). Although several previous reports suggest that SIT requires a total of only 3 minutes of exercise, the training sessions themselves entail 24 to 34 minutes of exercise (4-minute warm-up, plus four to six 30-second Wingate with 4.5-minute recovery between each sprint). Therefore, using SIT in its present form necessitates a duration that is no different to the current guidelines for promoting health [3] and is similar to other studies that have also found health benefits while using much lower intensities of exercise to which participants may have greater tolerance [8]. However, despite the total duration of SIT sessions being relatively long, because the duration of high-intensity exercise phases are very short, it is conceivable that this may help to increase adherence to exercise. Further research in a randomized controlled trial is warranted to determine whether adherence to SIT would be greater than a traditional moderate-intensity exercise program in a "real-world" setting.

Further research should consider different approaches to reduce the total duration of exercise. For example, studies could use shorter-duration sprints to establish whether there is a "threshold" sprint duration whereby health benefits occur

or whether fewer repetitions of longer-duration sprints (eg, 1-minute sprints) could be used. In addition, work is needed to ascertain whether the population studied could endure a reduction in recovery time between sprints. Therefore, the findings of this study provide an important first step towards an evidence base for the utilization of SIT as an exercise strategy for the overweight/obese sedentary population. However, much further study and refinement of the exercise protocol are required before this form of exercise is ready for widespread recommendation to the population at large.

Acknowledgment

We would like to dedicate this paper to the memory of Dr Andy Cathcart, who was tragically killed in a cycling accident shortly after completion of this study. He will be sorely missed by his colleagues, the students he taught, and his family and friends.

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