

# Mastergradsoppgave

**Effects of high and moderate intensity aerobic exercise on excess post-exercise oxygen consumption in men with metabolic syndrome**

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## **SAMTYKKE TIL HØGSKOLENS BRUK AV MASTEROPPGAVE I KROPPSØVING**

**Forfatter:** Ida Larsen

**Norsk tittel:** Effekten av høy og moderat intensitets utholdenhetstrening på det overskytende oksygenforbruket hos menn med metabolsk syndrom

**Engelsk tittel:** Effects of high and moderate intensity aerobic exercise on excess post-exercise oxygen consumption in men with metabolic syndrome

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## Norsk sammendrag

**Formål:** Fysisk aktivitet er en sentral faktor i forebygging og behandling av metabolsk syndrom, hvor effektiv vektreduksjon og økt energiforbruk er med på å redusere alle risikofaktorer knyttet til metabolsk syndrom. Fysisk aktivitet med høy intensitet krever mer energi under selve aktiviteten. Dette kan videre bidra til økt energiforbruk i restitusjonsfasen etter trening. Målet med denne studien var å undersøke de akutte effektene etter en treningsøkt på det overskytende oksygenforbruket (EPOC) mellom tre typer utholdenhetstrening hos menn med metabolsk syndrom. **Metode:** Syv menn fra 39 til 70 år med metabolsk syndrom deltok i denne studien. Alle gjennomførte tre ulike treningsøkter; ett intervall (1-AIT), fire intervaller (4-AIT) og 47 minutter kontinuerlig moderat trening (CME), på tre separate testdager, med min. 48 timers mellomrom. Hvilemetabolisme ble målt før trening, EPOC ble målt etter trening til hvileverdier var nådd. **Resultater:** En økning i  $O_2$  opptaket over hvileverdier med en varighet på  $70,4 \pm 24,8$  min. (4-AIT),  $35,9 \pm 17,3$  min. (1-AIT) og  $45,6 \pm 17,3$  min. (CME) ble observert. Total gjennomsnittlig EPOC var på  $2,9 \pm 1,7$  l  $O_2$  (4-AIT),  $1,3 \pm 1,1$  l  $O_2$  (1-AIT) og  $1,4 \pm 1,1$  l  $O_2$  (CME). Det var signifikante forskjeller ( $p < 0,05$ ) mellom 4-AIT og CME, og mellom 1-AIT og 4-AIT på varighet og mengde av EPOC, hvor 4-AIT varte lengst i antall minutter og total EPOC i liter var også høyest etterfulgt av CME og 1-AIT. **Konklusjon:** Total EPOC var størst etter 4-AIT. Disse data tyder på at treningens intensitet har en signifikant positiv effekt på EPOC hos menn med metabolsk syndrom.

Nøkkelord: Intensitet, metabolsk syndrom, oksygenopptak, trening.

## Abstract

**Purpose:** Physical activity is central in the prevention and treatment of metabolic syndrome, as effective weight reduction and increased energy expenditure reduce all risk factors associated with metabolic syndrome. High intensity aerobic exercise requires more energy during the activity. It may also induce superior energy expenditure during post-exercise recovery. The primary aim of this study was to compare the excess post-exercise oxygen consumption (EPOC) of three different exercise sessions of aerobic exercise in men with metabolic syndrome after a single exercise session. **Methods:** Seven men aged 39 and 70 years with metabolic syndrome participated in this study. All subjects performed three aerobic exercise treatments: One aerobic interval (1-AIT), four aerobic intervals (4-AIT), and 47 minutes of continuous moderate exercise (CME) in separate days with at least 48 hours between each test day. Resting metabolic rate (RMR) was measured pre-exercise and used as baseline values. EPOC was measured until baseline metabolic rate was re-established. **Results:** An increase in O<sub>2</sub> uptake lasting for 70.4 (± 24.8) min. (4-AIT), 35.9 ± 17.3 min. (1-AIT) and 45.6 ± 17.3 min. (CME) was observed. EPOC was 2.9 ± 1.7 l O<sub>2</sub> (4-AIT), 1.3 ± 1.1 l O<sub>2</sub> (1-AIT) and 1.4 ± 1.1 l O<sub>2</sub> (CME). There were significant differences (p< 0.05) between 4-AIT and CME and 1-AIT. The 4-AIT exercise session produces the highest average EPOC, followed by CME and then 1-AIT. **Conclusion:** Total EPOC was highest after 4-AIT. These data suggest that exercise intensity has a significant positive effect on EPOC in men with metabolic syndrome.

Keywords: Exercise, intensity, metabolic syndrome, oxygen consumption.

- i FULLMAKTSERKLÆRING
- ii NORSK SAMMENDRAG
- iii ABSTRACT

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# 1 Introduction

Metabolic syndrome is a cluster of interrelated risk factors for cardiovascular disease (CVD). It is estimated that approximately 20-25 per cent of the world's adult population suffer from metabolic syndrome (1). The syndrome is associated with the global epidemics of diabetes and obesity (2), and the last two decades have witnessed a stark increase in the number of patients with metabolic syndrome. The components that most commonly identify metabolic syndrome are central obesity, type 2 diabetes, impaired glucose tolerance or impaired fasting glycaemia, insulin resistance, dyslipidemia and hypertension (2). Individuals with metabolic syndrome are at increased risk of developing diabetes type 2 and cardiovascular disease, as well as increased mortality from cardiovascular disease (3). Exercise seems to have positive outcomes concerning preventing and reversing metabolic syndrome (4, 5).

Furthermore, abdominal obesity is closely related to metabolic syndrome. Effective weight reduction and increased energy expenditure decreases all risk factors associated with metabolic syndrome (2). Exercise is one of the key variables that can shift the energy equation in the direction of weight loss (6) and many exercise protocols designed to induce fat loss have focused on regular steady state aerobic exercise at moderate intensity (7). Growing evidence suggest that high intensity exercise has the potential to be an effective exercise protocol for weight reduction in overweight individuals (7).

A significant percentage of total caloric expenditure is due to the resting metabolic rate (RMR), where RMR is the energy expended by the active cell mass to sustain normal body function at rest (8). Previous research indicate that RMR is mainly dependent on fat-free mass (FFM) (9, 10), and some authors claim that chronic exercise produces training adaptations that may lead to higher RMR values (11). Changes in RMR may also occur over time following a single session of exercise (11), and such a session can lead to increased lipid oxidation for several hours following the exercise session (12). High intensity exercise may result in greater fat loss caused by secretion of lipolytic hormones, like growth hormone and epinephrine, which is suggested to cause superior post-exercise energy expenditure and fat oxidation (18). Additionally, research suggests that an exercise intensity of at least 70% of  $VO_{2max}$  is ideal optimal for weight loss (23).

Oxygen consumption is elevated above resting levels after exercise. This state is referred to as excess post-exercise oxygen consumption (EPOC) (13) and has been shown to be influenced by training status, exercise intensity and duration and the thermic effect of food (14, 16). The recovery process is biphasic, with an initial fast phase of recovery, lasting from 10 seconds to a few minutes. This is followed by a slower phase lasting from a few minutes to several hours (13). Several mechanisms are attributed to EPOC, such as replenishment of oxygen stores in muscle and blood, increased circulation and lactate removal, resynthesis of adenosine triphosphate (ATP) and creatine phosphate (CrP), increased heart rate, ventilation, body temperature and triglyceride/fatty acid cycling (14-16).

A number of studies have examined the effect of exercise intensity on EPOC (15-20), and have suggested that  $VO_2$  post-exercise is higher and more persistent for exercise of higher intensity (14). Gore and Withers (21) reported that exercise intensity is the most important determinant of the EPOC, as it explains the EPOC variance five times more than just exercise duration or total work accomplished. Bahr and Sejersted (15) reported a positive and exponential relationship between exercise intensity and EPOC, where the EPOC threshold appears to be at least above 50% of  $VO_{2max}$ .

There are few studies on the effect of exercise on EPOC in obese subjects. One study compared EPOC after short (30 min) exercise in obese versus lean men demonstrating reduced EPOC in obese men (16). Another study demonstrated that the alteration was related to lower growth hormone response and elevated cortisol response to exercise in the obese subjects (22). Exercise  $O_2$  uptake relative to body weight was, however, greater for the low-fat-mass group.

A recent study in healthy young men showed that exercise at 73% of  $VO_{2max}$  for 45 minutes resulted in a significant increase in EPOC, persisting for 14 hours (23). However, others have concluded that the energy expenditure throughout EPOC adds minimally compared to the energy expenditure during the exercise session (21, 24). No studies have focused on EPOC after high intensity interval exercise, though it is suggested that EPOC magnitude is significantly greater when the aerobic exercise sessions were divided into two parts (25, 26). EPOC was significantly greater when 30-minute (25) and 50-minute (26) aerobic exercise sessions were divided into two parts.



The amount of EPOC and post-exercise energy expenditure is suggested to be highest when the body experiences significant physiological stress like that of high intensity aerobic exercise (15, 23, 27). To the best of our knowledge, no studies have investigated EPOC after high intensity interval exercise compared to continuous moderate intensity exercise, in men with metabolic syndrome. This study thus aims to examine if exercise of higher intensity is more effective with regards to increased post-exercise oxygen consumption than moderate intensity exercise.

The primary aim of this study was to investigate post-exercise oxygen consumption after three sessions of aerobic exercise with different intensity, in patients with metabolic syndrome. It was hypothesized that total EPOC was greater after high intensity interval training compared to continuous moderate aerobic exercise and furthermore, that four aerobic intervals increase EPOC more than both continuous moderate exercise and one aerobic interval.

## 2 Methods

### *Subjects*

In total seven men aged 39 to 70 years from the Trondheim area, with metabolic syndrome defined according to the IDF-criteria (1) volunteered to participate in the study. The Regional Ethics Committee, Norway, approved the study. All subjects were fully acquainted with the nature of the study and informed of the experimental risks before signing a written consent form to participate. It was explicitly stated to the subjects that they could withdraw from the study at any point. Exclusion criteria included unstable angina, recently cardiac infarction (4 weeks), uncompensated heart failure, severe valvular illness, pulmonary disease, uncontrolled hypertension, kidney failure, orthopedic/neurological limitations, cardiomyopathy, planned surgery during the research period, reluctance to sign the consent form, drug or alcohol abuse or involvement in another study. The subjects' physiological characteristics and anthropometrics are shown in table 1.

**Table 1.** Physical characteristics of study participants (n=7)

<i>Variables</i>	<i>Mean ± s.d.</i>
Age (years)	56.7 ± 10.8
Height (cm)	181.1 ± 8.6
Body mass (kg)	106.0 ± 12.9
BMI (kg·m <sup>-2</sup> )	32.5 ± 4.6
VO <sub>2max</sub> (ml·kg <sup>-1</sup> min <sup>-1</sup> )	33.2 ± 8.8
HR <sub>max</sub>	174.0 ± 21.4

BMI, body mass index; HR<sub>max</sub>, the peak hearth rate reached during the VO<sub>2max</sub> test plus five beats.

### *Experimental approach to the problem*

In order to test the hypothesis that total EPOC is greater after high intensity interval training compared to continuous moderate exercise (CME) and further, that four aerobic intervals (4-AIT) increase EPOC more than both continuous moderate exercise (CME) and one aerobic interval (1-AIT), all participants completed the three different modes of aerobic exercise, on separate test days.

To equalize training volume (spending the same amount of kcals each session) between 4-AIT and CME, the CME session was performed by 47 minutes at 70% of  $HR_{max}$ , as previously described (28). The order of execution of the three different training sessions was decided by randomization carried out by The Unit of Applied Research unit at the Norwegian University of Science and Technology (NTNU).

### *Pre experimental procedures*

The study's subjects reported to the laboratory for preliminary testing approximately one week before the experiments started. Anthropometrical data was then recorded including height and body weight. Further,  $VO_{2max}$  was measured during uphill running or walking, by employing an individualized protocol using the Jaeger Oxycon Pro (Erich Jaeger, Viasys Healthcare, Germany). Subjects performed a warm up for 10 minutes at approximately 70% of maximum heart rate. Criteria for reaching the true  $VO_{2max}$  was a leveling off of oxygen uptake despite increased workload and a respiratory exchange ratio  $>1.05$  (29). This was achieved for all individuals. The heart rate was monitored by a Polar RS 400 (Polar Electro, Kempele Finland) and the highest HR value during the test was defined as  $HR_{max}$ .

### *Experimental protocol*

Subjects arrived at the laboratory between 07.00 a.m. and 08.00 a.m. on test days following a 12-hr overnight fast. They were instructed not to perform vigorous activity for 48 hours, not to consume alcohol in 36 hours and not to consume any caffeine or nicotine for 12 hours prior to each test day (30). They had their last meal at 08.00 p.m. on the day before testing.

The first 15-20 minutes after arrival to the laboratory the subjects rested, sitting comfortably in a chair. Weight was measured before they underwent a 20 minutes measurement of RMR. After completing the RMR measurements, they performed one exercise session with 1-AIT, 4-AIT or CME. Within five minutes of completing the exercise session subjects returned to the respiratory canopy and EPOC was measured until baseline  $VO_2$  was established. If EPOC lasted for more than 30 minutes, the participants had a ten minute break from the canopy every thirty minutes of measurements. Heart rate was measured continuously during and after exercise until  $VO_2$  reached baseline values.

All subjects performed three test days with at least 48 hours between each. Identical procedures were applied to each test day concerning RMR, post-exercise  $\text{VO}_2$  measurements, but with different aerobic exercise modes for each day of testing.

#### *Resting metabolic rate measurement*

The resting metabolic rate was measured with indirect calorimetry (Vmax29 Sensor Medics, Yorba Linda, CA, USA). Participants rested in a comfortable and supine position in a quiet room with their head enclosed in a Plexiglas canopy on a pleasant bed for approximately 20 minutes until steady state conditions were achieved with a 5-minute period with  $\leq 10\%$  coefficient of variation (CV) for  $\text{VO}_2$  and  $\text{VCO}_2$  (30). The average of the last five 1-minute measurements was used as baseline  $\text{VO}_2$ . A computerized, open-circuit indirect calorimetric system with a ventilated canopy was used to record RMR (Figure 1). Room temperature was kept at a constant 22-24 °C. Prior to each test the cart was calibrated for  $\text{CO}_2$  and  $\text{O}_2$  using certified calibration gases. Volume was calibrated using a calibrated 3-L syringe. The Vmax instrument used a ventilated canopy and mixing chamber and generated  $\text{VO}_2$  and  $\text{VCO}_2$  every minute. The subjects were checked frequently throughout the test to make sure they were comfortable and awake. The canopy and plastic sheet were also checked throughout the test to make sure that the canopy was tightly fitted and there were no air leaks.



**Figure 1.** View of the equipment used in the experiment.

### *Exercise training protocol*

The subjects performed all three aerobic exercise sessions walking or running on a treadmill (Woodway PPS Med, Munich Germany). The adjustments of incline and speed were made on an individual basis according to each subject's fitness level. When subjects performed the interval sessions they warmed up at 70% of  $HR_{max}$  for 10 minutes. Following this, they carried out one (1-AIT) or four intervals (4-AIT) at 85-95% of  $HR_{max}$  with three minutes active recovery period at 70% of  $HR_{max}$ . For the continuously moderate exercise (CME), they performed 47 minutes at 70% of  $HR_{max}$ . All subjects used heart rate monitors to ensure that the exercise was performed at the appropriate intensity.

### *EPOC measurement*

EPOC was measured in  $l\ O_2$  per minute post-exercise until baseline oxygen consumption was established. Substrate utilization was measured by respiratory exchange ratio (RER). When the average of five consecutive 1-min EPOC values was equaled to the baseline  $VO_2$  values, the subject was considered to have reached baseline and the  $VO_2$  measurement were stopped. EPOC duration was considered to be the time from five minutes after completion of exercise to the first minute of the 5-minute average that established baseline (20, 31).

### *Area under the curve calculation*

The trapezium rule (32) was used to calculate the total area under the curve (AUC) for each time period of the EPOC measurements, depending on the individual duration of EPOC. AUC was further determined by subtracting the pre-exercise resting oxygen consumption values for each data collection period from the total AUC. The area under the curve is calculated by adding the areas under the graph between each pair of consecutive observation. Thus we get  $(t_2-t_1) (Y_1+y_2)/2$ , and this is known as the trapezium rule because of the shape of each segment of the area under the curve (32).

### *Statistical analysis*

Statistical analysis was performed using Excel (Microsoft Office for Windows, version 2010) and SPSS software version 18 for Windows (Statistical Package for Social Science, Chicago, IL). The Shapiro-Wilk test showed that all data was normally distributed. A paired t-test was used to test for differences between subjects' responses from the three different exercise protocols. Statistical significance was accepted at  $p < 0.05$ . Results are presented as mean  $\pm$  standard deviation (SD).

## **Results**

### *Baseline VO<sub>2</sub>, EPOC magnitude and EPOC duration.*

Mean Baseline VO<sub>2</sub>, total EPOC magnitude in l O<sub>2</sub> and total EPOC duration in minutes are represented in Table 2. Mean baseline VO<sub>2</sub> values ( $\pm$  SD) for each exercise session were not statistically different at ( $p < 0.05$ ). The 4-AIT exercise session produced the highest average EPOC in l O<sub>2</sub> and duration in minutes, followed by CME and then 1-AIT.

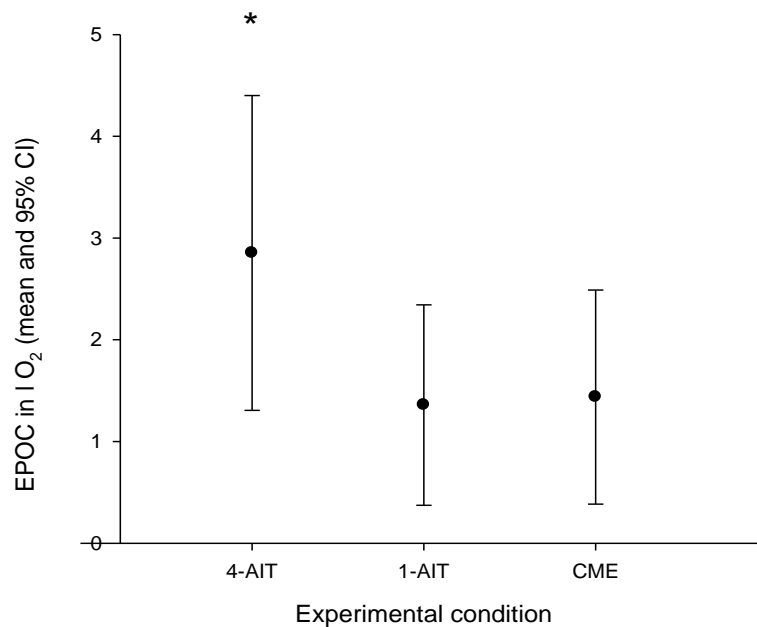
**Table 2.** Baseline VO<sub>2</sub> l/min, duration of EPOC in minutes, and total EPOC in liters for the three different exercise sessions.

<i>Variable</i>	<i>4-AIT</i>	<i>1-AIT</i>	<i>CME</i>
Baseline VO <sub>2</sub> (l/min)	0.25 $\pm$ 0.01	0.24 $\pm$ 0.01	0.24 $\pm$ 0.01
EPOC duration (min)	70.4 $\pm$ 24.8*	35.9 $\pm$ 17.3	45.6 $\pm$ 17.3
EPOC VO <sub>2</sub> (l)	2.8 $\pm$ 1.4*	1.3 $\pm$ 1.1	1.4 $\pm$ 1.1

Values are presented as means  $\pm$  SD. EPOC excess post-exercise oxygen consumption. 4-AIT four aerobic intervals training. 1-AIT one aerobic interval training. CME continuous moderate exercise.

\*Significantly different ( $p < 0.05$ ) from 1-AIT and CME.

There was a significant difference ( $p < 0.05$ ) between 4-AIT and CME and 1-AIT, while there were no significant difference between 1-AIT and CME ( $p > 0.05$ ), both in total magnitude of EPOC in  $l O_2$  and EPOC duration in minutes (fig.2). This indicated that EPOC in liters for 4-AIT was 49.8% higher than for CME, while total mean EPOC in  $l O_2$  for CME was 5.6 % higher than for 1-AIT.

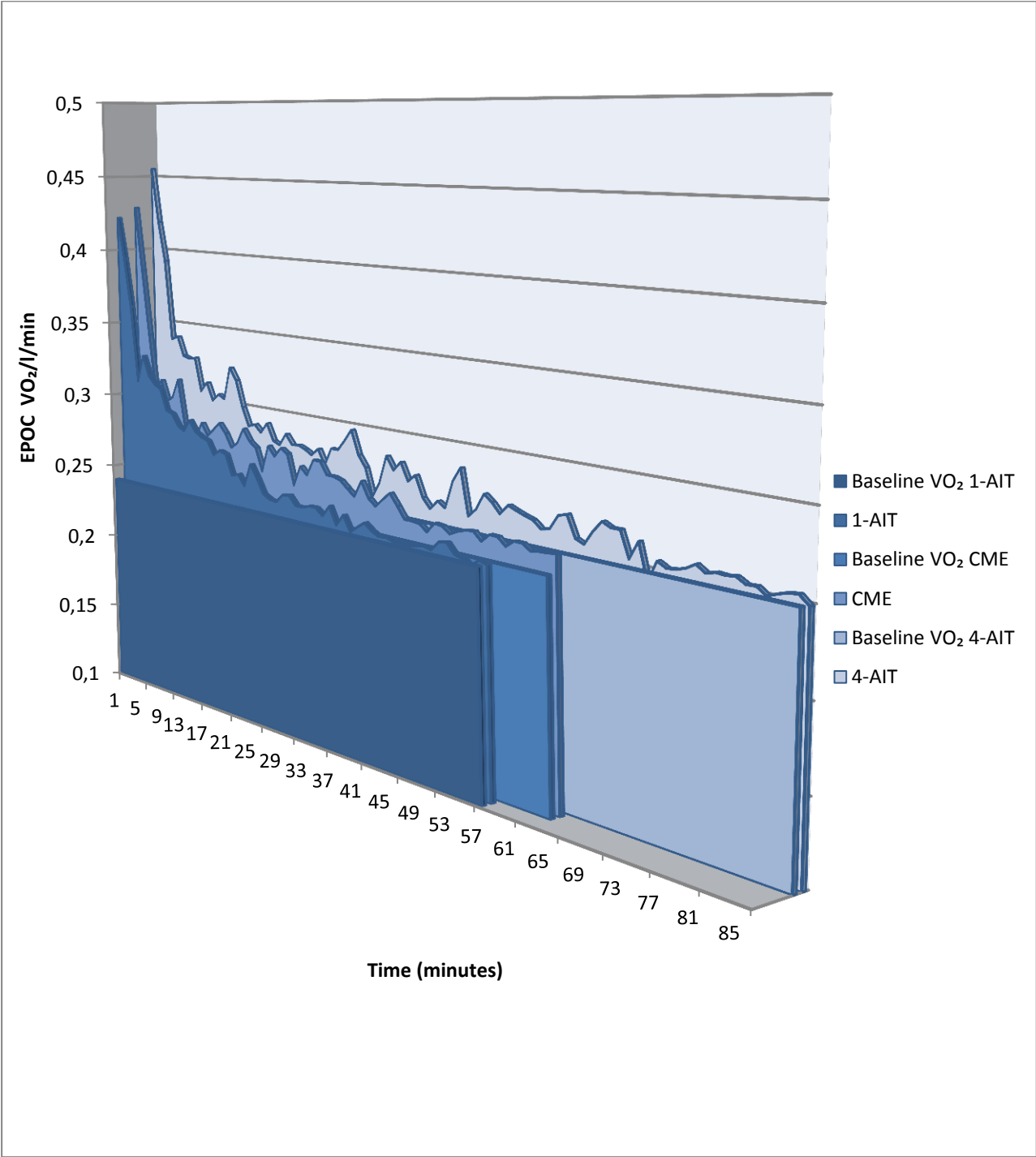


**Figure 2. EPOC in  $l O_2$ , mean and 95% CI.**

EPOC excess post-exercise oxygen consumption, 4-AIT four aerobic intervals training, 1-AIT one aerobic interval training, CME continuous moderate exercise CI confidence interval.

\*Significantly different ( $p < 0.05$ ) from 1-AIT and CME.

After completion of exercise, there was an initial rapid decline in O<sub>2</sub> consumption, but O<sub>2</sub> uptake remained elevated above pre-exercise/baseline O<sub>2</sub> values for 70.4 (± 24.8) min. (4-AIT), 35.8 (± 17.3) min. (1-AIT ) and 45.6 (± 17.5) min. (CME). Figure 3 illustrates the mean recovery curves for the three different exercise sessions.

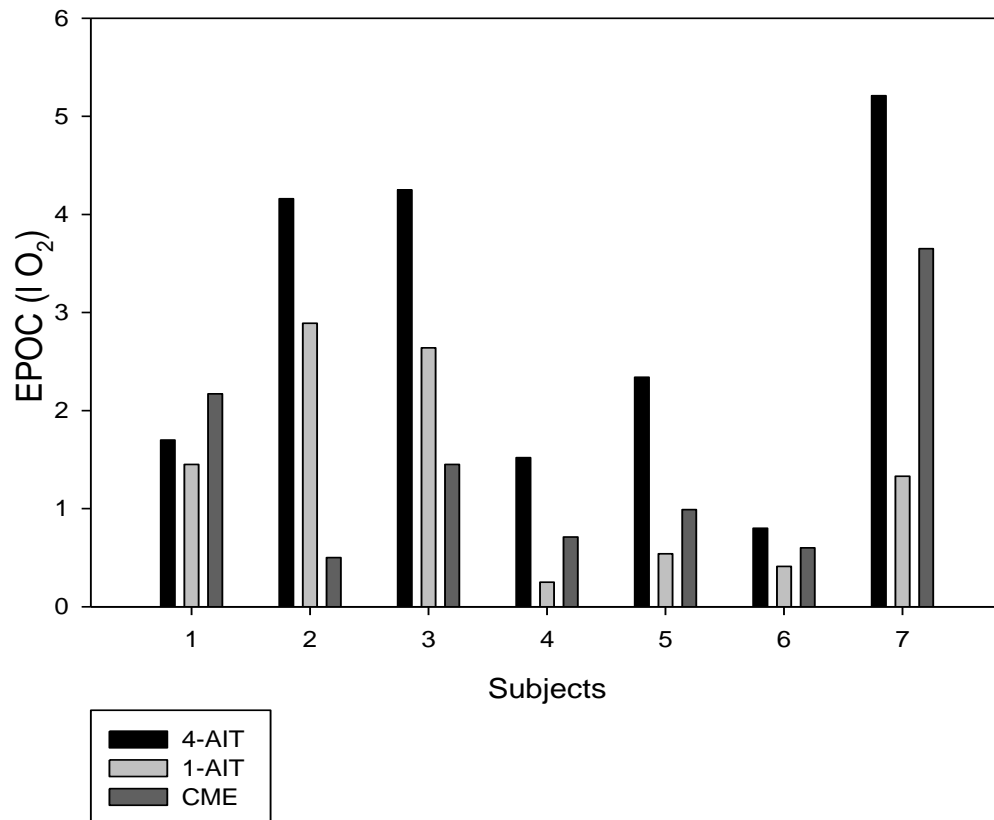


**Figure 3. Area graph of mean excess post-exercise oxygen consumption.**

4-AIT four aerobic intervals training. 1-AIT one aerobic interval training. CME continuous moderate exercise.



Individual EPOC values for the different exercise session are shown in figure 4. Considerable individual variations is observed among the subjects, but there is significant increase in EPOC related to exercise intensity between 4-AIT and CME ( $p=0.040$ ) and between 4-AIT and 1-AIT ( $p=0.017$ ).



**Figure 4. EPOC magnitude presented for all seven subjects.** EPOC excess post-exercise oxygen consumption. 4-AIT four aerobic intervals training. 1-AIT one aerobic interval training. CME continuous moderate exercise.

## 4 Discussion

Physical activity is central in the prevention and treatment of metabolic syndrome, as effective weight reduction and increased energy expenditure reduce all risk factors associated with metabolic syndrome. The aim of this study was to investigate the effect of high intensity interval exercise compared to continuous moderate exercise on EPOC, and whether exercise intensity could beneficially affect the post-exercise energy metabolism in men with metabolic syndrome.

The results of the study revealed that 4-AIT produced the highest total amount of EPOC in men with metabolic syndrome. Further, the results indicated that there was a significant difference between 4-AIT and CME, and between 4-AIT and 1-AIT, while there was no significant difference between 1-AIT and CME.

Evidence from previous research indicates that EPOC can be manipulated by changes in intensity and duration of the exercise sessions (14, 16). In the current study, examining the effect of exercise intensity, the mean EPOC was significantly greater for 4-AIT than for CME and 1-AIT. This result clearly shows that intensity of the exercise session can manipulate the resultant EPOC. Despite contradictory findings concerning the effect of exercise on EPOC, previous research investigating the effect of EPOC on exercise intensity has shown a positive relationship between exercise intensity and EPOC. Gore and Withers (21) found that exercise intensity was the determining factor in elevating EPOC accounting for 45.5% of the systematic variance of EPOC. Bahr and Sejerstedt (15) also reported an exponential increase in EPOC through increasing exercise intensity, where they observed EPOCs of  $1.3 \pm 0.5$ ,  $5.7 \pm 1.7$  and  $30.1 \pm 6.4$  l O<sub>2</sub> after an 80 minute cycling session at 29, 50 and 75% of VO<sub>2max</sub> respectively. The subjects of both studies were physically active men performing a different exercise protocol than the current study, so the amount of EPOC found in their studies may not be directly comparable to ours. Physiological adaptations associated with improved aerobic fitness are considered to be a variable that affects EPOC. Therefore, several studies have compared EPOC in subjects with differing training status. Mostly these studies found there was no EPOC differences between the trained and untrained subjects, despite that they exercised at the same relative intensity (16). However, Chad and Quigley (18) found a greater

EPOC response in trained female cyclists compared to untrained women after 30 minutes cycling at 50% and 70% of  $VO_{2max}$ .

The difference appeared immediately after exercise. This may be explained by higher absolute exercise intensity, thus a higher  $VO_2$  in the trained individuals. When exercise is executed at the same percentage of  $VO_{2max}$ , the trained individuals consume more oxygen than the untrained because of their higher  $VO_{2max}$  (33). Therefore, at the start of the post exercise recovery phase  $VO_2$  is elevated, resulting in a major potential amount for the fast phase of EPOC. The ATP/PCr stores appears to be higher in trained individuals, and because PCr replenishment has been associated with fast phase of EPOC (33), this may explain why exercise of the same relative intensity has a higher effect in endurance-trained individuals. With more  $VO_2$  consumed faster, a trained person may be able reestablish more ATP/PCr. Accordingly, it appears that trained individuals have larger magnitude of fast EPOC, but that total EPOC tends to be similar and total recovery time shorter than in untrained individuals (34). Looking at these results, one can see that the trend of findings is similar concerning the exercise intensity notwithstanding the different training status of the subjects. This is in agreement with the findings related to exercise intensity, presented in this study where EPOC in liters for 4-AIT at 85-90% of  $VO_{2max}$  was 49.8% higher than for CME at 70% of  $VO_{2max}$ .

Concerning the effect on EPOC by duration of exercise, Chad and Wenger (35) suggested that duration of exercise was more important than intensity in determining EPOC. In their study, one group was cycling for 15 and 30 minutes at 70% of  $VO_{2max}$ , while a second group cycled for 38 minutes at 50% of  $VO_{2max}$  (equal total work). This resulted in higher EPOC after the longer sessions for both groups. Later (36) they also suggested that when exercise duration was increased 1.5 and 2 times, EPOC in  $l O_2$  increased by 2.35 and 5.3 times respectively. As the results of our study show, there were no significant differences on EPOC in  $l O_2$  or duration in minutes between the CME sessions lasting for 47 minutes, and the 1-AIT sessions with a total duration of 17 minutes. The CME session was 2.7 times longer than the 1-AIT session, thus our results do not support the findings of Chad and Wenger. When magnitude of EPOC is considered, the manipulation of exercise duration may not be as effective as manipulation of exercise intensity.

Still, the interaction between intensity and duration of exercise is not completely understood, and it may be complicated to separate the effect of each of these variables. However, the exercise intensity has to reach a certain level for the EPOC magnitude to be affected (16). Interestingly, when a constant 50 minute run was compared to two 25 min runs at the same intensity (70% of  $VO_{2max}$ ), the split session significantly increased EPOC (25). Total mean EPOC after the two split sessions was equivalent to 3.1 l  $O_2$  versus 1.4 l  $O_2$  after the continuous session. This reveals that the difference of total EPOC magnitude was relatively small. The 4-AIT session of our study was divided in 4 interval runs with 3 minutes active pause between each interval. This session also produced the highest amount of EPOC, but this is possibly mainly caused by exercise intensity.

Also, inter-individual variability in EPOC response should be taken in consideration. Figure 3 shows each subject's response to exercise at the same relative exercise stimulus on EPOC, and discrepancy among the subjects is obvious. Consequently, it can be hypothesized that there are high, medium and low responders, corresponding to the findings of several parameters in response to exercise where studies have shown marked individual differences in responsiveness to exercise (37). For example age and sex are not main determinants of human responses to exercise, whereas the pre training level of a phenotype has a considerable impact in some cases. Familial factors (shared environment and genetic factors) also contribute significantly to variability in response to exercise (37). This makes it difficult to know whether the observed variability among the subjects is a result of biological variation or caused by measurement errors.

Moreover, to pursue the effect of EPOC on obesity, which is a result of positive energy balance over time, EPOC may play a part to the opposite when strenuous exercise such as 4-AIT is undertaken regularly. However, this has to be investigated further and over time before any conclusion can be made. Furthermore, it has to be noted that the exercise itself plays an important role in weight reduction. Still, protocols focusing on regular steady state exercise with moderate intensity have previously shown negligible weight loss (38). Regular high intensity exercise has been shown to increase aerobic and anaerobic fitness, and further, high intensity exercise also seems to lower insulin resistance, and may result in several muscle adaptations that may enhance skeletal muscle fat oxidation and improve glucose tolerance (7). High intensity exercise have also been shown to increase levels of catecholamines (39), and elevated levels are believed to be partly responsible for EPOC (13).

Interestingly, concentrations of catecholamines do not increase during exercise, except if the intensity of exercise exceeds 70% of  $VO_{2max}$  (7). The response of catecholamines can be an important feature, as especially epinephrine has shown to drive lipolysis, and are largely responsible for the fat release (7).

Concerning physical exercise as prevention and treatment of metabolic syndrome, where effective weight reduction decreases the risk factors associated with metabolic syndrome, it seems appropriate to recommend high intensity interval exercise. Higher exercise intensity training has been shown to be more effective in decreasing risk and preventing metabolic syndrome compared to lower intensity training. A previous study demonstrated that high intensity exercise training was superior to moderate-intensity training in reversing risk factors of metabolic syndrome (40). An increased EPOC in addition to reversing risk factors of metabolic syndrome makes high intensity intervals beneficial in preventing the metabolic syndrome.

There are several methodological concerns that are important to consider in EPOC experiments. An accurate control of the pre-experimental conditions and a precise reproducibility in the RMR measures are fundamental in order to detect small, but possible differences. Børsheim and Bahr (14) determined that body weight, food intake and exercise should be controlled, though pre experimental guidelines were given. Also it is suggested that subjects should sleep overnight in the laboratory before test days to avoid exercise in the morning. Due to limitations concerning the scale of the study some items were difficult to control. This may explain the disparity among the subjects in our study, though it is difficult to identify the factor/s that may have affected the result of our study, even if it was given as strict as possible guidelines as described in the experimental protocol. Time was the main limiting factor in this study, and the number of subjects could be too low for any statistical conclusions and generalizations regarding larger population samples. Because of inter-individual variability in EPOC, it is important with a high enough number of study participants, to be able to detect differences among different exercise protocols.

### *Conclusion*

In conclusion, the results of this study suggest that exercise intensity positively and exponentially affects EPOC in men with metabolic syndrome. Notwithstanding the higher EPOC for 4-AIT, the major contribution of both treatments to weight loss is via the energy expended during the actual exercise, and the exercise should likely be undertaken regularly for EPOC to have an increasing effect on weight reduction.

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