Physical activity is central in prevention and treatment of metabolic syndrome. High-intensity aerobic exercise can induce larger energy expenditure per unit of time compared with moderate-intensity exercise. Furthermore, it may induce larger energy expenditure at post-exercise recovery. The aim of this study is to compare the excess post-exercise oxygen consumption (EPOC) in three different aerobic exercise sessions in men with metabolic syndrome. Seven men (age: 56.7 ± 10.8) with metabolic syndrome participated in this crossover study. The sessions consisted of one aerobic interval (1-AIT), four aerobic intervals (4-AIT), and 47-min continuous moderate exercise (CME) on separate days, with at least 48 h between each test day. Resting metabolic rate (RMR) was measured pre-exercise and used as baseline value. EPOC was measured until baseline metabolic rate was re-established. An increase in O2 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 were 2.9 ± 1.7 L O2 (4-AIT), 1.3 ± 1.1 L O2 (1-AIT), and 1.4 ± 1.1 L O2 (CME). There were significant differences (P < 0.001) between 4-AIT, CME, and 1-AIT. 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.

Metabolic syndrome is a cluster of interrelated risk factors for cardiovascular disease (CVD). It is estimated that approximately 20–25% of the world’s adult population suffer from metabolic syndrome (IDF, 2005). The syndrome is associated with the global epidemics of diabetes and obesity (Eckel et al., 2005), and during the past two decades, we have witnessed a stark increase in the number of patients with metabolic syndrome. Individuals with metabolic syndrome are at increased risk of developing type 2 diabetes and CVD, as well as increased mortality from CVD (Ford et al., 2002). Exercise seems to have positive outcomes concerning preventing and reversing metabolic syndrome (Katzmarzyk et al., 2004, 2005).

Oxygen consumption is elevated above resting levels after acute exercise. This state is referred to as excess post-exercise oxygen consumption (EPOC; Gaesser & Brooks, 1984; Speakman & Selman, 2003) and can lead to increased lipid oxidation for several hours following the exercise session (Henderson et al., 2007).

EPOC has been showed to be influenced by training status, exercise intensity and duration, and the thermic effect of food (Børsheim & Bahr, 2003; LaForgia et al., 2006). 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 (HR), ventilation, body temperature, and triglyceride/fatty acid cycling (Bahr & Sejersted, 1991; Børsheim & Bahr, 2003; LaForgia et al., 2006). A number of studies have examined the effect of cycle-exercise intensity on EPOC (Sedlock et al., 1989; Bahr & Sejersted, 1991; Chad & Quigley, 1991; Sedlock, 1991; Phelain et al., 1997; LaForgia et al., 2006) and have suggested that VO2 post-exercise is higher and more persistent for exercise at higher intensity (Borsheim & Bahr, 2003). Gore and Withers (1990) 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 (1991) reported a positive and exponential relationship between exercise intensity and EPOC, where the EPOC threshold appears to be at least above 50% of VO2max.

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 vs lean men, demonstrating reduced EPOC in obese men (LaForgia et al., 2006). However, others have concluded that the energy expenditure throughout EPOC adds minimally compared with the energy expenditure during the exercise session (Gore & Withers, 1990; LaForgia et al., 1997). No studies have focused on EPOC after high-intensity interval exercise, although it has been shown that EPOC magnitude is significantly greater when an aerobic exercise session is divided into two bouts (Kaminsky et al., 1990; Almuzaini et al., 1998).

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 (Bahr & Sejersted, 1991; Irving et al., 2008; Knab et al., 2011). To the best of our knowledge, no studies have investigated EPOC after high-intensity interval exercise compared with continuous moderate intensity exercise, in men with metabolic syndrome.

The primary aim of this study was to investigate post-exercise oxygen consumption after three sessions of aerobic exercise with different intensity and duration, in patients with metabolic syndrome. It was hypothesized that total EPOC was greater after high-intensity interval training compared with continuous moderate aerobic exercise and, furthermore, that four aerobic intervals (4-AIT) increase EPOC more than both continuous moderate exercise (CME) and one aerobic interval (1-AIT).

**Methods**

Subjects

In total, seven untrained (no regular exercise the last 5 years) men with little or no experience to treadmill interval training, aged 39–70 years (mean age/SD: 56.7 ± 10.8, mean VO2max: 33.2 ± 8.8) from the Trondheim area, with metabolic syndrome defined according to the IDF criteria (IDF, 2005), 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.

Experimental approach to the problem

In order to test the hypothesis that total EPOC is greater after high-intensity interval training compared with CME and, furthermore, that 4-AIT increase EPOC more than both CME and 1-AIT, a crossover design was used, by which all participants completed the three sessions of aerobic exercise, on separate test days.

To equalize training volume (spending the same amount of energy per exercise session) between 4-AIT and CME, the CME session was performed for 47 min at 70% of HRmax, as previously described (Rognmo et al., 2004). The order of execution of the three different training sessions was random, decided by randomization carried out by The Unit of Applied Research at the Norwegian University of Science and Technology (NTNU).

**Pre-experimental procedures**

The study’s subjects reported to the laboratory for preliminary testing approximately 1 week before the experiments started. Anthropometrical data were then recorded, including height and body weight. Furthermore, VO2max was measured in 10-s interval during uphill running or walking, by employing an individualized protocol using the Jaeger Oxycon Pro (Erich Jaeger, Vihasys Healthcare, Hoechberg, Germany). Subjects performed a warm up for 10 min at approximately 70% of maximum HR. Criteria for reaching the true VO2max was a leveling off of oxygen uptake despite increased workload and a respiratory exchange ratio (RER) > 1.05 (Aspenes et al., 2011). This was achieved for all individuals using peak values over 30-s interval. The HR was monitored in 5-s intervals by a Polar RS 400 (Polar Electro, Kempele, Finland) and the highest 15 s HR value during the test was defined as HRmax.

**Experimental protocol**

Subjects arrived at the laboratory between 07:00 h and 08:00 h on test days following a 12-h overnight fast. They were instructed not to perform vigorous activity for 48 h, not to consume alcohol on the last 36 h, and not to consume any caffeine or nicotine for 12 h prior to each test day (Compher et al., 2006). They had their nutrient intake at 08:00 h on the day before testing.

The first 15–20 min after arrival to the laboratory the subjects rested, sitting comfortably on a chair. Weight was measured before they underwent a 20-min measurement of RMR. After completing the RMR measurements, they performed one exercise session with 1-AIT, 4-AIT, or CME. Within 5 min of completing the exercise session, subjects returned to the respiratory canopy and EPOC was measured until baseline VO2 was established. If EPOC lasted for more than 30 min, the participants had a 10-min break from the canopy every 30 min of measurements. HR was measured continuously during and after exercise until VO2 reached baseline values.

All subjects performed three test days with at least 48 h (mean 2.6 ± 0.7 days) between each. Identical procedures were applied to each test day concerning RMR, post-exercise VO2 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, California, 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 min until steady-state conditions were achieved with a 5-min period with ≤10% coefficient of variation for VO2 and VCO2 (Compher et al., 2006). The average of the last five 1-min measurements was used as baseline VO2. A computerized, open-circuit indirect calorimetric system with a ventilated canopy was used to record RMR. Room temperature was kept at a constant 22–24 °C. The subjects were checked frequently throughout the test to make sure they were comfortable and awake.

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. Before each exercise session, the subjects warmed up at 70% of HRmax for 10 min. Following this, they carried out 1 × 4 min interval (1-AIT) or 4 × 4 min intervals (4-AIT) at 85–95% of HRmax with 3-min active recovery period at 70% of HRmax. For the CME, they performed 47 min at 70% of HRmax. All subjects used HR monitors to ensure that the exercise was performed at the appropriate intensity.

EPOC measurement

Five minutes after the exercise was executed, EPOC was measured in liter O2 per minute post-exercise until baseline oxygen consumption was established. EPOC was measured with indirect calorimetry (Vmax29 Sensor Medics). Substrate utilization was measured by RER. When the average of five consecutive 1-min EPOC values was equaled to the baseline VO2 values, the subject was considered to have reached baseline and the VO2 measurement was stopped. EPOC duration was considered to be the time from 5 min after completion of exercise to the first minute of the 5-min average that established baseline (Sedlock, 1991; Lyons et al., 2007).

The trapezium rule was used to calculate the total area under the curve for each time period of the EPOC measurements.

Statistical analysis

All data were checked for normality and are presented as mean and standard deviation. Possible effects of the three different exercise protocols upon EPOC were tested by one-way repeated measures analysis of variance (ANOVA). The measure of association (ω2) was calculated to estimate the percentage of the total variance that can be explained by the influence of the three different exercise protocols. Pairwise comparisons following a significant main effect in the ANOVA involved the Bonferroni adjustment for multiple comparisons to evaluate significant differences in the adjusted means (M) between the exercise protocols. The effect size (ES) was calculated to measure the magnitude of a significant pairwise difference. The statistical power was also calculated. The level of significance was set at an alpha level of ≤0.05. All statistical tests were processed using a dedicated Excel spreadsheet and SPSS 19.0 software for Windows (SPSS Inc., Chicago, Illinois, USA).

Results

The mean VO2max was 33.2 ± 8.8 mL/kg/min. Mean baseline VO2, total EPOC magnitude in liter O2, and total EPOC duration in minutes are shown in Table 2. Mean baseline VO2 did not differ between each test day (P = 0.06, ω2 = 0.05, 1-β = 0.57). There was a significant effect of type of exercise protocol upon duration (P < 0.001, ω2 = 0.33, 1-β = 1.0) and magnitude (P = 0.02, ω2 = 0.18, 1-β = 0.72) of total EPOC. Follow-up tests showed that the 4-AIT exercise protocol (M = 70.4 ± 24.8 min) produced a significantly longer EPOC period than the 1-AIT (M = 35.9 ± 17.3 min, ES = 2.98) and the CME (M = 45.0 ± 17.8 min, ES = 2.19). Follow-up testing also showed that the 4-AIT (M = 2.86 ± 1.67 L O2) had a significantly higher accumulated O2 uptake during the EPOC period as compared with the 1-AIT (M = 1.36 ± 1.06 L O2, ES = 1.53), but not compared with the CME (M = 1.44 ± 1.14 L O2, ES = 1.45).

Figure 1 illustrates the mean recovery curves for the three different exercise protocols.

There was a significant difference (P < 0.05) between 4-AIT, 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 liter O2 and EPOC duration in minutes (Table 2). This indicated that EPOC in liters for 4-AIT was 49.8% higher than that for CME, while total mean EPOC in liter O2 between CME and 1-AIT was not significantly different.

After completion of exercise, there was an initial rapid decline in O2 consumption, but O2 uptake remained elevated above pre-exercise/baseline O2 values for 70.4 (±24.8) min (4-AIT), 35.8 (±17.3) min (1-AIT), and 45.6 (±17.5) min (CME). Figure 1 illustrates the mean recovery curves for the three different exercise sessions.

Discussion

The results of the present study revealed that 4-AIT produced the highest EPOC in men with metabolic syndrome compared with CME and 1-AIT. Furthermore, the results indicated that there was no significant difference between 1-AIT and CME.

Evidence from previous research has shown that EPOC can be manipulated by changing intensity and duration of the exercise sessions (Borsheim & Bahr, 2003; LaForgia et al., 2006). In the current study, examining the effect of different modes of exercise, the mean EPOC was significantly higher for 4-AIT than for CME and 1-AIT. This result clearly shows that the intensity of the exercise session is important in determining EPOC.
Despite the contradictory findings regarding the effect of exercise intensity on EPOC, the majority of the evidence shows a positive relationship between exercise intensity and EPOC (Borsheim & Bahr, 2003). Gore and Withers (1990) found that exercise intensity was the determining factor in elevating EPOC accounting for 45.5% of the systematic variance of EPOC. Bahr and Sejersted (1991) also reported an exponential increase in EPOC through increasing exercise intensity, with EPOCs of \(1.3 \pm 0.5\), \(5.7 \pm 1.7\) and \(30.1 \pm 6.4\) l O\(_2\) after an 80-min cycling session at 29%, 50%, and 75% of VO\(_{2\text{max}}\), respectively. Physiological adaptations associated with improved aerobic fitness are also considered to be a variable that affects EPOC. Therefore, several studies have compared EPOC in subjects with differing training status. Mostly these studies found that there were no EPOC differences between the trained and untrained subjects, even though participants exercised at the same relative intensity (LaForgia et al., 2006). With a higher VO\(_2\) and a higher anaerobic capacity, a trained person may be able to resynthesize more ATP/CrP. Accordingly, it appears that trained individuals have larger magnitude of fast EPOC, but that total EPOC tends to be similar and total recovery time is shorter than that in untrained individuals (Short & Sedlock, 1997).

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 HR\(_{\text{max}}\) was 49.8% higher than for CME at 70% of HR\(_{\text{max}}\).

As the results of our study show, there were no significant differences in EPOC in liter O\(_2\) or duration in minutes between the CME sessions lasting for 47 min, and the 1-AIT sessions with a total duration of 19 min. The CME session was 2.5 times longer than the 1-AIT session, thus our results do not support the findings of Chad and Wenger, which indicate that duration is more important than intensity in determining EPOC (Chad & Wenger, 1988). When magnitude of EPOC is considered, the manipulation of exercise duration may not be as effective as the manipulation of exercise intensity.

Still, the interaction between intensity and duration of exercise is not completely understood, and it may be difficult to separate the effect of each of these variables. However, there is evidence suggesting that exercise intensity has to reach a certain level for EPOC’s magnitude to be affected (LaForgia et al., 2006). Interestingly, when a constant 50-min run was compared with two 25-min runs at the same intensity (70% of VO\(_{2\text{max}}\)), the split session significantly increased EPOC (Kaminsky et al., 1990). Total mean EPOC after the two split sessions was equivalent to 3.1 L O\(_2\) vs 1.4 L O\(_2\) after the continuous session. This reveals that the difference in total EPOC magnitude was relatively small. The 4-AIT session of our study was divided into four interval runs at ∼90% HR\(_{\text{max}}\) with 3-min active pause at ∼70% HR\(_{\text{max}}\) between each interval. This session also produced the
highest amount of EPOC, but this is possibly mainly caused by exercise intensity.

Regular high-intensity exercise has been shown to increase aerobic and anaerobic fitness, and furthermore, 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 (Boutcher, 2011). High-intensity exercise has also been shown to increase levels of catecholamine’s (Zouhal et al., 2008), and the elevated levels are believed to be partly responsible for EPOC (Gaesser & Brooks, 1984).

Concerning physical exercise as prevention and treatment of metabolic syndrome, it seems appropriate to recommend high-intensity interval exercise. Higher intensity exercise training has been shown to be more effective in decreasing risk and preventing metabolic syndrome compared with lower intensity exercise training. A previous study demonstrated that high-intensity exercise training was superior to moderate-intensity training in reversing risk factors of metabolic syndrome (Tjonna et al., 2008). High-intensity intervals seems beneficial in preventing the metabolic syndrome by reversing risk factors constituting metabolic syndrome and increasing EPOC.

Limitations of the study

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 measurements are fundamental in order to detect small, but possible differences. Børsheim and Bahr (2003) determined that body weight, food intake, and exercise should be controlled, although 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 aspects were difficult to control. This may explain the interindividual variation in EPOC in our study, although it is difficult to identify the exact factors involved, even if a strict protocol was followed, as described in the Method section. Because of interindividual variability in EPOC, it is important to have a large sample size (and we only had seven participants) to be able to detect differences among exercise protocols. Other limitations to this study could be the wide age range between the subjects and potential training effect within three sessions over such a short time period.

Perspectives

Our results show that 4-AIT induces higher EPOC compared with CME and 1-AIT, and this support previous evidence in the area, indicating that EPOC can be manipulated by changes in intensity and duration of exercise, with a positive effect of intensity on EPOC. However, when comparing intensity and duration, we saw that 1-AIT compared with CME gave similar results in EPOC, even though 1-AIT exercised for 19 min compared with 47 min in the CME group. So training using a relatively brief, single bout of vigorous exercise may be a time-efficient strategy to improve health in individuals with metabolic syndrome. Such small bouts of exercise could be translated into public health policies and programs.

In conclusion, the results of this study suggest that 4-AIT induces a larger EPOC compared with CME and 1-AIT.

Key words: Energy expenditure, untrained subjects, aerobic interval training, continuous moderate exercise.

Disclosure

None.

References

Exercise and post-exercise oxygen consumption


IDF. The IDF consensus worldwide definition of the metabolic syndrome. International Diabetes Federation, 2005.


