Increases in maximal accumulated oxygen deficit following high-intensity interval training are not gender dependent

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Short title: MAOD, TRAINING, and GENDER

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Abstract

Gender differences in maximal accumulated oxygen deficit (MAOD) were examined before and after 4 and 8 wk of high-intensity interval training. Untrained men (n=7) and women (n=7) cycled at 120% of pre-training peak oxygen uptake (\(\dot{VO}_2\) peak) to exhaustion (MAOD test) pre-, mid-, and post-training. A post-training timed test was also completed at the MAOD test power output, but this test was stopped at the time to exhaustion (TE) achieved during the pre-training MAOD test. The 14.3±5.2% increase in MAOD observed in males after 4 wk of training was not different from the 14.0±3.0% increase seen in females (p>0.05). MAOD increased by a further 6.6±1.9% in males and this change was not different from the additional 5.1±2.3% increase observed in females after the final 4 wk of training.

Peak \(\dot{VO}_2\) measured during incremental cycling increased significantly (p<0.01) in male but not in female subjects after 8 wk of training. Moreover, the accumulated oxygen uptake (AO\(_2\) uptake) was higher in men during the post-training timed test compared to the pre-training MAOD test (p<0.01). In contrast, the AO\(_2\) uptake was unchanged from pre- to post-training in female subjects. The increase in MAOD with training was not different between men and women suggesting an enhanced ability to produce ATP anaerobically in both groups. However, the increase in \(\dot{VO}_2\) peak and AO\(_2\) uptake obtained in male subjects following training indicates improved oxidative metabolism in men but not in women. We conclude that there are basic gender differences that may predispose males and females to specific metabolic adaptations following a period of intense interval training.

Key words: Blood lactate concentration, males and females, supramaximal cycling, anaerobic capacity, active muscle mass
Introduction

The maximal amount of ATP that can be produced through anaerobic metabolism during a supramaximal exercise bout has been defined as a person's anaerobic capacity (AC) (6). Several researchers have suggested that the maximal accumulated oxygen deficit (MAOD), measured during 2-3 min of exhaustive exercise, is an accurate method of quantifying an individual's AC (14,15,26). Medbø and Burgers (11) reported a 16% increase in MAOD for males following 6 wk of high-intensity interval training (HIT), whereas females did not significantly improve their MAOD. Medbø and Burgers (11) speculated that AC might be more "trainable" in males than in females.

Exercise tests that are shorter in duration (e.g., 20-30 s) have also been used to examine anaerobic metabolism before and after intense interval training. Campbell et al. (3) demonstrated significant improvements in the peak power output achieved during a 20-s sprint cycling test for female subjects after 6 wk of training. Also, a significant increase in the peak power attained during a Wingate Anaerobic Test was demonstrated in females, but not in males following 4 wk of HIT (4). While all-out sprint exercise tests of less than 1 min in duration do not provide direct information about AC (6,31), these studies suggest that female subjects are able to increase the rate of anaerobic energy release during short-term exercise after HIT. Although it has been suggested that there is a close relationship between the rate of anaerobic energy release and MAOD (11), the lack of improvement in MAOD by female subjects is inconsistent with the significant increases in peak power obtained during sprint exercise reported after training in other studies.
There is no strong evidence to suggest that changes in anaerobic ATP production are different in men and women following a period of HIT. Furthermore, it is unclear if changes in oxidative metabolism after intense interval training are gender dependent. While several investigators have demonstrated an increase in oxidative enzyme activity and/or peak oxygen uptake (\( \dot{V}O_2 \) peak) in male subjects after sprint-type training \( (8,18,21,25) \), others have reported no change in aerobic energy production following HIT \( (4,16) \). Less is known about changes in oxidative metabolism in female subjects following short-term HIT. Ready et al. \( (20) \) demonstrated an 8% increase in maximal \( \dot{V}O_2 \), whereas Campbell et al. \( (3) \) found no change in \( \dot{V}O_2 \) peak after short-term sprint cycle training in female subjects. Furthermore, we are unaware of any study that has compared the aerobic contribution to a bout of intense cycling between men and women both before and after sprint-type training. Increased aerobic metabolism during high-intensity cycling could reduce the reliance on anaerobic energy production and consequently delay fatigue.

The purpose of the present study was to examine the MAOD in male and female subjects before and after 4 and 8 wk of HIT. Furthermore, this study investigated the effects of intense interval training on peak \( \dot{V}O_2 \) and aerobic energy production during 2-3 min of exhaustive cycling performed at 120% of \( \dot{V}O_2 \) peak in men and women. We hypothesized that the changes in MAOD after 4 and 8 wk of HIT are not gender dependant and that oxidative metabolism would increase following intense interval training in both groups.
Methods

Subjects. Seven untrained male and 7 untrained female subjects volunteered to participate in the present study. Subjects were considered untrained if they were not training and had not regularly participated or competed in a sport for 24 months. Additionally, no subject had a past history of highly competitive sport. Subjects were familiarized with the experimental procedures and provided written informed consent prior to testing. The Griffith University Ethics Committee for Human Experimentation approved the testing procedures used in this study. All female subjects had regular menstrual cycles and performed the pre-, mid- and post-training cycling tests in the follicular phase of their menstrual cycle. It is important to control for the potential effect of menstrual cycle status on exercise performance (27) and it has been demonstrated previously that the peak power output obtained during sprint cycling may be lower during the luteal phase compared to the follicular phase (17).

Experimental protocol. The present study involved 8 wk of intense interval training. Subjects performed 6 submaximal cycling tests 2 wk prior to the initiation of training in order to determine their \( \dot{\text{VO}}_2 \)-power relationship. In the week preceding the commencement of training (pre-training), the subjects \( \dot{\text{VO}}_2 \text{peak} \) was measured and the MAOD for cycling was determined at least 2 d later. MAOD was also determined after 4 wk of training (mid-training). Following 8 wk of HIT (post-training), subjects completed an additional cycling test (post-training timed test) 48 h after the final training session. The post-training timed test was performed at the same power output used in the MAOD tests, but the test was stopped at the time to exhaustion (TE) achieved during the pre-training MAOD test. Subjects rested for 2 d before the post-training MAOD test was conducted. After an additional 48 h of rest,
the \( \dot{V}O_2 \) peak was measured. The active muscle mass (AMM) for cycling was measured pre- and post-training.

**Determination of peak \( \dot{V}O_2 \).** The \( \dot{V}O_2 \) peak for cycling was measured using a continuous ramp protocol conducted on a Lode electronically-braked cycle ergometer (Excalibur Sport V2.0, Groningen, The Netherlands). Pedal rate was maintained at 70 rev·min\(^{-1}\) and the power output was increased by 20 W·min\(^{-1}\) for females and by 25 W·min\(^{-1}\) for males until exhaustion. Heart rate (HR) was monitored continuously during exercise using an electrocardiograph (Lohmeier M 607, Munich, Germany) and \( \dot{V}O_2 \) was measured breath-by-breath (MedGraphics\textsuperscript{®} Cardiorespiratory Diagnostic Systems, St. Paul, MN, USA) and averaged over 30-s intervals. The two highest 30-s values for \( \dot{V}O_2 \) were averaged and reported as the \( \dot{V}O_2 \) peak for cycling.

**Submaximal exercise bouts.** Steady-state \( \dot{V}O_2 \) was measured at 6 submaximal power outputs between 20 and 75% of \( \dot{V}O_2 \) peak prior to training. Subjects cycled at 70 rev·min\(^{-1}\) for 10 min and the \( \dot{V}O_2 \) values measured at min 9 and 10 were averaged and reported as the steady-state \( \dot{V}O_2 \) for the corresponding power output. Data collected from the 6 submaximal bouts were used to establish the \( \dot{V}O_2 \)-power relationship for cycling. The linear regression of the \( \dot{V}O_2 \)-power relationship was used to calculate the power output that corresponded to 120% of \( \dot{V}O_2 \) peak. This power output was then used in all subsequent MAOD tests (pre-, mid- and post-training) and in the timed cycling test conducted after training.
The MAOD test and post-training timed cycling test. Subjects warmed up by cycling on a Lode cycle ergometer for 5 min at 50 W for males and at 35 W for females. Subjects were then asked to rest quietly on the cycle ergometer for 5 min. Immediately prior to the MAOD test, the subject’s hyperemic earlobe was sterilized with 70% ethanol and punctured with a 1.5 mm lancet (Microlancet, Becton Dickson, Sandy, UT, USA). The first drop of blood was wiped away and 50 μL of free-flowing blood was collected in a capillary tube and immediately dispensed into a pre-chilled Eppendorf tube for subsequent analysis of blood lactate concentration ([La\(^{-}\)]) (Yellow Spring Instruments, 2700 SELECT, OH, USA). Following 2 min of unloaded cycling at 70 rev-min\(^{-1}\), the pre-determined power output of 120% \(\dot{V}_{\text{O}_2}\) peak was applied immediately. Heart rate was monitored continuously while \(\dot{V}_{\text{O}_2}\) and minute ventilation (\(\dot{V}_{E}\)) were measured breath-by-breath throughout the exercise bout. Subjects were required to maintain pedal cadence at 70 rev-min\(^{-1}\) throughout the MAOD tests and the test was terminated when the subject could no longer maintain a pedal cadence of 60 rev-min\(^{-1}\) despite verbal encouragement. Blood samples were obtained for subsequent lactate analysis 3 min after ([La\(^{-}\)]\(_{3\text{ min}}\)) the MAOD test while the subject cycled at 50 W for males and at 35 W for females.

The accumulated oxygen deficit (AO\(_{2}\) deficit) was calculated as the difference between the accumulated oxygen demand (AO\(_{2}\) demand) and the accumulated oxygen uptake (AO\(_{2}\) uptake) measured during the MAOD test (13). The AO\(_{2}\) deficit calculated for the MAOD test was reported as the “maximal AO\(_{2}\) deficit” (MAOD) for cycling, whereas the AO\(_{2}\) deficit measured during the post-training timed test was not “maximal” as subjects did not cycle to exhaustion. Absolute MAOD values were decreased by 9% to correct for reductions in the \(\text{O}_2\) stores of the body (13). Weber and Schneider (29) have demonstrated that this method...
of determining MAOD for cycling is highly repeatable in untrained male and female subjects (intra-class correlation coefficients of 0.983 for TE and 0.968 for MAOD values). Changes in MAOD determined after 4 and 8 wk of training were reported as the percent increase calculated from pre- to mid-training, mid- to post-training and pre- to post-training.

**Training protocol.** Training was performed on a basket-loading Monark cycle ergometer (Monark Ergomedic 824E, Varberg, Sweden) so the load could be applied immediately. Subjects trained 3 d/wk for a total of 8 wk. The training sessions consisted of three, 2-min constant-load cycling intervals performed at 70 rev-min⁻¹. Recovery between intervals was set at 6 min. All training parameters (recovery time, number of intervals and cadence) except cycling intensity, were kept constant throughout the 8-wk training period. The intensity of training began at 82.5% of the power output used in the MAOD tests for each subject and was increased by 2.5% of the initial workrate every week. Each subject was training at an intensity equal to 100% of the power output used in the MAOD test by wk 8 of training. Peak HR was measured during each 2-min cycling interval using a Polar Beat® (Polar Electro Oy, Kempele, Finland) heart rate monitor. Blood [La⁺] was determined during the first training session in week 1 and during the third session in weeks 4 and 8 for all subjects. Blood samples were collected at rest and 3 min after the third cycling repetition of the training session.

**Determination of AMM.** Body composition was assessed pre- and post-training using dual energy X-ray absorptiometry (DEXA) (Norland XR36, Fort Atkinson, WI, USA). The total lean mass for both legs and the gluteal muscle group was measured and reported as the AMM for cycling. The gluteal muscle mass has been shown to be one of the major muscle groups involved in cycling (19) and has been largely ignored when traditional methods of
determining the AMM for cycling are used (30). AMM is reported independently of fat mass and bone mineral content (BMC). Body composition values obtained using DEXA were used to express MAOD relative to the AMM for cycling in each subject.

Statistical analysis. MAOD test variables (MAOD, TE and HR) were examined using a 2 x 3 (gender – between group factor; pre-, mid-, and post-training – within group factors) ANOVA with repeated measures for training. Increases (%) in MAOD and TE measured for males and females after 4 wk and again after 8 wk of training were compared using a 2 x 2 ANOVA with repeated measures. In addition, a 2 x 2 ANOVA with repeated measures was used to examine gender differences in peak exercise values measured pre- and post-training. Post-hoc analyses were performed where appropriate using pairwise comparisons with Bonferroni adjustments. A linear regression analysis was used to determine the $\dot{VO}_2$-power relationship for the 6 submaximal cycling bouts. Statistical significance was accepted at p<0.05.

Results

Body composition. The physical characteristics of the subjects determined before and after 8 wk of training are presented in Table 1. Body mass (BM) did not change significantly in male or female subjects with training. In addition, the determination of body composition using DEXA did not reveal any significant changes in the AMM for cycling in either group following 8 wk of HIT.

Peak cardiorespiratory values obtained during incremental cycling. The peak exercise values obtained during incremental cycling pre- and post-training are presented in Table 2.
Male subjects obtained a significantly higher absolute \( \dot{V}O_2 \) peak (L-min\(^{-1}\)) than female subjects before training. The gender difference remained significant when \( \dot{V}O_2 \) peak values were expressed relative to BM (M, 44.4±2.4 v F, 39.6±0.9 mL·kg\(^{-1}\)·min\(^{-1}\), p<0.05). \( \dot{V}O_2 \) peak increased significantly in males after 8 wk of training, whereas the change in \( \dot{V}O_2 \) peak with training was not significant in females. Both male and female subjects obtained significantly higher peak power outputs after training than before training. There were no significant gender differences in peak HR before training and the peak HR obtained during incremental cycling did not change with training in either group.

**MAOD test results.** Table 3 presents the MAOD and TE values determined pre-, mid- and post-training in male and female subjects. Males obtained a greater absolute MAOD for cycling than females pre-, mid- and post-training. In addition, when MAOD was expressed relative to the AMM for cycling, males maintained a higher MAOD than females pre- (M, 132.2±4.5 v F, 116.5±6.6 mL·kg AMM\(^{-1}\), p<0.05) and post-training (M, 166.0±10.6 v F, 142.7±7.1 mL·kg AMM\(^{-1}\), p<0.05). Both male and female subjects demonstrated a significant increase in MAOD after only 4 wk of training (pre- to mid-training). Following the final 4 wk of training (mid- to post-training), both male and female subjects demonstrated an additional increase in MAOD. There was no gender-dependent difference in the percent increase in MAOD at either 4 or 8 wk of training. The total increase in MAOD of 21.9±6.3% for males was not different from the 19.6±3.1% increase obtained for females after 8 wk of training. There was no difference between male and female subjects in the percent increase in TE after 4 or 8 wk of training.
Peak HR values attained during the pre-training MAOD test were not different between males (187±3 beats·min⁻¹) and females (188±3 beats·min⁻¹) and there was no change in peak HR for either group after 8 wk of training. Pre-training, blood [La]₃_min for males was significantly higher than for females (p<0.001). Following 8 wk of training, blood [La]₃_min increased to 19.9±0.9 mmol·L⁻¹ in males (p<0.01) and to 16.0±0.6 mmol·L⁻¹ in females (p<0.01), but [La]₃_min remained significantly higher in male than in female subjects. However, the percent increase in blood [La]₃_min from pre- to post-training was not different between the two groups.

**Blood La⁻ and HR responses to a training session.** Blood [La]₃_min measured after the third cycling repetition of the three training sessions was significantly higher in males than in females pre- (M, 14.3±0.5 v F, 11.5±0.6 mmol·L⁻¹, p<0.01), mid- (M, 14.6±0.6 v F, 12.5±0.4 mmol·L⁻¹, p<0.01) and post-training (M, 15.8±0.4 v F, 12.7±0.3 mmol·L⁻¹, p<0.001). However, the significant increase of 9.6±3.0% (p<0.05) in blood [La]₃_min reported for males was not different from the significant increase of 9.6±4.6% (p<0.05) measured in females following 8 wk of training. The peak HR recorded during the third cycling interval of a training session in wk 1 was not different between male (183±4 beats·min⁻¹) and female (185±4 beats·min⁻¹) subjects. There was no significant change in the peak HR obtained during a training session after 8 wk of HIT for either male (184±3 beats·min⁻¹) or female (185±3 beats·min⁻¹) subjects.

**Pre-training MAOD test and post-training timed cycling test.** The mean AO₂ deficit determined during the post-training timed cycling test (see Table 4) was significantly lower than the AO₂ deficit achieved during the pre-training MAOD test in male subjects. In contrast, the AO₂ deficit was not different between the two tests in female subjects. The AO₂
uptake measured during the timed cycling test after training was significantly higher when compared to the AO2 uptake obtained during the pre-training MAOD test in male subjects. There was no change in AO2 uptake in female subjects as a result of training. During the post-training timed cycling test, mean $\dot{V}_E$ was significantly ($p<0.01$) lower when compared to the pre-training MAOD test in both male (post-training timed, 87.6±4.0 v pre-training, 109.7±6.2 L·min$^{-1}$) and female (post-training timed, 63.9±4.1 v pre-training, 75.6±3.9 L·min$^{-1}$) subjects. The peak HR obtained for both male and female subjects was significantly lower during the timed cycling test after training compared to the pre-training MAOD test. Blood $[\text{La}]^3_{\text{min}}$ was observed to be 21.1±7.2% lower in males and 15.8±4.1% lower in females during the post-training timed cycling test compared to the pre-training MAOD test. The relative decrease in blood $[\text{La}]^3_{\text{min}}$ during the post-training timed test was not different between the two groups.

Discussion

The primary finding of this study is that the increase in MAOD after 4 and 8 wk of intense interval training was not gender dependent. The total increase in MAOD of 21.9±6.3% for males and the 19.6±3.1% for females in the present study is comparable to the 16-28% increase in MAOD measured for male subjects in previous HIT studies (7,11,26). However, the present study is the first to demonstrate a significant increase in MAOD with training in untrained female subjects.

Prior to training, males demonstrated a significantly higher MAOD than females even when values were expressed relative to the AMM for cycling. This finding is consistent with our
earlier study in untrained men and women (28). Higher MAOD (mL·kg AMM−1) values in men suggest differences between untrained men and women in morphological and/or biochemical skeletal muscle characteristics. While Esbjörnsson et al. (4) failed to show any gender-related difference in muscle phosphofructokinase (PFK) activity measured before 4 wk of HIT, PFK activity as well as the proportion of type II muscle fibers have previously been demonstrated to be higher in male than in female subjects (2,14,23). These factors may contribute to a greater ability to produce ATP anaerobically during sprint-type exercise in males than in females. Despite such gender differences in muscle enzyme activity and fiber type, the increases observed in MAOD and blood [La]_{3\text{min}} in the present study, suggest that the ability to increase anaerobic ATP production in response to HIT is not different between men and women.

Both male and female subjects achieved large increases (~14%) in MAOD after 4 wk followed by a smaller increase (~5-7%) after the final 4 wk of training. This time-course of change is similar to that reported by Tabata et al. (26) for male subjects after 4 wk (23%) and a further 2 wk (5%) of intense interval training. Similarly, Ready et al. (20) reported that the greatest increment in peak blood [La] in female subjects occurred in the first 2 wk of a 6 wk sprint-training program. The time-course of changes in MAOD found for men and women in the present study support the concept that the adaptive response to training becomes less with time (1).

It has been demonstrated that the resting muscle content of creatine phosphate and ATP as well as the degradation of these high-energy phosphates during sprinting are unchanged after 8 wk of HIT (16). In addition, energy derived from anaerobic glycolysis has been reported to contribute about 70-80% of the MAOD (12,13). Thus, an increase in anaerobic
glycolysis is likely to be the main metabolic process accounting for the greater MAOD following training. The possible mechanisms that contribute to the increase in MAOD following HIT may include an increase in glycolytic flux rate due in part, to an increased activity of glycolytic enzymes such as PFK and lactate dehydrogenase (LDH). However, some researchers have reported increases in PFK activity after HIT (8, 18, 25), whereas others have reported no change in PFK activity with sprint-type training (4). Nevertheless, PFK activity has been reported to be greater in trained compared to untrained men (22). In addition, Esbjörnsson et al. (4) reported that total LDH activity increased by the same relative amount in male and female subjects following 4 wk of HIT. This suggests an increased glycolytic rate and an enhanced ability to stimulate anaerobic ATP production in both men and women after intense interval training. Furthermore, McKenna et al. (10) demonstrated improved skeletal muscle potassium regulation with intense sprint training. While the relationship between plasma potassium concentration and work output requires further investigation, improved potassium regulation by skeletal muscle is consistent with reduced fatigability after sprint training. Alternatively, an improvement in MAOD following intense training may be explained in part by an increase in muscle strength. While the anthropometric measurements obtained from DEXA in the present study did not indicate a significant change in muscle mass with training, an increase in muscle recruitment could contribute to an enhanced ability to sustain anaerobic energy production.

In contrast to the findings of the present study, Medbø and Burgers (11) failed to demonstrate a significant increase in MAOD for women following 6 wk of HIT, whereas men achieved a 16% increase. Several limitations in the investigation by Medbø and Burgers (11) could account for these conflicting observations. Medbø and Burgers (11) divided five men and seven women into two different training groups. They did not state if subjects completed
the same volume of training, or if the response to the different training protocols was gender dependent. In addition, male and female subjects achieved similar peak [La] following the pre-training MAOD test. This suggests that the female subjects were more anaerobically trained than the male subjects given that women have been reported to have lower peak [La⁻] than men following high-intensity activity when equally trained (4,15,28). Therefore, any improvement in MAOD may have been attenuated in female subjects because the training response may be less in subjects closer to their upper limit of performance. Furthermore, Medbø and Burgers (11) made no attempt to control for menstrual cycle phase as the pre- and post-MAOD tests were 6 wk apart. Parish and Jakeman (17) demonstrated that women achieved a greater mean and peak power output for a sprint cycling test during the follicular phase of their menstrual cycle when compared to values recorded during the luteal phase. While the magnitude of the effect of menstrual cycle status on intense exercise remains unclear, Tarnopolsky (27) has suggested that researches should consider the potential effects of menstrual cycle status on exercise performance when designing research studies. The present study tested female subjects in the follicular phase of their menstrual cycle and testing sessions were 4 wk apart to control for any effect of menstrual cycle phase on exercise performance.

Few other studies have examined the changes in anaerobic ATP production pre- and post-training in female subjects. Ready et al. (20) demonstrated a 20% increase in maximal "oxygen debt" as well as a concomitant increase in peak [La⁻] following 6 wk of intense cycle training in females. In addition, some researchers have measured the total work performed during 20-30 s of sprint cycling to determine "anaerobic performance capacity" in female subjects before and after 6 wk of sprint cycle training (3,4). These investigations report
significant improvements in the ability of female subjects to produce ATP anaerobically with training. However, the interpretation of these results should be questioned as several researchers indicate that both maximal oxygen debt and total work achieved during short-term (<30 s) cycling are not a valid measure of AC (6,24).

Medbø and Tabata (12) suggested that about 65% of the energy required for an exhaustive 2 min exercise bout is provided by aerobic energy systems. Thus, an increase in aerobic power found in response to HIT by some researchers is not surprising (8,9,21,25). We found a significant increase of 7.9±2.0% in peak VO$_2$ in male subjects following 8 wk of training. However, the present study failed to demonstrate a significant improvement in VO$_2$peak in female subjects in response to HIT. This finding is in agreement with the results reported by Campbell et al. (3) who found no change in VO$_2$max after 6 wk of sprint cycle training in female subjects.

The increase in VO$_2$peak demonstrated in men but not in women in the present study, suggests that male subjects increased maximal cardiac output and/or increased maximal oxygen extraction in response to training to a greater extent than female subjects. Improvements in VO$_2$peak reported in men after HIT have been related to increases in oxidative enzyme activity (8,18,21) and muscle blood flow (9). However, Esbjörnsson et al. (4) reported no change in the activity of oxidative enzymes following 4 wk of HIT, for either men or women. Alternatively, it has been suggested that women may have a more rapid recovery between training repetitions compared to men as indicated by the lower accumulation of inosine monophosphate and inosine following high-intensity exercise (5). This would allow each successive training repetition to be performed more anaerobically in
females compared to males. Thus, female subjects in the present study may have placed less stress on the aerobic energy system during this type of training than male subjects. It is clear that further research is required to examine the mechanisms that control gender-specific changes in peak \( \dot{V}O_2 \) with intense interval training.

Nevertheless, the improvement of \( \dot{V}O_2 \) peak in male subjects may be associated with the greater \( AO_2 \) uptake observed in men during the timed cycling test when compared to the pre-training MAOD test. Harmer et al. (7) measured the \( AO_2 \) uptake in male subjects during exhaustive cycling at 130% of \( \dot{V}O_2 \) peak during a timed test conducted after 7 wk of HIT. In contrast to the present study, they reported no change in \( AO_2 \) uptake in male subjects with training. However, Harmer et al. (7) used 30 s training intervals whereas we used 2 min training repetitions. Oxidative ATP generation would be greater with longer repetitions, perhaps explaining the resultant increase in \( AO_2 \) uptake observed in male subjects in the present study.

It has been suggested that a higher \( V_E \) obtained after HIT may contribute to a greater \( \dot{V}O_2 \) and improved acid-base regulation during sprinting (9). However, it is unlikely that the greater \( AO_2 \) uptake seen in male subjects during the post-training timed test, compared to the pre-training MAOD test, could be accounted for by an increase in \( V_E \) since mean \( V_E \) was actually decreased in the timed test. Further evidence that an increase in \( AO_2 \) uptake in males was not secondary to changes in cardiorespiratory function relates to the fact that the relative decrease in mean \( V_E \) and peak HR during the timed test was not different between men and women. Nonetheless, no change in \( AO_2 \) uptake was found in female subjects with training. These findings suggest that adaptations in \( V_E \) and HR did not contribute to the
gender-specific increase in \( \text{AO}_2 \) uptake observed in males during the post-training timed test. Alternatively, the increase in \( \text{AO}_2 \) uptake in male subjects could have been a result of enhanced skeletal muscle oxygen extraction after training. McKenna and colleagues (9) suggested improved gas exchange in the active musculature during sprinting following 7 wk of HIT in men. Thus, the unchanged \( \text{AO}_2 \) uptake observed in female subjects in the present study suggests that skeletal muscle oxygen extraction was not improved with training. In the absence of cardiac output and arteriovenous oxygen difference measurements, it is difficult to speculate about the possible mechanisms that account for gender-specific training adaptations in aerobic metabolism.

Females demonstrated a similar decrease in blood \( [\text{La}]_{3\text{min}} \) compared to males during the post-training timed test. In light of these results, it is possible that female subjects achieved an increase in cycling efficiency due in part to a decrease in the energy expenditure of the respiratory and/or stabilizing musculature of the upper body. This would allow an increased active muscle \( \dot{\text{VO}}_2 \) without any change in whole body \( \dot{\text{VO}}_2 \) in female subjects. It is also possible that 8 wk of intense interval training enhanced blood lactate removal during the post-training MAOD test and during the first 3 min of recovery in both male and female subjects. This would account for a decrease in blood \( [\text{La}]_{3\text{min}} \) with no change in \( \text{AO}_2 \) deficit or \( \text{AO}_2 \) uptake in female subjects.

In summary, the present study demonstrated that the increase in MAOD after 4 and 8 wk of intense interval training was not different between men and women. However, an increase in \( \dot{\text{VO}}_2\text{peak} \) and a greater \( \text{AO}_2 \) uptake measured during the post-training timed test in male subjects only, suggests that 8 wk of HIT improves oxidative metabolism in men but not in
women. These findings suggest that there are basic gender differences that may predispose males and females to specific metabolic adaptations following a period of intense interval training. Therefore, the findings of the present investigation are important for the implementation of gender-specific training programs where improvement in both anaerobic and aerobic metabolism is required.
Acknowledgments

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References


Table 1. Physical characteristics of the subjects determined before (pre) and after (post) 8 weeks of training.

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<th>Males (n=7)</th>
<th>Females (n=7)</th>
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<tbody>
<tr>
<td></td>
<td>Pre-training</td>
<td>Post-training</td>
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<tr>
<td>Age (yr)</td>
<td>23.7±1.6\textsuperscript{a}</td>
<td>-</td>
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<tr>
<td>Height (cm)</td>
<td>177.1±1.7\textsuperscript{b}</td>
<td>-</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>80.8±2.3\textsuperscript{b}</td>
<td>82.6±2.8</td>
</tr>
<tr>
<td>AMM (kg)</td>
<td>29.6±1.1\textsuperscript{b}</td>
<td>28.9±1.6</td>
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Values presented are means±SEM. AMM is the estimated active muscle mass for cycling and does not include fat mass or bone mineral content. Males significantly higher than females; \textsuperscript{a}p<0.01, \textsuperscript{b}p<0.001.
Table 2. Peak exercise values obtained during incremental cycling before and after 8 weeks of training.

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<th></th>
<th>Males (n=7)</th>
<th>Females (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-training</td>
<td>Post-Training</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) peak (L·min(^{-1}))</td>
<td>3.58±0.19(^a)</td>
<td>3.85±0.17(^{ab})</td>
</tr>
<tr>
<td>Peak power (W)</td>
<td>379±19(^a)</td>
<td>419±23(^{ab})</td>
</tr>
<tr>
<td>Peak HR (beats·min(^{-1}))</td>
<td>192±3</td>
<td>193±3</td>
</tr>
</tbody>
</table>

Values presented are means±SEM. \( \dot{V}O_2 \) peak = peak oxygen uptake for cycling. HR = heart rate. Males significantly higher than females; \(^a\)\(p<0.001\). Post-training significantly higher than pre-training; \(^b\)\(p<0.01\). Significant difference between males and females in the percent increase from pre- to post-training; \(^c\)\(p<0.05\).
Table 3. Maximal accumulated oxygen deficit (MAOD) and time to exhaustion (TE) measured pre-, mid- and post-training in male and female subjects.

<table>
<thead>
<tr>
<th></th>
<th>Males (n=7)</th>
<th>Females (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MAOD (L)</td>
<td>TE (s)</td>
</tr>
<tr>
<td>Pre-training</td>
<td>3.93±0.22(^a)</td>
<td>175±16</td>
</tr>
<tr>
<td>Mid-training</td>
<td>4.53±0.43(^{ab})</td>
<td>262±35(^c)</td>
</tr>
<tr>
<td>Post-training</td>
<td>4.82±0.46(^{ae})</td>
<td>303±42(^d)</td>
</tr>
<tr>
<td>Increase (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre- to mid-training</td>
<td>14.3±5.2</td>
<td>30.6±3.4</td>
</tr>
<tr>
<td>Mid- to post-training</td>
<td>6.6±1.9</td>
<td>12.4±3.7</td>
</tr>
</tbody>
</table>

Values presented are means±SEM. Males significantly higher than females; \(^a\)p<0.01. Mid-training significantly higher than pre-training; \(^b\)p<0.05, \(^c\)p<0.01. Post-training significantly higher than mid-training; \(^d\)p<0.05, \(^e\)p<0.01.
Table 4. Pre-training MAOD and post-training timed cycling test comparison of accumulated oxygen deficit (AO₂ deficit) and accumulated oxygen uptake (AO₂ uptake).

<table>
<thead>
<tr>
<th></th>
<th>Males (n=7)</th>
<th>Females (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MAOD test</td>
<td>Timed cycling test</td>
</tr>
<tr>
<td></td>
<td>Pre-training</td>
<td></td>
</tr>
<tr>
<td><strong>AO₂ deficit (L)</strong></td>
<td>3.93±0.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.71±0.28&lt;sup&gt;bd&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>AO₂ uptake (L)</strong></td>
<td>8.75±0.99&lt;sup&gt;c&lt;/sup&gt;</td>
<td>8.95±0.95&lt;sup&gt;cd&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Peak HR (beats·min&lt;sup&gt;-1&lt;/sup&gt;)</strong></td>
<td>187±3</td>
<td>182±2&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>[La&lt;sup&gt;-&lt;/sup&gt;]&lt;sub&gt;3 min&lt;/sub&gt; (mmol·L&lt;sup&gt;-1&lt;/sup&gt;)</strong></td>
<td>16.9±0.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>14.2±0.7&lt;sup&gt;bd&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values presented are means±SEM. HR = heart rate. [La<sup>-</sup>]<sub>3 min</sub> is the blood lactate concentration measured 3 min post exercise. Males significantly higher than females; <sup>a</sup>p<0.001, <sup>b</sup>p<0.01, <sup>c</sup>p<0.05. Significant change between pre-training and timed tests; <sup>d</sup>p<0.01, <sup>e</sup>p<0.05. MAOD = maximal accumulated oxygen deficit.