Dietary carbohydrate, muscle glycogen content, and endurance performance in well-trained women

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Dietary carbohydrate, muscle glycogen content, and endurance performance in well-trained women. J Appl Physiol 88: 2151–2158, 2000.—This study examined the ability of well-trained eumenorrheic women to increase muscle glycogen content and endurance performance in response to a high-carbohydrate diet (HCD; ~78% carbohydrate) compared with a moderate-carbohydrate diet (MD; ~48% carbohydrate). Preexercise muscle glycogen content was higher after the MD (62.5 ± 50.1 mmol/kg dry muscle) and 13% greater after the HCD (70.9 ± 44.8 mmol/kg dry muscle). Postexercise muscle glycogen was low after both trials (MD, 91.4 ± 34.5; HCD, 80.3 ± 19.5 mmol/kg dry muscle), and net glycogen utilization during exercise was greater after the HCD. The subjects also cycled longer at ~80% VO2max after the HCD vs. MD (115.31 ± 10.47 vs. 106.35 ± 8.36 min, respectively). In conclusion, aerobically trained women increased muscle glycogen content in response to a high dietary carbohydrate intake during the luteal phase of the menstrual cycle, but the magnitude was smaller than previously observed in men. The increase in muscle glycogen, and possibly liver glycogen, after the HCD was associated with increased cycling performance to volitional exhaustion at ~80% VO2max.

PREVIOUS STUDIES HAVE DEMONSTRATED that a combination of intense aerobic exercise, increased dietary carbohydrate (CHO) intake, and exercise tapering can lead to an increased resting muscle glycogen content or “supercompensation” (3, 7, 8, 13, 21, 34) and ultimately enhance exercise endurance performance (3, 8, 21, 23), as reviewed by Hawley et al. (17). These studies examined primarily male subjects, and the assumption has been that the results also apply to women, although there are few data examining these relationships in aerobically trained women.

Recent, well-controlled, gender-comparison studies have matched male and female endurance athletes for many of the following parameters: maximal oxygen uptake [VO2max; expressed per kg lean body mass (LBM)]; preexercise dietary intake; exercise training volume, intensity, and history; and controlled-for menstrual status (19, 30, 38, 40, 41). Women maintained lower respiratory exchange ratios (RER), suggesting an increased reliance on fat oxidation, compared with men during cycling and running at power outputs between 40 and 75% VO2max (19, 30, 38, 40, 41). These studies and recent reviews have discussed the potential reasons for the greater reliance on fat oxidation in women (11, 33, 39). In one study, muscle glycogen stores were unchanged in women in response to a 4-day high-CHO diet (HCD; ~75% CHO) compared with a moderate-CHO diet (MD; ~57% CHO), whereas the men increased muscle glycogen content by ~40% after the high-CHO regimen (40). Furthermore, only men increased cycling time to exhaustion (80–85% VO2max, after 1 h at 70–75% VO2max) in response to the dietary CHO-loading regimen. Therefore, women may not be able to supercompensate muscle glycogen stores before exercise and increase exercise performance in response to increases in dietary CHO.

The female athletes employed in the above studies were tested during the follicular phase (FP) of the menstrual cycle when circulating reproductive hormones are low (19, 38, 40, 41). Hackney et al. (15) observed 13% higher resting muscle glycogen contents in the luteal phase (LP) compared with the FP of the menstrual cycle when they controlled for diet and exercise in the 36 h before muscle glycogen sampling. In contrast, Nicklas et al. (24) studied moderately trained women and reported no significant difference in muscle glycogen content between phases of the menstrual cycle after a glycogen-depleting exercise bout and 3 days of a controlled diet (~56% CHO) with no exercise. However, a significantly greater amount of muscle glycogen repletion occurred during the 3 days in the LP vs. the FP (379 ± 20 vs. 313 ± 25 mmol/kg dry muscle (dm)). These studies suggest that glycogen synthesis may be increased during the LP of the menstrual cycle. Animal studies support this sugges-
tion, because elevations in the reproductive hormones, estradiol (E2) and progesterone (P), in the LP increase liver and skeletal muscle glycogen synthesis and the duration of exhaustive exercise in rats (10, 22, 37). Therefore, the testing of women during the LP of the menstrual cycle may demonstrate that female athletes are able to supercompensate muscle glycogen stores and improve endurance performance.

The purpose of this study was to examine the influence of increased dietary CHO intake, employed during an exercise-tapering program, on resting muscle glycogen content and performance during cycle exercise to volitional exhaustion at ~80% VO2max in aerobically trained women during the LP of the menstrual cycle.

METHODS

Subjects. Six well-trained female athletes gave written informed consent and volunteered to participate in the study after approval by the University of Guelph Human Ethics Committee. All subjects cycled regularly because three were triathletes, two were cyclists, and one was an endurance runner who trained on a cycle twice a week. All subjects were eumenorrheic with regular cycles of 24–29 days. Characteristics of the subjects are presented in Table 1.

Preexperimental protocols. The subjects performed a progressive VO2max test on a cycle ergometer during their initial visit to the laboratory. On a separate day, all subjects performed a practice cycle to voluntary exhaustion at a constant power output of ~80% VO2max. Exhaustion was defined as the point when the subject could no longer maintain a cadence 20 rpm below their predetermined cadence (80–100 rpm), despite verbal encouragement. If a subject was unable to cycle for 75 min, a second practice ride to exhaustion was performed at a later date at a slightly reduced power output.

On a morning distinct from the exercise tests, subjects arrived at the laboratory in a fasted, nonexercised state for the determination of body composition by using underwater hydrostatic weighing. The subject wore a light bathing suit during dry and underwater weighing. Underwater weight was determined after the subject expired as much air as possible. Body density was determined by using water temperature, dry and wet weights, and a correction value of 1.25 possible. Body density was determined by using water hydrostatic weighing. The subject wore a light bathing suit for air trapped in the lungs. Percent body fat and LBM were calculated as previously described (36).

Subjects were given an oral digital thermometer and were instructed how to accurately record basal body temperature every morning before getting out of bed for at least one entire cycle before the experimental testing. An increase of 0.3°C above the mean temperature was used to indicate ovulation.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Subjects</th>
<th>MD</th>
<th>HCD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass, kg</td>
<td>57.0 ± 1.1</td>
<td>57.0 ± 1.0</td>
<td>57.0 ± 1.1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>162.6 ± 1.5</td>
<td>162.6 ± 1.5</td>
<td>162.6 ± 1.5</td>
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<tr>
<td>LBM, kg</td>
<td>45.9 ± 1.2</td>
<td>45.9 ± 1.2</td>
<td>45.9 ± 1.2</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>19.2 ± 1.7</td>
<td>19.2 ± 1.7</td>
<td>19.2 ± 1.7</td>
</tr>
<tr>
<td>Age, yr</td>
<td>27.4 ± 1.7</td>
<td>27.4 ± 1.7</td>
<td>27.4 ± 1.7</td>
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<tr>
<td>VO2max, ml·kg·BM·1·min−1</td>
<td>56.4 ± 1.5</td>
<td>56.4 ± 1.5</td>
<td>56.4 ± 1.5</td>
</tr>
<tr>
<td>VO2max, ml·kg·LBM·1·min−1</td>
<td>69.6 ± 2.2</td>
<td>69.6 ± 2.2</td>
<td>69.6 ± 2.2</td>
</tr>
<tr>
<td>Training frequency, times/wk</td>
<td>6.0 ± 0.4</td>
<td>6.0 ± 0.4</td>
<td>6.0 ± 0.4</td>
</tr>
<tr>
<td>Training duration, h/wk</td>
<td>7.0 ± 1.0</td>
<td>7.0 ± 1.0</td>
<td>7.0 ± 1.0</td>
</tr>
</tbody>
</table>

Values are means ± SE for 6 subjects. BM, body mass; LBM, lean body mass; VO2max, maximal oxygen uptake.

Confirmation of the LP of the menstrual cycle was later verified with blood P measurements of >9.5 nM during the diet and/or exercise test (9, 31). The subject began one of the two individually designed diets 2–4 days after ovulation. The second trial occurred during one of the two subsequent menstrual cycles. The 7-day diet and testing protocols were completed 1 or more days before menses.

Diet preparation. The subjects were given detailed instructions to accurately record food intake, and they recorded daily food records for 4–7 days, including 1–2 weekend days. In addition, a list of specific food likes and dislikes were compiled. All food records were analyzed by using the NUTRIPRO diet-analysis software (West Publishing, ESHA Research, 1991) with manual adjustments when specific foods were not available in the nutrition program. Total daily caloric intake; total grams of CHO, protein, and fat consumed per day; and percent contributions of CHO, protein, and fat to daily total caloric intake were determined. Two 7-day diets, one consisting of MD (~48% total daily energy from CHO) and one of 3 days of MD followed by 4 days of HCD (~78% CHO) were designed and randomly assigned to the subjects.

Diet and exercise-tapering regimen. On the day before day 1 of each of the two diet and exercise-tapering regimens, all food and drink to be consumed over the next 7 days was purchased and delivered to the subject. The subject followed the designed diet and recorded the actual amount of food eaten. Frequent communication occurred between the subject and researcher during the 7-day regimen to ensure accurate recording of the diet. After the initial 7-day regimen, the actual dietary consumption was analyzed, and these results were used to adjust the diet of the second 7-day regimen. The characteristics of all diets (habitual, MD, and HCD) are presented in Table 2.

The subjects also followed an exercise-tapering program during the 7-day regimen. The taper consisted of 90 min of exercise on day 1 , ~45 min of exercise on days 2 and 3, 20–30 min of exercise on days 4 and 5, and rest on day 6 (Fig. 1). Subjects were instructed to maintain their normal exercise intensity during the taper, because this has been shown to maximize subsequent exercise performance (35). The mode of exercise during the taper depended on the individual subject. Cyclists performed only cycling on all 5 exercise days, whereas the runner and triathletes ran and cycled on various days. The tapering program used during the second regimen was identical in exercise mode and duration to that used during the first.

Experimental protocol. Subjects arrived at the laboratory at the same time of the day for both trials, having eaten the appropriate meal within the previous 2–4 h to simulate
CARBOHYDRATE LOADING IN ENDURANCE-TRAINED WOMEN

PRECOMPETITION PRACTICES

Table 3. Pulmonary and heart rate analyses

<table>
<thead>
<tr>
<th>Variable</th>
<th>Diet</th>
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<th>30</th>
<th>45</th>
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<tr>
<td>V̇O₂max,%</td>
<td>MD</td>
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<td>81</td>
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<td>80</td>
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<tr>
<td></td>
<td>HCD</td>
<td>75</td>
<td>81</td>
<td>83</td>
<td>82</td>
<td>81</td>
<td>80</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>MD</td>
<td>162</td>
<td>166</td>
<td>167</td>
<td>168</td>
<td>169</td>
<td>169</td>
</tr>
<tr>
<td></td>
<td>HCD</td>
<td>162</td>
<td>166</td>
<td>170</td>
<td>168</td>
<td>169</td>
<td>168</td>
</tr>
<tr>
<td>RER</td>
<td>MD</td>
<td>0.99±0.03</td>
<td>0.93±0.01*</td>
<td>0.89±0.02*</td>
<td>0.90±0.02*</td>
<td>0.88±0.01*</td>
<td>0.86±0.02*</td>
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<tr>
<td></td>
<td>HCD†</td>
<td>1.01±0.03</td>
<td>0.97±0.02*</td>
<td>0.94±0.01*</td>
<td>0.93±0.01*</td>
<td>0.92±0.02*</td>
<td>0.88±0.02*</td>
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</tbody>
</table>

Values are means ± SE for 6 subjects. RER, respiratory exchange ratio. *Significantly different from 5-min values, P < 0.05. †Significant main effect for trial, P < 0.05.

RESULTS

Performance variables. The women cycled longer at ~80–82% V̇O₂max after the HCD (115:31 ± 10:47, min:s) compared with the MD (106:35 ± 8:36, min:s). There were no significant differences in V̇O₂ or heart rate throughout the exercise between trials (Table 3). However, RER was significantly higher during the initial 75 min of the HCD vs. MD trial (significant main effect for trial).

Muscle glycogen measurements. Preexercise glycogen content increased in five of the six athletes after the HCD and tapering regimen, resulting in a significant increase of 84 mmol glucosyl units/kg dm (13%) over the MD regimen (Table 4, Fig. 2). Postexercise muscle glycogen contents were below 100 mmol/kg dm in both trials and were not significantly different (Fig. 2). Net glycogen utilization during the HCD trial was
also significantly greater than that during the MD trial.

Blood measurements. Plasma insulin concentrations were constant during the initial 60 min of exercise and decreased at exhaustion in both conditions (Fig. 3). Whole blood glucose increased during exercise and returned to rest levels at exhaustion in both conditions (Fig. 4). There were no differences between dietary conditions in insulin or glucose. Whole blood lactate was significantly elevated in response to exercise and was significantly higher during the HCD compared with the MD trial (Fig. 5). Plasma FFA remained at low resting concentrations in both trials throughout exercise (Table 5). Whole blood glycerol concentrations increased significantly above rest in both trials but were unaffected by diet (Table 5).

Reproductive hormones. Resting plasma P and E_2 were high on days 1 and 7 of the diet and tapering regimen in both trials and indicative of the LP of the menstrual cycle (Table 6). During exercise on day 7, plasma E_2 and P were generally unchanged over time, although plasma E_2 increased above rest at 40 and 60 min in HCD (Table 5).

Estimated CHO and fat oxidation. The subjects oxidized significantly more CHO during the initial 75 min of the HCD (2.79 ± 0.22 g/min) compared with the MD trial (2.35 ± 0.19 g/min). The total estimated CHO oxidation in the initial 75 min of cycling was 209 ± 16 g in HCD and 176 ± 14 g in MD. Fat oxidation was significantly less during the HCD (0.25 ± 0.05 g/min) compared with the MD trial (0.42 ± 0.04 g/min), resulting in estimated total fat utilization of 19 ± 4 g in HCD and 31 ± 3 g in MD during the first 75 min of exercise.

Total CHO oxidation during the entire trial was also significantly greater after the HCD (317 ± 28 g) compared with the MD (252 ± 33 g). Total fat oxidation was significantly lower during the HCD (34 ± 8 g) compared with the MD trial (49 ± 7 g). CHO accounted for 81% of the oxidized substrate in the HCD trial and 70% during the MD trial.

**DISCUSSION**

A 7-day diet and exercise-tapering regimen, containing 48% of the total caloric intake as CHO, resulted in high skeletal muscle glycogen contents in well-trained eumenorrheic women during the LP of the menstrual cycle. The subjects oxidized significantly more CHO during the initial 75 min of the HCD compared with the MD trial. Whole blood glucose and lactate increased during exercise, with lactate being significantly higher in the HCD trial. Plasma insulin decreased during exercise, with no differences between dietary conditions. Whole blood glucose returned to rest levels at exhaustion in both conditions. Plasma FFA remained low throughout exercise, with no differences between dietary conditions. Whole blood glycerol increased above rest in both trials but was unaffected by diet. Plasma P and E_2 were high on days 1 and 7 of the diet and tapering regimen in both trials and indicative of the LP of the menstrual cycle. During exercise on day 7, plasma E_2 and P were generally unchanged over time, although plasma E_2 increased above rest at 40 and 60 min in HCD. Estimated CHO and fat oxidation were significantly higher during the HCD trial. Total CHO oxidation during the entire trial was also significantly greater after the HCD trial compared with the MD trial. Total fat oxidation was significantly lower during the HCD trial compared with the MD trial. CHO accounted for 81% of the oxidized substrate in the HCD trial and 70% during the MD trial.
cycle. Glycogen content increased by 13% (84 mmol/kg dm) when the diet was altered to contain 78% CHO in the final 3–4 days of the regimen. The increase in muscle glycogen, and possibly liver glycogen, was associated with increased CHO oxidation and cycle time to volitional exhaustion at ~80% \( VO_2\text{max} \). Therefore, the women were able to supercompensate glycogen but not to the same magnitude as generally reported in men.

Dietary CHO and resting muscle glycogen content. The present findings of a small but significant increase in glycogen content with an increased dietary intake of CHO differ from an earlier study reporting an inability of trained women to supercompensate glycogen (40). There are several potential explanations to account for the differing effectiveness of the HCD regimen in the two studies. The most obvious is the testing of subjects in the LP of the menstrual cycle in the present study, as opposed to the FP in the previous study (41). Others include the higher muscle glycogen values obtained after the MD in the present study and the higher amounts of CHO consumed during the HCD in the present study.

During the LP, the reproductive hormones \( E_2 \) and \( P \) are elevated, in contrast to during the FP when the hormones are suppressed. Rodent data suggest that the reproductive hormones may augment glycogen synthesis and resting muscle glycogen contents (10, 22, 37). In humans, only two studies have measured resting glycogen concentration in both menstrual phases in the same individuals, and neither study employed well-trained endurance athletes. Hackney (15) measured a higher resting muscle glycogen content in the LP compared with the FP (439 ± 21 vs. 390 ± 15 mmol/kg dm) while controlling for diet and exercise in the 36 h before muscle sampling. Nicklas et al. (24) measured muscle glycogen content after a bout of depleting exercise and again after 3 days of rest and a controlled diet (~56% CHO) in both menstrual phases in moderately trained women (\( VO_2\text{max} \), 44.9 ml·kg\(^{-1} \)·min\(^{-1} \)). Glycogen repletion was significantly greater during the 3 day in the LP vs. the FP (379 ± 20 vs. 313 ± 25 mmol/kg dm), although the resulting muscle glycogen was not statistically higher (LP, 446 ± 24 vs. FP, 400 ± 25 mmol/kg dm). Therefore, whereas the LP may be associated with marginally higher rates of glycogen synthesis and higher glycogen contents, no study has examined the ability to supercompensate muscle glycogen in both phases of the menstrual cycle in the same women.

The subjects in the present study were well trained and included cycling in their normal training programs. After the MD, they achieved resting vastus lateralis muscle glycogen contents that were ~50% higher than those reported by Tarnopolsky et al. (40). Although the training status and \( VO_2\text{max} \) of the two groups of subjects were similar, it may be that including cycling as a regular component of the training programs in the present study contributed to the difference. Cycling places localized stress on the vastus lateralis muscle and may have augmented the ability to increase glycogen during the HCD and tapering regimen. In support of this, Greiwe et al. (14) reported muscle glycogen contents of ~800 mmol/kg dm in a group of four women and two men who trained for 10 wk on a cycle ergometer.

Other explanations relate to the larger difference in the percent CHO intake between the MD and HCD in the present study or, more importantly, the attainment of higher absolute and relative amounts of ingested CHO in the HCD. In the present study, the percent CHO intake was 48 and 78% in the MD and HCD, respectively, as opposed to 57 and 75% in the previous study (40). The absolute and relative CHO consumption in the HCD was 464 g and 10.1 g/kg LBM in the present study and was 370 g and 7.9 g/kg LBM in a previous study, respectively (40). Earlier studies with men reported that a CHO intake >500 g/day and 8.5 g/kg LBM was consumed to increase glycogen by 300 to

<table>
<thead>
<tr>
<th>Variable</th>
<th>Diet</th>
<th>Rest</th>
<th>20</th>
<th>40</th>
<th>60</th>
<th>Exhaustion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood glycerol</strong></td>
<td>MD</td>
<td>35.3 ± 10.3</td>
<td>100.2 ± 24.1*</td>
<td>27.5 ± 31.5*</td>
<td>56.5 ± 39.2*</td>
<td>93.8 ± 53.6*</td>
</tr>
<tr>
<td></td>
<td>HCD</td>
<td>31.6 ± 9.2</td>
<td>86.0 ± 13.9*</td>
<td>39.4 ± 29.1*</td>
<td>76.4 ± 33.9*</td>
<td>278.1 ± 55.2*†</td>
</tr>
<tr>
<td><strong>Plasma FFA</strong></td>
<td>MD</td>
<td>191 ± 68</td>
<td>199 ± 43</td>
<td>194 ± 63</td>
<td>216 ± 74</td>
<td>312 ± 113</td>
</tr>
<tr>
<td></td>
<td>HCD</td>
<td>133 ± 64</td>
<td>137 ± 40</td>
<td>217 ± 55</td>
<td>323 ± 102</td>
<td>405 ± 85*</td>
</tr>
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</table>

Values are means ± SE given in µM for 5 subjects. FFA, free fatty acids. *Significantly higher than rest, \( P < 0.05 \). †Significantly different from MD, \( P < 0.05 \).
Table 6. Plasma reproductive hormones

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Diet</th>
<th>Day 1 of Diet</th>
<th>Rest (day 7)</th>
<th>Time, min</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>E₂, pM</td>
<td>MD</td>
<td>444 ± 37</td>
<td>252 ± 57</td>
<td>279 ± 65</td>
</tr>
<tr>
<td></td>
<td>HCD</td>
<td>409 ± 56</td>
<td>302 ± 84</td>
<td>373 ± 111</td>
</tr>
<tr>
<td>P, nM</td>
<td>MD</td>
<td>29.6 ± 6.7</td>
<td>21.1 ± 7.7</td>
<td>21.7 ± 7.1</td>
</tr>
<tr>
<td></td>
<td>HCD</td>
<td>21.9 ± 4.9</td>
<td>26.3 ± 9.7</td>
<td>30.5 ± 11.7</td>
</tr>
</tbody>
</table>

Values are means ± SE for 5 subjects (except day 1 of diet and exercise-tapering regimen, which is for 6 subjects). E₂, estradiol; P, progesterone. *Significantly higher than values at rest, P < 0.05.

>400 mmol/kg dm, compared with MDs (7, 13, 20, 21, 34). In the present study, the women approached this absolute level and achieved this relative level of CHO intake, possibly accounting for the supercompensation that did occur.

However, as discussed by Tarnopolsky et al. (40), it may be difficult for women whose habitual caloric intakes are <2,400 kcal/day (12, 28, 40) to achieve higher CHO-intake values. This is lower than male athletes who consume >3,000 and often >5,000 kcal/day during CHO loading and can easily consume >500 g CHO/day during a 70–75% CHO diet. To test whether higher dietary CHO intake would increase muscle glycogen supercompensation, female athletes would need to increase their caloric intake to ~2,700 kcal/day for 3–4 days, with ~78% of the energy derived from CHO.

Dietary CHO, resting muscle glycogen content, and endurance performance. The increase in muscle glycogen in the HCD and tapering regimen was associated with a prolonged cycle time to exhaustion at ~80% VO₂peak during the LP in the female athletes. The performance increase (8%) was smaller than reported in some studies with men (20%) but was consistent with the general pattern of performance increases in CHO-loading studies where exercise lasts >90 min and muscle glycogen concentration is below ~110 mmol/kg dm at exhaustion (see Ref. 17 for review).

O’Keefe et al. (27) had trained female triathletes cycle to exhaustion at ~80% VO₂peak after three dietary conditions (72, 54, and 13% of the total caloric intake as CHO). They reported improved cycle performance after the HCD and MD compared with the low-CHO diet (113 ± 28, 98 ± 13, and 60 ± 12 min, respectively). The cycle times were longer on the HCD because five of seven subjects increased their performance after the HCD compared with the MD, but the increase was not statistically significant. However, muscle glycogen was not measured and menstrual status was not controlled for in this study (27).

It is unlikely that the increased cycle performance in the present study after the HCD regimen was solely dependent on an increased muscle glycogen store, because the HCD may have increased liver glycogen stores. Both liver glycogen and glucose production by the liver are very responsive to dietary CHO intake in men. A moderate-CHO diet (~40% CHO) resulted in a threefold greater hepatic glucose release than after a CHO-poor diet (25), and substantial increases in liver glycogen content were observed after a high-CHO diet compared with starvation or a CHO-poor diet (26).

Although the influence of the HCD vs. MD regimens on liver glycogen in women is unknown, the HCD would be expected to increase resting liver glycogen content. This may have resulted in a higher rate of glucose release from the liver in the latter stages of exercise, when muscle glycogen supply is low and contributed to the longer cycle time in the HCD performance ride.

Whole blood venous glucose concentrations were not different between the MD and HCD trials during cycling and were not at hypoglycemic levels at exhaustion in either trial. However, venous concentrations do not reflect the rate of appearance and disappearance of glucose during exercise. Furthermore, CHO oxidation was higher during cycling after the HCD regimen, implying that liver glucose provision may have contributed to the increased CHO oxidation throughout the trial. Additional studies examining glucose rates of appearance and disappearance during exercise after HCD and MD regimens are required to clarify this issue.

Potential mechanisms for reduced muscle glycogen synthesis in women. The present study and an earlier study (40) both suggest that well-trained women supercompensate glycogen to a smaller extent than do men. An explanation would likely involve gender differences related to the uptake and storage of glucose. These may include differences in the uptake of glucose during the basal state via GLUT-1; glucose uptake after exercise via the exercise and insulin-sensitive GLUT-4; phosphorylation of glucose by the enzyme hexokinase (HK); and factors associated with the synthesis of glycogen, including the activity of glycogen synthase (GS), the availability of glycogenin, and the formation of pro- and macroglycogen.

Although still controversial, there is strong evidence that the rate-limiting step for glucose uptake and glycogen synthesis in muscle is glucose transport (as reviewed in Refs. 4, 18). However, we are unaware of any gender comparisons of GLUT-1 and -4 contents in human skeletal muscle, although no difference was reported in the rate of muscle glycogen synthesis in the 4-h period after 90 min of cycling in well-trained men and women (41).

This argues that glucose uptake from the basal GLUT-1 and GLUT-4 remaining in the plasma membrane and T tubules was not limiting glucose uptake in the women during this 4-h postexercise period. There have been reports of higher maximal HK activities in untrained men vs. women, but no differences were reported between active men and women.
(as reviewed in Refs. 33, 39). However, we were unable to find comparisons of maximal HK and GS activities in well-trained men and women, although the presence of testosterone has been shown to increase GS activity and glycogen synthesis in rat skeletal muscle (2). Lastly, the importance of glycogenin availability and the formation of pro- and macroglycogen for maximal glycogen storage have recently been investigated in human skeletal muscle (1), but gender comparisons do not exist.

Summary. We observed that well-trained, eumenorheic female endurance athletes who follow a 7-day diet and exercise-tapering regimen containing 48% of the total caloric intake as CHO achieved high muscle glycogen contents (625 mmol/kg dm) during the LP of the menstrual cycle. When the dietary CHO content was increased to 78% in the final 3–4 days of the diet and tapering regimen, muscle glycogen content increased a further 13%. The increase in muscle glycogen, and possibly liver glycogen, after the high-CHO regimen was associated with increased CHO oxidation and cycling time to exhaustion at ~80% \( \dot{V}O_{2max} \). Therefore, female athletes were able to supercompensate glycogen but to a smaller extent than generally reported in male athletes undergoing the same dietary and exercise-tapering regimen. Little information presently exists to explain this gender difference in the ability to store muscle glycogen.

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