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Mechanical efficiency improvement in relation to metabolic changes in sedentary obese adults

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ABSTRACT

Purpose: Mechanical efficiency (ME) refers to the ability of an individual to transfer energy consumed by external work. This performance indicator is impaired by obesity and is associated with decreased high-intensity exercise performance. However, it is unclear if ME may be improved in response to high intensity training (HIT). This study aimed to determine if ME increases in response to HIT in obese adults and to identify the factors associated with these changes.

Methods: 24 obese adults (body mass index=~33 kg/m^2) were randomised into control (n=12) and trained (n=12) groups. Following baseline metabolic, anthropometric, fitness and ME measurements, the participants completed a 6-week exercise intervention that included 18 sessions of six repeats of 6 s supramaximal sprints on an electromagnetically braked cycle ergometer. The metabolic, anthropometric and fitness assessments were repeated postintervention. ME (expressed as a %) was calculated during an incremental maximal cycling test at stages of 25, 50, 75, 100 and 125 W.

Results: ME did not differ across the groups at 25 and 50 W. Following HIT, ME increased significantly at 75, 100 and 125 W ($p<0.01$, respectively) compared with the control group ($p<0.01$, respectively). Although no changes in fat-free mass were observed following HIT, the increases in ME at 75, 100 and 125 W correlated positively with both homeostasis model assessment-estimated insulin resistance index decreases (r=0.9; r=0.89 and r=0.88, $p<0.01$, respectively) and peak power increases (r=0.87, r=0.88 and r=0.9, $p<0.01$, respectively).

Conclusions: Although there were no changes in the participants' anthropometric variables, HIT improved ME in obese adults, an enhancement that appears to be related to increases in muscle strength and metabolic adaptations.

What are the new findings?

- High intensity training increased mechanical efficiency levels during incremental exercise in obese adults although no changes in fat-free mass were observed.
- Following high intensity training the amelioration of mechanical efficiency was associated with improved homeostasis model assessment-estimated insulin resistance and concomitant increases in power output.

How might it impact on clinical practice in the near future?

- The utilisation of high intensity training may be considered as an exercise strategy for the obese sedentary population.
- The mechanical efficiency constitutes an important parameter to be evaluated among obese individuals with respect to the detection of muscle dysfunction and any subsequent adaptations in response to training.

INTRODUCTION

Obese adults may exhibit considerable functional limitations with respect to motor activity due to reductions in several fitness indicators, including aerobic[1] and anaerobic.[2] Excessive fat mass reduces oxygen uptake by working muscles, motor unit activation and muscle strength.[3 4] Additionally, obesity affects mechanical efficiency (ME), or the ability of an individual to transfer energy consumed during external work.[5] Studying the efficacy of an intervention programme among obese individuals often entails the evaluation of aerobic and anaerobic modifications in response to exercise training. However, ME may also be an important predictor of said efficacy and may provide relevant data regarding performance and energy use adaptations in response to training.

In the setting of obesity, studies examining ME in adult populations have reported lower ME levels compared with non-obese individuals.[6] Lower ME indicates that more energy is consumed at a given work output. Therefore, individuals with lower ME values should be less efficient with respect to performance and may therefore be limited in terms of physical activity. In spite of the fact

that the numbers of interventional studies evaluating the effects of exercise training on ME values are scarce, the available observational data obtained from obese children and adults are indicative of a strong correlation between physical activity levels and ME values.[7] More specifically, it appears that moderate-to-vigorous physical activity levels prevent ME alterations, as higher intensity training is associated with higher muscle performance.[8]

Regarding training efficacy, several studies have demonstrated that high intensity training (HIT) (lasting between 2 and 15 weeks) results in significant increases in muscle performance in untrained males, even over brief durations,[9] as well as in overweight and obese men.[10] These adaptations are likely the result of skeletal muscle adaptations related to metabolic improvement associated with increased insulin sensitivity[11] and ameliorations in muscle strength.[12] Given that metabolic milieu and muscle function may condition both muscle performance and an individual's muscle energy profile, it is possible that any ameliorations in these parameters may be predictive of a subsequent amelioration in ME values among obese individuals.

Given that at higher intensity levels, the ability of a muscle to produce mechanical work requires greater energy producing and optimal motor unit involvement, we hypothesised that this form of exercise may promote ME. This study aimed to determine the effect of HIT on ME values among sedentary obese adults, as well as the factors (eg, metabolic and power) associated with ME changes among trained individuals.

METHODS

Experimental participants

Twenty four young adults (12 women and 12 men) were recruited from the Moncton campus of the University of Moncton. This study was approved by the University's Human Research Ethics Committee (UHRC) of the University of Moncton, and the participants were asked to sign a full consent form prior to beginning the study. In addition to being obese, the inclusion criteria for participation were as follows: a sedentary lifestyle (participating in <1 h/week of structured exercise as assessed via the International Physical Activity Questionnaire,[13] no history of either cardiovascular disease or chronic health problems, no history of drug use before the study and no history of smoking. Before entering our protocol, each of the participants was thoroughly familiarised with all of the testing equipment and procedures. Each participant cycled for an extended period of time on the same cycle ergometer used during the study. Additionally, each participant was asked to determine the height of seat at which they are able to pedal comfortably. Unfortunately, we do not have any objective positioning (knee angle, hip angle, etc) and this may be considered as limitation of the study. However, it is important to note that the position of each participant, for example, the seat height, was the same through the study.

The protocol began with three sessions of preliminary testing to determine certain key variables. The testing was conducted on two different days (D1 and D2). Each day was separated by a minimum of 48 h, and all participants were asked to avoid physical activity for 48 h prior to each session.

Anthropometric measurements

Body mass was measured to the nearest 0.1 kg, with the participant in light clothing, without shoes, using an electronic scale (Kern, MFB 150K100). Height was determined to the nearest 0.5 cm using a measuring tape fixed to a wall. Waist and hip circumferences were each measured to the nearest 0.1 cm. Body mass index (BMI) was calculated as the ratio of mass (kg) to height^2 (m^2). Body fat percentage was estimated using a bioimpedance machine (Vacumed, Bodystat 1500). Following the determination of body composition, the obese participants (BMI >30 kg/m^2) were selected based on the Canadian guidelines for body weight classification in adults[14] and separated in the following two groups: a control group (without any intervention; n=12) and a training group (n=12). Fat-free mass was calculated by subtracting fat mass from body mass.

Physiology and metabolic testing

On day 1 (D1), the participants arrived at the laboratory following a 12 h overnight fast. Following 5 min of rest, venous blood samples were drawn from an antecubital vein. On extraction, the blood was collected in a vacutainer tube containing *EDTA*. Haematocrit was measured three times for each blood sample via microcentrifugation *(JOUAN-HEMAC)*. Plasma from the venous blood samples was separated via centrifugation at 3000 g for 20 min (4°C) (*ORTO ALRESA mod. Digicen.R, Spain*). The aliquots were immediately frozen and stored at −80°C for use in subsequent chemical analyses. Commercially available kits were used to determine blood glucose levels (ABX Pentra, Montpellier, France). A single analyser was used for each participant, and each sample was analysed in duplicate. Plasma insulin concentrations were measured in the centralised laboratory via a radioimmunoassay procedure (Phaadebas Insulin Kit; Pharmacia Diag-nostics AB, Piscataway, New Jersey, USA). An estimate of insulin resistance was calculated via the homeostasis model assessment-estimated insulin resistance (HOMA-IR) index, as follows: (fasting insulin (μU/mL)×fasting glucose (mmol/L)/22.5.

The patients were asked to remain in a supine position for 5 min before the continuous measurement of pulmonary gas exchange using a breath-by-breath automated metabolic system (CPX, Medical Graphics, St. Paul, Minnesota, USA) to allow for an assessment of resting oxygen consumption based on the mean oxygen consumption of the last 30 s of minutes 2, 3, 4 and 5.

The participants then performed a maximal test using an upright cycle ergometer (Monark ergomedic 839E electronic test cycle, USA) to determine their maximal

oxygen consumption (VO_{2max}). Before beginning the test, adults remained seated for 5 min on the bicycle ergometer in the same position used in subsequent exercise. Resting oxygen consumption was measured based on the mean oxygen consumption of the last 30 s of minutes 3, 4, and 5. No proper warm-up was performed. The initial power was set at 25 W and was progressively increased by 25 W every 2 min until exhaustion to determine the VO_{2max} of each participant. During the test, the participants were instructed to pedal at a rate of 50–70 revolutions per minute. Maximal oxygen consumption was achieved when the participant fulfilled at least three of the following criteria: a plateau in VO_2 in spite of an increase in exercise intensity, a respiratory exchange ratio greater than 1.1, a maximal HR above 90% of the predicted maximal theoretical HR (220—age in years) or apparent exhaustion.[15] In this study, all experimental participants achieved their maximal test at 125 W during their preintervention and postintervention visits.

On day 2 (D2), following 10 min of warm-up, the participants performed a force-velocity test using a cycle ergometer, using a technique adapted from the study performed by Vandewalle *et al*.[16] This test consists of a succession of supramaximal bouts of approximately 6 s, with exercise loads increasing by 1 kg following each bout until the participant is unable to perform the test. A period of passive recovery (5 min) was allowed between successive bouts. The peak velocity for each bout was recorded, and the power output was calculated by multiplying the load and speed. The optimal load corresponded to the load at which maximal power (PO_{max}) was achieved. As previously developed by our laboratory[17] this load was then used for the training protocol that followed. The force-velocity test was also performed every 2 weeks to adjust the individual power level of the HIT.

TRAINING SESSION

Once the participants completed the preliminary testing, they were instructed to complete a total of 18 training sessions (three sessions per week for 6 weeks). Each of the prescribed sessions began with a 5 min warm-up of continuous cycling at moderate intensity (40% of their individual VO_{2max} power), followed by six repetitions of supramaximal sprint intervals with 2 min of passive recovery between each repetition. Each supramaximal repetition lasted 6 s, and the participants were asked to pedal at maximal velocity against the resistance determined during D2. This form of exercise has been previously developed by Jabbour *et al*.[17] The repeat sprint cycling test was conducted under the supervision of a member of the research team, and velocities (in RPM) were recorded for each second of the bout in order to ensure that said velocities were constant. Based on the linear regression and the individual VO_{2max}, the workload approximately corresponding to (~350% of VO_{2max}).[19] The total duration of each session was approximately 15 min.

Training specification: Regarding high-intensity exercise training, the most commonly utilised protocol is the Wingate test (30 s of all-out sprinting). Although most researchers agree that this intervention is extremely beneficial in individuals with excess body weight,[10] this protocol is extremely difficult, as participants must tolerate considerable discomfort. Given that very brief high-intensity exercise in the form of sprint, which typically lasts 6–10 s, induces substantial improvements in performance and health-related outcomes,[9] the present training model was remarkably short in duration and was tolerated extremely well by our experimental participants as previously shown by Jabbour *et al*.[17]

The training sessions were conducted under the supervision of a member of the research team, and velocities (in RPM) were recorded for each second of the bout to ensure that said velocities were constant. Postintervention anthropometric, metabolic, aerobic, anaerobic and ME measurements were conducted approximately 72 h following the final training session. Throughout the intervention, the participants were asked to refrain from consuming alcohol and encouraged to continue their normal diet and maintain their typically sedentary behaviour. The participants recorded a 48 h food diary before baseline testing and repeated this before any subsequent tests. There were no significant differences in energy, carbohydrate, protein or fat intake between the tests.

ENERGY CONSUMPTION AND ME CALCULATIONS

ME was calculated for all workloads (25, 50, 75, 100 and 125 W) of the maximal incremental test using the formula developed by Lafortuna *et al*[18] as follows: work produced, in Watts/(total energy consumption, in Watts) 100. In the present study, resting energy consumption (E_{rest}) was subtracted from total energy consumption at each exercise stage[5] allowing us to calculate a net ME. The energy consumption, (E) in Watts, was calculated as follows: (4.94 respiratory exchange ratio+16.04) (VO_2, in mL/min)/60.[20] For E, the resting oxygen consumption (VO_{2rest}) was subtracted from the total oxygen consumption for each exercise stage.

STATISTICAL ANALYSIS

After testing for normality (Kolmogorov–Smirnov test), statistical comparisons were made between the control group and the training group on two separate occasions (before and after training). Two way repeated measures analysis of variance were used to determine whether significant changes in ME emerged between the two groups, and if ME differed between the two groups. Furthermore, Bonferroni's post hoc test was performed. Pearson correlations were used to assess the relationship among changes in muscle strength and metabolic adaptations and ME modification. A value of $p<0.05$ was

statistically significant. Analyses were performed using IBM SPSS Statistics V. 19 software.

RESULTS

Anthropometric and fitness parameters.

Height, body mass, BMI, fat mass and fat-free mass were similar across the two groups (table 1). The VO_{2max} values assessed following 6 weeks of HIT training did not differ from the baseline values (table 1). Peak power output obtained during the charge-velocity test increased significantly in the training group compared with the baseline values ($p<0.01$; table 1) and was significantly higher than the corresponding values of the control group.

Blood variables

The insulin, glucose and HOMA-IR (insulin resistance index) values at baseline and following the intervention are included in table 1. Fasting glucose and insulin were significantly lower (by: ~7% and ~41%; $p<0.01$, respectively), and HOMA-IR was significantly lower (by ~44%, $p<0.01$) following the intervention compared with baseline in the trained group. Additionally, these values were significantly lower than the corresponding values of the control group following the intervention (table 1).

Oxygen and energy consumption and ME values

At rest, oxygen consumption and energy consumption (E) were significantly lower in the trained group compared with baseline and with the control group (table 2). Following HIT, the oxygen consumption values were similar among the groups for all stages. Additionally, E did not differ between the groups at 25 and 50 W. By contrast, E was significantly lower in the trained group compared with the control group at 75, 100 and 125 W (table 2). ME, as measured at submaximal and peak effort, is included in table 2. ME increased with increasing ergometric workload in the trained group following the intervention. For the control group and the trained group before the intervention, ME deceased significantly at 75, 100 and 125 W compared with the value obtained at 50 W (table 2). Moreover, ME did not differ significantly between the groups at 25 and 50 W. However, following HIT, ME increased significantly in the trained group at 75, 100 and 125 W compared with baseline ($p<0.01$, respectively) and with the control group ($p<0.01$, respectively).

In this study, the increased ME levels observed at 75, 100 and 125 W in the trained group following HIT correlated positively with both HOMA-IR index decreases ($r=0.9$; $r=0.89$ and $r=0.88$, $p<0.01$, respectively) and peak power increases ($r=0.87$, $r=0.88$ and $r=0.9$, $p<0.01$).

DISCUSSION

This interventional study was the first to examine the effects of HIT on ME levels in obese adults. Our analysis revealed that for each stage, all participants cycled at the same relative intensity of peak power output, as follows: stages 25, 50, 75, 100 and 125 W corresponded to 20%, 40%, 60%, 80% and 100% of peak power, respectively. The primary finding of this study was that our training model increased ME levels during incremental exercise, changes associated with improved HOMA-IR and concomitant increases in power output. Additionally, the ME levels obtained for the control group and the trained group before HIT increased significantly at 50 W compared with the values obtained at 25 W. At 75, 100 and 125 W, ME values decreased significantly, reaching as low as ~−2.5% at 125 W. This result highlights the fact that at maximal intensity, the body's energy demands are met via anaerobic processes; the ability of muscle to produce work may be reduced due to impairments in muscle strength and metabolic milieu.

Following 6 weeks of HIT, maximal oxygen uptake did not differ significantly compared with values obtained before the intervention in both groups. These results contradict data obtained in response to high intensity interval training (eg, the Wingate test protocol)[10] and continuous exercise training.[21] The brief supramaximal exercise training used in the present study does not provide an adequate stimulus with which to improve the indices of aerobic fitness. However, the peak power output in response to HIT improved significantly among the obese adults (+105 W) in spite of any concomitant improvements in anthropometric variables. Several studies indicate that obese individuals frequently suffer from an increased prevalence of motor limitations,[6] which contribute to general sense of fatigue;[22] any increases in free-fat mass have been linked to muscle performance improvements.[23] Interestingly, the present study demonstrated that metabolic improvement may also be associated with muscle performance increases among obese individuals. Indeed, in the training group, the peak power increases correlated significantly with improvements in HOMA-IR values. Data obtained in a cross-sectional study indicate that metabolic abnormalities are independently associated with low muscle power among obese individuals.[24] These metabolic abnormalities are related to hyperinsulinaemia-induced alterations in glucose metabolism and reduce glucose uptake into the muscle,[25] a major factor limiting anaerobic performance. HIT may have the potential to increase muscle power by improving metabolic disorders, as demonstrated in this study.

ME increases were observed only at 25 and 50 W in response to incremental exercise in the control and the trained group before the intervention. Up to 50 W, the ME levels were significantly lower compared with those obtained at 50 W. As mentioned previously, an increase in ME with workload may be explained by the constant amount of energy required to move the pedals, regardless of the ergometric load, to maintain cycling posture and overcome the internal ergometric friction.[5] As workload increases, the proportion of this energy decreases compared with the total energy requirement, resulting

Table 1 Age, anthropometric, aerobic fitness parameters and metabolic profiles of obese adults

	Control		Trained		Δ Postintervention versus baseline for control group	Δ Postintervention versus baseline for trained group
	Baseline	Postintervention	Baseline	Postintervention		
Age and anthropometrics						
Age (year)	23.1 (3.3)	–	23.3 (2.3)	–	–	–
Height (cm)	1.71 (0.11)	–	1.71 (0.11)	–	–	–
Body mass (kg)	99.5 (24.1)	100.5 (21.1)	101.1 (21.1)	99.9 (9.1)	+1	−1.2
BMI (kg/m^2)	33.3 (4.8)	33.2 (2.8)	33.7 (3.8)	33.1 (3.7)	−0.1	−0.6
FM (%)	42.3 (9.4)	42.8 (7.4)	44.3 (9.4)	42.1 (7.1)	+0.5	−2.2
FFM (kg)	51.2 (10.1)	50.2 (9.1)	50.2 (9.1)	50.8 (5.7)	−1	−0.6
Fitness indicators						
HR rest (bpm)	96 (13)	97 (14)	94 (10)	96 (13)	+1	+2
HR peak (bpm)	196 (12)	197 (06)	196 (09)	198 (12)	+1	+2
RER peak	1.1 (0.1)	1.1 (0.3)	1.1 (0.3)	1.1 (0.2)	–	–
VO_{2max} (mL/min/kg)	23.4 (8.4)	23.6 (4.4)	22.2 (7.4)	22.8 (3.4)	+0.2	+0.6
Peak power (W)	470 (30)	465 (25)	465 (35)	570 (40)[a b]	−5	+105
Metabolic profiles						
Fasting glucose (mmol/L)	4.61 (0.11)	4.61 (0.14)	4.64 (0.15)	4.31 (0.24)[a b]	–	−0.33
Fasting insulin (µmol/mL)	22.6 (4.6)	21.9 (4.6)	23.2 (4.6)	13.6 (3.8)[a b]	−0.7	−9.6
HOMA-IR	4.63 (1.7)	4.61 (1.7)	4.78 (1.2)	2.63 (1.7)[a b]	−0.2	−2.15

Data are presented as the mean (SD).
Significant difference between groups ([a]p<0.01), significant difference from baseline values ([b]p<0.01).
BMI, body mass index; FM, fat mass; FFM, fat free mass; HOMA-IR, homeostasis model assessment-estimated insulin resistance; RER, respiratory exchange ratio, VO_{2max}, maximal oxygen consumption, peak power: maximal power output developed during the charge-velocity test.

Table 2 Mean values of oxygen and energy consumption and ME

	Control		Trained		Δ Postintervention versus baseline for control group	Δ Postintervention versus baseline for trained group
	Baseline	Postintervention	Baseline	Postintervention		
$VO_{2\ rest}$ (mL/min)	280 (55)	284 (83)	286 (67)	232 (50)[a b]	+4	−54
E_{rest} (W)	92 (22)	92 (22)	96 (26)	79 (30)[a b]	–	−17
25 W						
VO_2 (mL/min)	479 (40)[c]	470 (119)[c]	471 (79)[c]	466 (77)[c]	−9	−4
E (W)	164 (41)[c]	168 (51)[c]	168 (26)[c]	169 (26)[c]	+4	+1
ME (%)	15.1 (4.1)	14.8 (8.1)	14.8 (3.1)	14.7 (4.1)	−0.3	−0.1
ΔME (%)	–	–	–	–	–	–
50 W						
VO_2 (mL/min)	781 (86)[c]	786 (97)[c]	792 (59)[c]	781 (61)[c]	+5	−5
E (W)	267 (29)[c]	263 (32)[c]	260 (20)[c]	260 (14)[c]	−3	−3
ME (%)	18.7 (2.1)[c]	19.1 (3.1)[c]	19.2 (1.5)[c]	19.2 (3.4)[c]	+0.4	+0.1
ΔME (%)	+3.6	+4.3	+4.4	+4.5	–	–
75 W						
VO_2 (mL/min)	1301 (80)[c]	1298 (102)[c]	1311 (66)[c]	1300 (102)[c]	−3	+2
E (W)	422 (27)[c]	432 (38)[c]	431 (24)[c]	380 (35)[a b c]	+10	−51
ME (%)	17.7 (2.6)[c]	17.3(2.2)[c]	17.4 (2.4)[c]	19.8 (2.2)[a b c]	−0.3	+2.5
ΔME (%)	−1	−1.8	−1.8	+0.6	–	–
100 W						
VO_2 (mL/min)	1509 (95)[c]	1499 (139)[c]	1460 (83)[c]	1480 (188)[c]	−10	−19
E (W)	548 (32)[c]	551 (47)[c]	555 (31)[c]	487 (65)[a b c]	+3	−64
ME (%)	18.2 (1.9)	18.1 (1.2)	18.1 (1.2)	20.5 (2.8)[a b c]	−0.1	+2.4
ΔME (%)	+0.5	+0.8	+0.7	+0.7	–	–
125 W						
VO_2 (mL/min)	1610 (190)[c]	1598 (212)[c]	1595 (200)[c]	1596 (499)[c]	−12	−2
E (W)	746 (62)[c]	750 (73)[c]	753 (19)[c]	571 (34)[a b c]	+4	−179
ME (%)	16.7 (4.1)[c]	16.6 (4.3)[c]	16.6 (2.8)[c]	21.9 (1.3)[a b c]	−0.1	+5.3
ΔME (%)	−1.5	−1.5	−1.5	+1.4	–	–

Data are presented as the mean (SD).
Significant difference between groups ([a]p<0.01), significant difference from baseline values ([b]p<0.01), significant difference from the values obtained above ([c]p<0.01).
E, energy consumption; ME, mechanical efficiency; ΔME, changes in mechanical efficiency; VO_2, oxygen consumption.

in higher ME values.[26] The lower ME observed in the present study at 75, 100 and 125 W may be a consequence of lower muscle performance. As previously reported, obesity in adults is associated with an increased proportion of glycolytic muscle fibres,[27] which are substantially less efficient than type I fibres, during cycling.[25] This profile may contribute to decreased muscle efficacy among obese adults.[19]

However, following the intervention, the ME values increased significantly in the obese adults compared with the values obtained before HIT. These increases ranged from +2.5% at 75 W to +5.3% at 125 W compared with the participants' baseline values and were significantly higher compared with the corresponding values obtained in the control group. The improvements in ME observed in the present study reflect the amelioration of the transfer of internal energy consumption to external work output. These data support the fact that HIT enhances muscle efficacy to perform work, particularly at higher intensity levels, and highlight the importance of high exercise intensity in preserving ME in obese adolescents.[7] Additionally, the improvements in the ME levels observed in the trained group following HIT correlated positively with both HOMA-IR index decreases and peak power increases. As mentioned above, alterations in metabolic milieu may impair substrate use (eg, carbohydrate) and muscle performance; therefore, HIT may improve these parameters.[28 29]

In conclusion, the present study demonstrated that HIT may improve peak power output and ME levels in obese adults and that these ameliorations may be attributed to improvements in metabolic milieu, without excluding increases in muscle motor function. The findings of the present study represent an important first step in an evidence based approach regarding the utilisation of HIT as an exercise strategy for the obese sedentary population. However, ME may be considered an important parameter to be evaluated among obese individuals, in addition to other classical parameters (oxygen consumption and peak power output). Additionally, given that obese people experience considerable functional limitations and suffer from an increased prevalence of health problems secondary to insufficient levels of skeletal muscle power relative to their body mass, the use of ME may be valuable with respect to the detection of muscle dysfunction and any subsequent adaptations in response to training.

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