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

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Weber, Clare L., and Donald A. Schneider. Increases in maximal accumulated oxygen deficit after high-intensity interval training are not gender dependent. J Appl Physiol 92: 1795–1801, 2002—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 pretraining peak oxygen uptake (V\textsubscript{\text{\textcircled{O}}2\text{peak}}) to exhaustion (MAOD test) pre-, mid-, and postraining. A postraining timed test was also completed at the MAOD test power output, but this test was stopped at the time to exhaustion achieved during the pretraining MAOD test. The 14.3 ± 5.2% increase in MAOD observed in men after 4 wk of training was not different from the 14.0 ± 3.0% increase seen in women (P > 0.05). MAOD increased by a further 6.6 ± 1.9% in men, and this change was not different from the additional 5.1 ± 2.3% increase observed in women after the final 4 wk of training. V\textsubscript{\text{\textcircled{O}}2\text{peak}} 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 (AO\textsubscript{2}) uptake was higher in men during the postraining timed test compared with the pretraining MAOD test (P < 0.01). In contrast, the AO\textsubscript{2} uptake was unchanged from pre- to postraining 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 V\textsubscript{\text{\textcircled{O}}2\text{peak}} and AO\textsubscript{2} uptake obtained in male subjects after training indicates improved oxidative metabolism in men but not in women. We conclude that there are basic gender differences that may predispose men and women to specific metabolic adaptations after a period of intense interval training.

blood lactate concentration; men and women; supramaximal cycling; anaerobic capacity; active muscle mass

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 men after 6 wk of high-intensity interval training (HIT) but no significant improvement in MAOD for women. Medbø and Burgers speculated that AC might be more "trainable" in men than in women.

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 women, but not in men, after 4 wk of HIT (5). Although all-out sprint exercise tests of <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 after a period of HIT. Furthermore, it is unclear whether changes in oxidative metabolism after intense interval training are gender dependent. Whereas several investigators have demonstrated an increase in oxidative enzyme activity and/or peak oxygen uptake (V\textsubscript{\text{\textcircled{O}}2\text{peak}}) in male subjects after sprint-type training (8, 18, 21, 25), others have reported no change in aerobic energy production after HIT (5, 16). Less is known about changes in oxidative metabolism in female subjects after short-term HIT. Ready et al. (20) demonstrated an 8% increase in maximal oxygen uptake (V\textsubscript{\text{\textcircled{O}}2\text{max}}), whereas Campbell et al. (3) found no change in V\textsubscript{\text{\textcircled{O}}2\text{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

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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 \( \dot{V}_\text{O}_2 \) peak and aerobic energy production during 2–3 min of exhaustive cycling performed at 120% of \( \dot{V}_\text{O}_2 \) peak in men and women. We hypothesized that the changes in MAOD after 4 and 8 wk of HIT are not gender dependent and that oxidative metabolism would increase after intense interval training in both groups.

**METHODS**

**Subjects.** Seven untrained male and seven 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 mo. Additionally, no subject had a past history of participation in a highly competitive sport. Subjects were familiarized with the experimental procedures and provided written informed consent before 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 posttraining 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 with the follicular phase (17).

**Experimental protocol.** The present study involved 8 wk of intense interval training. Subjects performed six submaximal cycling tests 2 wk before the initiation of training to determine their oxygen uptake (\( \dot{V}_\text{O}_2 \))-power relationship. In the week preceding the commencement of training (pretraining), the subjects \( \dot{V}_\text{O}_2 \) peak was measured, and the MAOD for cycling was determined at least 2 days later. MAOD was also determined after 4 wk of training (midtraining). After 8 wk of HIT (posttraining), subjects completed an additional cycling test (posttraining timed test) 48 h after the final training session. The posttraining 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 pretraining MAOD test. Subjects rested for 2 days before the posttraining MAOD test was conducted. After an additional 48 h of rest, the \( \dot{V}_\text{O}_2 \) peak was measured. The active muscle mass (AMM) for cycling was measured pre- and posttraining.

**Determination of \( \dot{V}_\text{O}_2 \) peak.** The \( \dot{V}_\text{O}_2 \) peak for cycling was measured by using a continuous ramp protocol conducted on an electronically braked cycle ergometer (Excalibur Sport V2.0, Groningen, Lode, The Netherlands). Pedal rate was maintained at 70 rpm, and the power output was increased by 20 W/min for women and by 25 W/min for men until exhaustion. Heart rate (HR) was monitored continuously during exercise by using an electrocardiograph (model M 607, Lohmeier, Munich, Germany), and \( \dot{V}_\text{O}_2 \) was measured breath by breath by using a metabolic measurement system (MedGraphics Cardiorespiratory Diagnostic Systems, St. Paul, MN) and averaged over 30-s intervals. The two highest 30-s values for \( \dot{V}_\text{O}_2 \) were averaged and reported as the \( \dot{V}_\text{O}_2 \) peak for cycling.

**Submaximal exercise bouts.** Steady-state \( \dot{V}_\text{O}_2 \) was measured at six submaximal power outputs between 20 and 75% of \( \dot{V}_\text{O}_2 \) peak before training. Subjects cycled at 70 rpm for 10 min and the \( \dot{V}_\text{O}_2 \) values measured at minutes 9 and 10 were averaged and reported as the steady-state \( \dot{V}_\text{O}_2 \) for the corresponding power output. Data collected from the six submaximal bouts were used to establish the \( \dot{V}_\text{O}_2 \)-power relationship for cycling. The linear regression of the \( \dot{V}_\text{O}_2 \)-power relationship was used to calculate the power output that corresponded to 120% of \( \dot{V}_\text{O}_2 \) peak. This power output was then used in all subsequent MAOD tests (pre-, mid-, and posttraining) and in the timed cycling test conducted after training.

**MAOD test and posttraining timed cycling test.** Subjects warmed up by cycling on a Lode cycle ergometer for 5 min at 50 W for men and at 35 W for women. Subjects were then asked to rest quietly on the cycle ergometer for 5 min. Immediately before the MAOD test, the subject’s hyperventilating earlobe was sterilized with 70% ethanol and punctured with a 1.5-mm lancet (Microlancet, Becton Dickson, Sandy, UT). The first drop of blood was wiped away, and 50 µl of free-flowing blood were collected in a capillary tube and immediately dispensed into a prechilled Eppendorf tube for subsequent analysis of blood lactate concentration (\([\text{Lac}^-] \)) [model 2700 SELECT, Yellow Spring Instruments, Yellow Springs, OH]. After 2 min of unloaded cycling at 70 rpm, the predetermined power output of 120% \( \dot{V}_\text{O}_2 \) peak was applied immediately. HR was monitored continuously while \( \dot{V}_\text{O}_2 \) and minute ventilation (Ve) were measured breath by breath throughout the exercise bout. Subjects were required to maintain pedal cadence at 70 rpm throughout the MAOD tests, and the test was terminated when the subject could no longer maintain a pedal cadence of 60 rpm despite verbal encouragement. Blood samples were obtained for subsequent lactate analysis 3 min after (\([\text{Lac}^-]_{3 \text{min}} \)) the MAOD test while the subject cycled at 50 W for men and at 35 W for women.

The accumulated oxygen (AO2) deficit was calculated as the difference between the AO2 demand and the AO2 uptake measured during the MAOD test (12). The AO2 deficit calculated for the MAOD test was reported as the “maximal AO2 deficit” (MAOD) for cycling, whereas the AO2 deficit measured during the posttraining timed test was not “maximal” because subjects did not cycle to exhaustion. Absolute MAOD values were decreased by 9% to correct for reductions in the oxygen stores of the body (12). Weber and Schneider (29) have demonstrated that this method of determining MAOD for cycling is highly repeatable in untrained male and female subjects (intraclass 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 midtraining, mid- to posttraining, and pre- to posttraining.

**Training protocol.** Training was performed on a basket-loading cycle ergometer (model 824E, Monark Ergomedic, Varberg, Sweden) so the load could be applied immediately. Subjects trained 3 days/wk for a total of 8 wk. The training sessions consisted of three, 2-min constant-load cycling intervals performed at 70 rpm. 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 work rate every week. Each subject was training at an intensity equal to 100% of the power output used in the MAOD test by week 8 of training. Peak HR was measured.
Table 1. Physical characteristics of the subjects determined before and after 8 wk of training

<table>
<thead>
<tr>
<th></th>
<th>Men (n = 7)</th>
<th>Women (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>22.7 ± 1.6*</td>
<td>22.7 ± 2.6</td>
</tr>
<tr>
<td>Height, cm</td>
<td>177.1 ± 1.7†</td>
<td>168.7 ± 1.8</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>80.8 ± 2.3†</td>
<td>64.3 ± 1.6</td>
</tr>
<tr>
<td>AMM, kg</td>
<td>29.6 ± 1.1†</td>
<td>23.6 ± 0.4</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. Pretraining, 1 wk before training; posttraining, after 8 wk of training; AMM, estimated active muscle mass for cycling not including fat mass or bone mineral content. Men significantly higher than women: *P < 0.01; †P < 0.001.

Table 2. Peak exercise values obtained during incremental cycling before and after 8 wk of training

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>Increase</th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ peak, l/min</td>
<td>3.55 ± 0.19†</td>
<td>3.85 ± 0.17†</td>
<td>7.9 ± 2.0%‡</td>
<td>2.55 ± 0.11</td>
<td>2.62 ± 0.08</td>
<td>2.9 ± 1.5%</td>
</tr>
<tr>
<td>Peak power, W</td>
<td>379 ± 19*</td>
<td>419 ± 23†</td>
<td>10.7 ± 2.0%</td>
<td>269 ± 13</td>
<td>298 ± 15†</td>
<td>11.2 ± 1.1%</td>
</tr>
<tr>
<td>Peak HR, beats/min</td>
<td>192 ± 3</td>
<td>193 ± 3</td>
<td>0.9 ± 1.4%</td>
<td>193 ± 2</td>
<td>196 ± 3</td>
<td>2.0 ± 1.1%</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. VO₂ peak, peak oxygen uptake for cycling; HR, heart rate. *Men significantly higher than women, P < 0.001. †Posttraining significantly higher than pretraining, P < 0.01. ‡Significant difference between men and women in the percent increase from pre- to postraining, P < 0.05.

RESULTS

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

Peak cardiorespiratory values obtained during incremental cycling. The peak exercise values obtained during incremental cycling pre- and posttraining are presented in Table 2. Male subjects obtained a significantly higher absolute VO₂ peak (l/min) than did female subjects before training. The gender difference remained significant when VO₂ peak values were expressed relative to body mass (men 44.4 ± 2.4 vs. women 39.6 ± 0.9 ml·kg⁻¹·min⁻¹; P < 0.05). VO₂ peak increased significantly in men after 8 wk of training, whereas the change in VO₂ peak with training was not significant in women. 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 postraining in male and female subjects. Men obtained a greater absolute MAOD for cycling than did women pre-, mid-, and postraining. In addition, when MAOD was expressed relative to the AMM for cycling, men maintained a higher MAOD than did women pretraining (men 132.2 ± 4.5 vs. women 116.5 ± 6.6 ml/kg AMM; P < 0.05) and postraining (men 166.0 ± 10.6 vs. women 142.7 ± 7.1 ml/kg AMM; P < 0.05). Both male and female subjects demonstrated a significant increase in MAOD after only 4 wk of training (pre- to midtraining). After the final 4 wk of training (mid- to postraining), 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 men was not different from the 19.6 ± 3.1% increase obtained for women 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 pretraining MAOD test were not different between men (187 ± 3 beats/min) and women (188 ± 3 beats/min), and there was no change in peak HR for either group after 8 wk.
of training. Pretraining, blood \([Lac^-]_{3\text{ min}}\) for men was significantly higher than for women \((P < 0.001)\). After 8 wk of training, blood \([Lac^-]_{3\text{ min}}\) increased to 19.9 ± 0.9 mmol/l in men \((P < 0.01)\) and to 16.0 ± 0.6 mmol/l in women \((P < 0.01)\), but \([Lac^-]_{3\text{ min}}\) remained significantly higher in male than in female subjects. However, the percent increase in blood \([Lac^-]_{3\text{ min}}\) from pre- to posttraining was not different between the two groups.

Blood \([Lac^-]\) and HR responses to a training session. Blood \([Lac^-]_{3\text{ min}}\), measured after the third cycling repetition of the three training sessions was significantly higher in men than in women pretraining \((14.3 ± 0.5\) vs. \(11.5 ± 0.6\) mmol/l; \(P < 0.01\)), midtraining \((14.6 ± 0.6\) vs. \(12.5 ± 0.4\) mmol/l; \(P < 0.01\)), and posttraining \((15.8 ± 0.4\) vs. \(12.7 ± 0.3\) mmol/l; \(P < 0.001)\). However, the significant increase of 9.6 ± 3.0% \((P < 0.05)\) in blood \([Lac^-]_{3\text{ min}}\) reported for men was not different from the significant increase of 9.6 ± 4.6% \((P < 0.05)\) measured in women after 8 wk of training. The peak HR recorded during the third cycling interval of a training session in week 1 was not different between male \((183 ± 4\) beats/min) and female \((185 ± 4\) beats/min) 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) or female \((185 ± 3\) beats/min) subjects.

Pretraining MAOD test and posttraining timed cycling test. The mean \(AO_2\) deficit determined during the posttraining timed cycling test (see Table 4) was significantly lower than the \(AO_2\) deficit achieved during the pretraining MAOD test in male subjects. In contrast, the \(AO_2\) deficit was not different between the two tests in female subjects. The \(AO_2\) uptake measured during the timed cycling test after training was significantly higher compared with the \(AO_2\) uptake obtained during the pretraining MAOD test in male subjects. There was no change in \(AO_2\) uptake in female subjects as a result of training. During the posttraining timed cycling test, mean \(\dot{V}_{O_2}\) was significantly \((P < 0.01)\) lower compared with the pretraining MAOD test in both male \((\text{posttraining timed} 87.6 ± 4.0\) vs. pretraining 109.7 ± 6.2 l/min) and female \((\text{posttraining timed} 63.9 ± 4.1\) vs. pretraining 75.6 ± 3.9 l/min) subjects. The peak HR obtained for both male and female subjects was significantly lower during the timed cycling test after training compared with the pretraining MAOD test. Blood \([Lac^-]_{3\text{ min}}\) was observed to be 21.1 ± 7.2% lower in men and 15.8 ± 4.1% lower in women during the posttraining timed cycling test compared with the pretraining MAOD test. The relative decrease in blood \([Lac^-]_{3\text{ min}}\) during the posttraining 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 men and the 19.6 ± 3.1% for women 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.

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### Table 3. Maximal accumulated oxygen deficit and time to exhaustion measured pre-, mid-, and posttraining in male and female subjects

<table>
<thead>
<tr>
<th></th>
<th>Men (n = 7)</th>
<th>Women (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MAOD, liters</td>
<td>TE, s</td>
</tr>
<tr>
<td>Pretraining</td>
<td>3.93 ± 0.22a</td>
<td>175 ± 16</td>
</tr>
<tr>
<td>Midtraining</td>
<td>4.53 ± 0.43ab</td>
<td>262 ± 35c</td>
</tr>
<tr>
<td>Posttraining</td>
<td>4.82 ± 0.46ab</td>
<td>303 ± 42d</td>
</tr>
<tr>
<td>Increase pre- to midtraining</td>
<td>14.3 ± 5.2%</td>
<td>30.6 ± 3.4%</td>
</tr>
<tr>
<td>Increase mid- to posttraining</td>
<td>6.6 ± 1.9%</td>
<td>12.4 ± 3.7%</td>
</tr>
</tbody>
</table>

Values are means ± SE; \(n\), no. of subjects. MAOD, maximal accumulated oxygen deficit; TE, time to exhaustion; midtraining, after 4 wk of training. *Men significantly higher than women; \(P < 0.01\). Midtraining significantly higher than pretraining; \({}^bP < 0.05; {}^cP < 0.01\). Posttraining significantly higher than midtraining; \({}^dP < 0.05; {}^eP < 0.01\).

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### Table 4. Pretraining MAOD and posttraining timed cycling test comparison of accumulated oxygen deficit and uptake

<table>
<thead>
<tr>
<th></th>
<th>Men (n = 7)</th>
<th>Women (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MAOD test pretraining</td>
<td>Timed cycling test</td>
</tr>
<tr>
<td>(AO_2) deficit, liters</td>
<td>3.93 ± 0.22b</td>
<td>3.71 ± 0.28bd</td>
</tr>
<tr>
<td>(AO_2) uptake, liters</td>
<td>8.75 ± 0.99c</td>
<td>8.95 ± 0.95cd</td>
</tr>
<tr>
<td>Peak HR, beats/min</td>
<td>187 ± 3</td>
<td>182 ± 2e</td>
</tr>
<tr>
<td>([Lac^-]_{3\text{ min}}) mmol/l</td>
<td>16.9 ± 0.4a</td>
<td>14.2 ± 0.7bd</td>
</tr>
</tbody>
</table>

Values presented are means ± SE; \(n\), no. of subjects. \(AO_2\), accumulated oxygen. \([Lac^-]_{3\text{ min}}\), blood lactate concentration measured 3 min postexercise. Men significantly higher than women: \(^aP < 0.001; {}^bP < 0.01; {}^cP < 0.05\). Significant change between pretraining and timed tests: \(^dP < 0.01; {}^eP < 0.05\).
Before training, men demonstrated a significantly higher MAOD than did women 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) values in men suggest differences between untrained men and women in morphological and/or biochemical skeletal muscle characteristics. Whereas Esbjörnsson et al. (5) 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 been previously demonstrated to be higher in male than in female subjects (2, 14, 22). These factors may contribute to a greater ability to produce ATP anaerobically during sprint-type exercise in men than in women. Despite such gender differences in muscle enzyme activity and fiber type, the increases observed in MAOD and blood [Lac\(^{-}\)]\(_{\text{3 min}}\) 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 [Lac\(^{-}\)] 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 supports 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 ~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 after training. The possible mechanisms that contribute to the increase in MAOD after 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 (5). Nevertheless, PFK activity has been reported to be greater in trained compared with untrained men (23). In addition, Esbjörnsson-Liljedahl et al. (5) reported that total LDH activity increased by the same relative amount in male and female subjects after 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. Although 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 after intense training may be explained in part by an increase in muscle strength. Although the anthropometric measurements obtained from DXA 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 after 6 wk of HIT, whereas men achieved a 16% increase. Several limitations in the investigation by Medbø and Burgers could account for these conflicting observations. Medbø and Burgers divided five men and seven women into two different training groups. They did not state whether subjects completed the same volume of training or whether the response to the different training protocols was gender dependent. In addition, male and female subjects achieved similar peak [Lac\(^{-}\)] after the pretraining 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 [Lac\(^{-}\)] than men after high-intensity activity when equally trained (5, 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 made no attempt to control for menstrual cycle phase because 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 compared with values recorded during the luteal phase. Although the magnitude of the effect of menstrual cycle status on intense exercise remains unclear, Tarnopolsky (27) has suggested that researchers 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 posttraining in female subjects. Ready et al. (20) demonstrated a 20% increase in maximal “oxygen debt” as well as a concomitant increase in peak [Lac\(^{-}\)] after 6 wk of intense cycle training in women. 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, 5). These investigations report significant improvements in the ability of female subjects to produce ATP anaerobically with training. How-
ever, the interpretation of these results should be questioned because 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 (13) suggested that ∼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 \( \dot{V}O_2 \) peak in male subjects after 8 wk of training. However, the present study failed to demonstrate a significant improvement in \( \dot{V}O_2 \) 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 \( \dot{V}O_2 \) max after 6 wk of sprint cycle training in female subjects.

The increase in \( \dot{V}O_2 \) 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 \( \dot{V}O_2 \) peak reported in men after HIT have been related to increases in oxidative enzyme activity (8, 18, 21) and muscle blood flow (9). However, Eshjörnsson et al. (5) reported no change in the activity of oxidative enzymes after 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 with men, as indicated by the lower accumulation of inosine monophosphate and inosine after high-intensity exercise (4). This would allow each successive training repetition to be performed more anaerobically in women compared with men. Thus female subjects in the present study may have placed less stress on the aerobic energy system during this type of training than did the male subjects. It is clear that further research is required to examine the mechanisms that control gender-specific changes in \( \dot{V}O_2 \) peak with intense interval training.

Nevertheless, the improvement of \( \dot{V}O_2 \) peak in male subjects may be associated with the greater \( \dot{AO}_2 \) uptake observed in men during the timed cycling test compared with the pretraining MAOD test. Harmer et al. (7) measured the \( \dot{AO}_2 \) 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 \( \dot{AO}_2 \) uptake in male subjects with training. However, Harmer et al. 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 \( \dot{AO}_2 \) uptake observed in male subjects in the present study.

It has been suggested that a higher \( \dot{V}O_2 \) 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 \( \dot{AO}_2 \) uptake seen in male subjects during the posttraining timed test, compared with the pretraining MAOD test, could be accounted for by an increase in \( \dot{V}O_2 \) because mean \( \dot{V}O_2 \) was actually decreased in the timed test. Further evidence that an increase in \( \dot{AO}_2 \) uptake in men was not secondary to changes in cardiorespiratory function relates to the fact that the relative decrease in mean \( \dot{V}O_2 \) and peak HR during the timed test was not different between men and women. Nonetheless, no change in \( \dot{AO}_2 \) uptake was found in female subjects with training. These findings suggest that adaptations in \( \dot{V}O_2 \) and HR did not contribute to the gender-specific increase in \( \dot{AO}_2 \) uptake observed in men during the posttraining timed test. Alternatively, the increase in \( \dot{AO}_2 \) 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 after 7 wk of HIT in men. Thus the unchanged \( \dot{AO}_2 \) 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.

Women demonstrated a similar decrease in blood \( [\text{Lac}]_{13 \text{min}} \) compared with men during the posttraining 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 \( \dot{V}O_2 \) without any change in whole body \( \dot{V}O_2 \) in female subjects. It is also possible that 8 wk of intense interval training enhanced blood lactate removal during the posttraining MAOD test and during the first 3 min of recovery in both male and female subjects. This would account for a decrease in blood \( [\text{Lac}]_{13 \text{min}} \) with no change in \( \dot{AO}_2 \) deficit or \( \dot{AO}_2 \) 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 \( \dot{V}O_2 \) peak and a greater \( \dot{AO}_2 \) uptake measured during the posttraining 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 men and women to specific metabolic adaptations after 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.

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