Effect of short-term fat adaptation on high-intensity training

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ABSTRACT

STEPTO, N. K., A. L. CAREY, H. M. STAUDACHER, N. K. CUMMINGS, L. M. BURKE, and J. A. HAWLEY. Effect of short-term fat adaptation on high-intensity training. Med. Sci. Sports Exerc., Vol. 34, No. 3, pp. 449–455, 2002. Purpose: To determine the effect of short-term (3-d) fat adaptation on high-intensity exercise training in seven competitive endurance athletes (maximal VO₂ uptake 5.0 ± 0.5 L·min⁻¹, mean ± SD). Methods: Subjects consumed a standardized diet on d-0 then, in a randomized cross-over design, either 3-d of high-CHO (11 g·kg⁻¹·d⁻¹ CHO, 1 g·kg⁻¹·d⁻¹ fat; HICHO) or an isonenergetic high-fat (2.6 g CHO·kg⁻¹·d⁻¹, 4.6 g FAT·kg⁻¹·d⁻¹; HIFAT) diet separated by an 18-d wash out. On the 1st (d-1) and 4th (d-4) day of each treatment, subjects completed a standardized laboratory training session consisting of a 20-min warm-up at 65% of VO₂peak (232 ± 23 W) immediately followed by 8 × 5 min work bouts at 86 ± 2% of VO₂peak (323 ± 32 W) with 60-s recovery. Results: Respiratory exchange ratio (mean for bouts 1, 4, and 8) was similar on d-1 for HIFAT and HICHO (0.91 ± 0.04 vs 0.92 ± 0.03) and on d-4 after HICHO (0.92 ± 0.03) but fell to 0.85 ± 0.03 (P < 0.05) on d-4 after HIFAT. Accordingly, the rate of fat oxidation increased from 31 ± 13 on d-1 to 61 ± 25 μmol·kg⁻¹·min⁻¹ on d-4 after HIFAT (P < 0.05). Blood lactate concentration was similar on d-1 and d-4 of HICHO and on d-1 of HIFAT (3.5 ± 0.9 and 3.2 ± 1.0 vs 3.7 ± 1.2 mM) but declined to 2.4 ± 0.5 mM on d-4 after HIFAT (P < 0.05). Ratings of perception of effort (legs) were similar on d-1 for HIFAT and HICHO (14.8 ± 1.5 vs 14.1 ± 1.4) and on d-4 after HICHO (13.8 ± 1.8) but increased to 16.0 ± 1.3 on d-4 after HIFAT (P < 0.05). Conclusions: 1) competitive endurance athletes can perform intense interval training during 3-d exposure to a high-fat diet, 2) such exercise elicited high rates of fat oxidation, but 3) compared with a high-carbohydrate diet, training sessions were associated with increased ratings of perceived exertion. Key Words: CYCLING, INTERVAL TRAINING, HIGH-FAT DIET

The effect of fat adaptation on the capacity to perform intense aerobic exercise is equivocal (5,13,14,16,19). Such a nutritional strategy, however, has the potential to substantially enhance endurance via dietary-induced changes in the patterns of fuel utilization, which promote fat oxidation and spare muscle glycogen (5). Indeed, we have previously reported that 5 d of high-fat intake combined with prolonged endurance training elicited a twofold increase in the rate of fat oxidation during moderate-intensity cycling (5).

Immediately before major endurance events, competitive athletes frequently undertake specialized training (24) and nutritional (10) practices. During this period, the intensity of training is increased, with a concomitant reduction in volume (24). Endurance athletes may also choose to undergo a short (3–5 d) period of a high-fat diet, followed by the traditional carbohydrate-loading regimen in the 2 d before the event (9). However, in untrained individuals, the ingestion of a high-fat diet for 3–5 d impairs exercise capacity, even when the training intensity is as low as 70% of maximal oxygen uptake (VO₂max) (6,7). We too have previously observed that, compared with a high-CHO diet, well-trained athletes on a high-fat diet experience symptoms of lethargy and increased fatigue, which is particularly evident during intense training sessions (5).

Unfortunately, in our previous investigation, we did not quantify the metabolic demands of intense training nor the athlete’s subjective responses to the different dietary regimens (5). Other workers who have studied the effect of fat adaptation on metabolism and performance also failed to monitor the training responses of their moderately trained (16) or well-trained (19) subjects. If athletes are to benefit from specific nutritional/training regimens, it is necessary that training intensity can be maintained while consuming a high-fat diet (12). Therefore, the aim of the current investigation was to determine whether competitive endurance athletes could complete high-intensity training sessions, typical of those that are incorporated into a taper, while consuming a high-fat diet.

METHODS

Subjects

Seven well-trained competitive male cyclists or triathletes (age 24 ± 6 yr, mass 75.3 ± 5.8 kg, peak O₂ uptake

0195-9131/02/3403-0449/$3.00/0

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Submitted for publication March 2001.
Accepted for publication June 2001.

449
[\text{VO}_{2\text{peak}}] 5.0 \pm 0.5 \text{ L.min}^{-1}, \text{peak aerobic power output [PPO]} 404 \pm 40 \text{ W}, \text{mean} \pm \text{SD} \) with a history (>4 yr) of regular endurance training (20 \pm 6 \text{ h} \cdot \text{wk}^{-1}) were recruited to participate in this study. Subjects were informed of the experimental procedures and possible risks before providing their written consent in accordance with guidelines outlined by the Human Research Ethics Committee of RMIT University. These subjects were either national or international level athletes (three of them competed in the 2000 Hawaii Ironman Triathlon World Championship within weeks of completing this study).

**Preliminary Testing**

Each subject performed a maximal, incremental cycle test to exhaustion on an electro-magnetically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). The test protocol (11) and gas collection procedures (4) have been previously described in detail. The results of the maximal test were used to determine the power outputs that corresponded to 65% and 85% of \text{VO}_{2\text{peak}} which were the intensities chosen for the laboratory training sessions (subsequently described).

**Experimental Protocol**

After 1 day (d-0) of standardized diet (58% CHO: 9.7 g CHO-kg\(^{-1}\)-d\(^{-1}\), 27% fat, 15% protein; total energy 0.25 MJ-kg\(^{-1}\)-d\(^{-1}\)) and light training (2–4 h of submaximal road cycling), subjects undertook two 3-d dietary treatments in a randomized, cross-over design with an 18-d washout period separating each diet. Each subject was prescribed either a high-fat (>65% of energy; HIFAT), low-CHO (<20% of energy) diet, or an isoenergetic (0.25 MJ-kg\(^{-1}\) BM) high-CHO (70–75% of energy; HICHO), low-fat (<15% of energy) diet. The energy intake in each diet was estimated, based on self-reports of habitual intake and factorial calculations of anticipated energy expenditure, to maintain energy balance for these subjects. The habitual diets of these subjects were estimated to be similar in macronutrient composition to the standard diet (i.e., ~ 10 g-kg\(^{-1}\)-d\(^{-1}\) CHO, providing 60% of energy). Diets were constructed to maximize, or at least match, absorbable energy; fiber intake was kept to a mean daily intake of 50 g and matched to within 5–10 g each day between treatments. Carbohydrate-containing foods with a very low glycemic index or high content of resistant starch were avoided. All meals and snacks were supplied to subjects, with diets individualized for food preferences. At least one meal each day was eaten under supervision in the laboratory, with the remaining food for each 24-h period being provided in pre-prepared packages. Subjects kept a food diary, reporting all food and drink intake on a daily basis to maximize compliance to the prescribed diets. Any food not consumed was returned and deducted from that day’s total intake. In addition, daily training records were maintained in a diary provided to each subject to ensure training was similar during both periods of dietary intervention. Although subjects were aware of the treatment being received (because it was not possible to completely blind the diets), the investigator responsible for data collection was kept blind to the order of treatments.

To determine the effect of the dietary treatments on substrate metabolism during exercise, two high-intensity interval-training sessions were scheduled into each athlete’s training program. These standardized sessions were conducted under supervision in the laboratory on the Lode cycle ergometer. The first interval training session was undertaken after an overnight fast and before the commencement of each dietary intervention (d-1). The intention of this session was to cause a marked lowering of muscle glycogen concentration (25) and initiate a rapid differentiation between dietary treatments based on the subject’s ability to restore depleted glycogen. A second laboratory session was undertaken on the last day (d-4) of each treatment (i.e., after 3 d of dietary intervention), also after an overnight fast.

On the day of an interval training session, subjects reported to the laboratory between 0700 and 0800 h after a 10- to 12-h overnight fast. They were weighed and a Teflon cannula (20 G, OptivaTM, Johnson & Johnson Intl., Brussels, Belgium) was inserted in a forearm antecubital vein for rapid continuous blood sampling via a sterile stopcock (Safe-site valve, B. Braun Medical Inc., Lakeside, PA). At this time, a 10-mL blood sample was taken. After each draw, the cannula was flushed with 1–2 mL of 0.9% sterile saline to keep the vein patent. After resting quietly in a seated position for 10 min, subjects mounted the cycle ergometer and commenced a 20-min warm-up ride at a constant work load that elicited ~65% of \text{VO}_{2\text{peak}}. A further blood sample was taken during the last 60 s of this ride. After a 10-min rest during which time they remained seated on the ergometer, subjects began the interval training session, which consisted of 8 × 5 min work bouts at 86 ± 2% \text{VO}_2\text{peak} (323 ± 32W) with a 60-s period of active recovery (100 W) between bouts (a work: rest ratio of 5:1). Such a training session is typical of a workout performed by ultra-endurance athletes during a taper (Hawley JA, unpublished observations). Laboratory conditions were maintained at 20°C and 46% relative humidity. Subjects were cooled with a fan (wind speed of ~ 7 m-s\(^{-1}\)) and provided with water \textit{ad libitum} throughout the training session. During the last 10 min of the 20-min ride and throughout the 1st, 4th, and 8th work bouts of the interval training session, pulmonary gases were sampled. Blood samples (~10 mL) were obtained upon completion of the 1st, 4th, and 8th repetitions, and subjects’ heart rates (HR) were continuously monitored via telemetry (Accurex Plus; Polar Electro OY, Kempele, Finland). Subjective ratings of perceived exertion (RPE) for the legs on a 6–20 scale (2) were recorded at the completion of each work bout.

On days 2 and 3 of each dietary treatment, subjects maintained their normal training, which was recorded in a logbook. Immediately upon the completion of a training session, subjects rated the workout according to a 7-point scale, where 1 indicated that the subject “felt good during all parts of the workout” and 7 indicated they “felt terrible throughout the workout.” During the cross-over dietary intervention, subjects replicated their nonlaboratory training program. At the end of d-1 and d-4 of each treatment,
Subjects completed the Profile of Moods State (POMS) inventory (17) to detect any psychological changes associated with the dietary intervention.

**Analyses**

**Rates of substrate oxidation.** Rates of whole body CHO and fat oxidation (g·min⁻¹) were calculated from VCO₂ and VO₂ values by using nonprotein RER values (18). Accordingly, we have assumed that the amount of protein oxidized is small and that other metabolic processes that involve the production and/or utilization of O₂ and CO₂ (e.g., gluconeogenesis from proteins, ketone body formation, and lipogenesis) are negligible compared with the oxidation of glucose and fatty acids (18). Such assumptions seem reasonable: even when consuming the high-fat diet, subjects were ingesting ~200 g CHO·d⁻¹. These equations are based on the premise that VO₂ and VCO₂ accurately reflect tissue O₂ consumption and CO₂ production. In well-trained subjects similar to those employed in the current investigation, indirect calorimetry has previously been shown to be a valid method for quantifying rates of substrate oxidation during strenuous exercise at ~85% of VO₂peak (21). Rates of fat oxidation (μmol·kg⁻¹·min⁻¹) were determined by converting the g·min⁻¹ rate of triglyceride oxidation to its molar equivalent, assuming the average molecular weight of human triglyceride to be 855.3 g·mole⁻¹ and multiplying the molar rate of triglyceride oxidation by 3, because each molecule contains 3 mole of FA. Rates of CHO oxidation (μmol·kg⁻¹·min⁻¹) were determined by converting the g·min⁻¹ rate of CHO oxidation to its molar equivalent.

Total energy expenditure during the laboratory training sessions was estimated from steady-state VO₂ and VCO₂ values assuming 37.5 kJ·g⁻¹ and 16.9 kJ·g⁻¹ for fat and CHO, respectively.

**Blood Metabolites**

Blood samples (~3 mL) were immediately analyzed for glucose and lactate concentrations using a 2300 Stat Plus automated analyzer (Yellow Springs Instruments, Yellow Springs, OH). A further 3 mL of blood was added to a tube containing an aliquot of preservative consisting of ethylene glycol-bis (-aminoethyl ether)-N, N, N', N'-tetra-acetic acid and reduced glutathione in normal saline, mixed gently and spun in a centrifuge (J6-MC Beckman Instruments Inc., Yellow Springs, CA), at 4500 rev for 8 min. The plasma was later analyzed for free fatty acid (FFA) concentration using an enzymatic colorimetric method (Wako, NEFAC code 279–75409, Tokyo, Japan). The remaining blood was placed in a tube containing lithium heparin and spun at 4500 rev for 15 min; 500 μL of plasma were placed in a tube containing 500 μL of ice cold 3 M perchloric acid, mixed vigorously on a vortex and spun; 800 μL of supernatant was added to a tube containing 200 μL of 6 M potassium hydroxide, mixed and spun at 10,000 rev for 5 min. The resultant supernatant was analyzed for plasma glycerol concentration using an enzymatic fluorometric analysis (20).

**Statistical Analysis**

Data from the laboratory training sessions were analyzed using a repeated measures three-way ANOVA (diet × day × time). The volume of training, and RPE during training sessions on the two different diets, was analyzed with paired t-tests. These analyses were all conducted using the computer software Statistica for Windows version 5.1 (StatSoft Inc., 1997, Tulsa, OK). All data are expressed as mean ± SD, and significance was accepted when P ≤ 0.05.

**RESULTS**

**Dietary and training compliance.** Subjects had excellent compliance to the two dietary treatments: reported CHO intakes from d-1 to d-4 averaged 11.00 ± 0.03 and 2.60 ± 0.07 g·kg⁻¹·d⁻¹ for HICHO and HIFAT, respectively, and were significantly different (P < 0.001) as intended. Reported fat intakes for the same period were 1.01 ± 0.03 g·kg⁻¹·d⁻¹ for HICHO and 4.62 ± 0.23 g·kg⁻¹·d⁻¹ for HIFAT (P < 0.001). The total nonlaboratory training time completed during the dietary intervention periods was 820 ± 201 and 788 ± 196 min for HICHO and HIFAT, respectively (NS). Of this, 608 ± 198 and 578 ± 172 min was spent cycling (NS) and 210 ± 176 and 210 ± 172 min was spent in “other training” (swimming, running, or weights) for HICHO and HIFAT, respectively (NS).

**Heart rates, ratings of perceived exertion during training and profile of mood states (POMS).** The ratings of perceived exertion during nonlaboratory training were greater for both cycling (P < 0.05) and all “other training” (P < 0.01) during the HIFAT diet compared with the HICHO diet. There was no difference in the global POMS score on d-1 for the two dietary treatments (143 ± 71 and 134 ± 120). However, the POMS score was greater on d-4 of the HIFAT diet (232 ± 89) compared to d-4 of the HICHO diet (94 ± 90, P < 0.01). On d-1 of both dietary treatments, the individual POMS score for fatigue was similar (42 ± 21 vs 35 ± 20 for HICHO and HIFAT, respectively) but was higher on d-4 of HIFAT (66 ± 18, P < 0.01).

HRs during the 20-min ride before each laboratory interval training session averaged 130 beats·min⁻¹ and were not different between dietary treatments. During the interval training sessions, HR typically rose from 155 beats·min⁻¹ at the end of the first work bout to ~165 beats·min⁻¹ at the end of the last repetition: this rise was similar across treatment conditions. RPE (legs) was the same on d-1 for HICHO and HIFAT (14.1 ± 1.4 and 14.8 ± 1.5) and on d-4 of HICHO (13.8 ± 1.8). However, RPE was significantly greater on d-4 of HIFAT (16.0 ± 1.3) compared with all other conditions (P < 0.05). One subject was unable to complete the laboratory training session on d-4 of the HIFAT diet. In this case, he managed the first four work bouts at the prescribed intensity (85% of VO₂peak) but completed the last four repetitions at a reduced work rate (~70% of VO₂peak).

**Pulmonary gas measures and fuel oxidation.** Table 1 summarizes the pulmonary gas measures taken during...
the 20-min ride at 65% of \( \dot{V}O_2 \) peak and throughout work bouts 1, 4, and 8 of the interval training sessions performed at 86 ± 2% of \( \dot{V}O_2 \) peak. \( \dot{V}O_2 \) was similar for all 20-min rides (~3.2 L·min\(^{-1}\)), increasing to ~4.4 ± 0.3 L·min\(^{-1}\) during the high-intensity work bouts \((P < 0.001)\). RER values, which were similar during the 20-min ride for both days on the HICHO treatment and on d-1 of the HIFAT diet, were lower on d-4 of the high-fat diet \((0.79 ± 0.03 \text{ vs } 0.85 ± 0.03, P < 0.01)\). During the high-intensity work bouts, RER declined from bout 1 to bout 8 for both the HICHO treatments and d-1 of HIFAT \((P < 0.01)\). However, on d-4 of HIFAT, RER was lower for all work bouts compared with similar time points for all other conditions \((P < 0.01)\) and did not change significantly from the first to last work bout.

Figure 1 displays the rates of CHO oxidation (panel A) and fat oxidation (panel B) determined during the 20-min ride and work bouts 1, 4, and 8 of the interval training session. Rates of CHO oxidation were lower \((P < 0.01)\) during both the 20-min ride and for each work bout of the training session on d-4 of HIFAT compared with all other conditions. Accordingly, rates of fat oxidation (panel B) were significantly higher \((P < 0.05)\) on d-4 of HIFAT for the 20-min ride and throughout work bouts 1, 4, and 8 compared with all other measures. Figure 2 shows the contribution from fat and CHO oxidation to total energy expenditure during the 20-min ride and averaged over work bouts 1, 4, and 8 of the high-intensity interval training session. Total energy expenditure was not affected by dietary treatment during either the 20-min ride at 65% of \( \dot{V}O_2_{\text{max}} \) (~880 J·kg\(^{-1}\)·min\(^{-1}\)) or the high-intensity training sessions (~1225 J·kg\(^{-1}\)·min\(^{-1}\); Fig. 2). However, the contribution from fat oxidation to total energy expenditure increased from 45.8 ± 11.1% to 66.4 ± 11.9% \((P < 0.05)\) during the 20-min ride and from 22.3 ± 9.8 to 43.1 ± 8.5% during exercise at 85% of \( \dot{V}O_2_{\text{max}} \) \((P < 0.05); \text{Fig. 2}\) by d-4 of the high-fat diet.

**Blood and plasma parameters.** Figure 3 shows blood glucose concentration (panel A) and blood lactate concentration (panel B) at rest, at the end of the 20-min ride, and immediately upon completion of work bouts 1, 4, and 8 of the interval training sessions. Blood glucose concentration remained between 3.8 and 4.5 mmol·L\(^{-1}\) at all times and was not different between dietary treatments or over time (Fig. 3A). There was a significant diet \( \times \) day \( \times \) time interaction \((P < 0.05)\) such that blood lactate concentration during the interval training session was lower on d-4 of HIFAT compared with all other time points (Fig. 3B).

The concentrations of plasma FFA (panel A) and plasma glycerol (panel B) determined during both the 20-min ride and immediately after work bouts 1, 4, and 8 of the interval training session are displayed in Figure 4. Compared with d-1, plasma FFA concentrations were higher on d-4 for both the HICHO and HIFAT dietary interventions \((P < 0.01), \text{Fig. 4}\).

### TABLE 1. Pulmonary gas exchange measures during the laboratory exercise sessions.

<table>
<thead>
<tr>
<th>Measure</th>
<th>HICHO d-1</th>
<th>HICHO d-4</th>
<th>HIFAT d-1</th>
<th>HIFAT d-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 ) (L·min(^{-1}))</td>
<td>3.19 ± 0.22</td>
<td>3.18 ± 0.27</td>
<td>3.21 ± 0.24</td>
<td>3.27 ± 0.22</td>
</tr>
<tr>
<td>SS-ride</td>
<td>3.35 ± 0.31</td>
<td>3.32 ± 0.30</td>
<td>3.40 ± 0.33</td>
<td>3.45 ± 0.23</td>
</tr>
<tr>
<td>Work bout 1</td>
<td>4.25 ± 0.40</td>
<td>4.26 ± 0.40</td>
<td>4.25 ± 0.36</td>
<td>4.33 ± 0.38</td>
</tr>
<tr>
<td>Work bout 4</td>
<td>4.32 ± 0.42</td>
<td>4.32 ± 0.37</td>
<td>4.32 ± 0.40</td>
<td>4.38 ± 0.30</td>
</tr>
<tr>
<td>Work bout 8</td>
<td>4.35 ± 0.39</td>
<td>4.32 ± 0.32</td>
<td>4.40 ± 0.33</td>
<td>4.53 ± 0.23</td>
</tr>
<tr>
<td>RER</td>
<td>0.85 ± 0.03</td>
<td>0.85 ± 0.03</td>
<td>0.85 ± 0.04</td>
<td>0.79 ± 0.03</td>
</tr>
<tr>
<td>SS-ride</td>
<td>0.94 ± 0.04</td>
<td>0.94 ± 0.03</td>
<td>0.94 ± 0.05</td>
<td>0.86 ± 0.03</td>
</tr>
<tr>
<td>Work bout 1</td>
<td>0.91 ± 0.03</td>
<td>0.91 ± 0.03</td>
<td>0.90 ± 0.03</td>
<td>0.85 ± 0.03</td>
</tr>
<tr>
<td>Work bout 4</td>
<td>0.91 ± 0.03</td>
<td>0.90 ± 0.04</td>
<td>0.90 ± 0.03</td>
<td>0.85 ± 0.02</td>
</tr>
</tbody>
</table>

All values are mean ± SD for \( N = 7 \). \( \dot{V}O_2 \), oxygen consumption; HICHO, high-carbohydrate diet; HIFAT, high-fat diet; SS-ride, 20 min of cycling at a constant work load corresponding to 65% of peak \( \dot{V}O_2 \) uptake. * Significantly different from work bout 1 \((P < 0.01)\). † Significantly different to HICHO d-1, HICHO d-4, and HIFAT d-1 \((P < 0.01)\).
Fig. 4A). There was a significant diet \times \text{day} \times \text{time} interaction ($P < 0.05$) for plasma glycerol. Plasma glycerol concentration (panel B) increased over time such that the concentration was higher on d-4 of HIFAT after 20 min and after work bouts 4 and 8 of the high-intensity training session ($P < 0.01$). Plasma glycerol concentration was also higher after work bouts 4 and 8 compared with the first repetition on d-4 of HIFAT ($P < 0.01$; Fig. 4B).

**DISCUSSION**

Improvements in endurance capacity during long-term (7 wk) training are markedly impaired when untrained subjects ingest a fat-rich diet compared with a high-carbohydrate diet (13). Short-term ($\leq 6$ d) adaptation to a high-fat, low-carbohydrate diet is also detrimental to submaximal endurance performance in untrained individuals (9,12,13). Phinney et al. (19) also proposed that there was a limitation to the intensity of exercise that can be performed by “elite bicyclists” after a high-fat diet. However, as has been recently noted (12), it is fundamental to competitive athletes that training capacity is not compromised in the immediate pre-competition or taper phase when specialized nutritional (10) and/or training (24) practices are followed. Hence, the first finding of the current study was that highly trained competitive endurance athletes were able to perform intense aerobic interval training sessions while consuming a high-fat, low-carbohydrate diet. To the best of our knowledge, the absolute (325 W) and relative (86% of VO$_{2\text{max}}$) work rates sustained by our subjects during laboratory training sessions are among the highest ever reported in the literature for human subjects consuming a high-fat diet.

Commensurate with the impressive absolute work rates sustained by our subjects during intense interval training after 4 d of a fat-adaptation were the high rates of fat oxidation. Previously, we have reported whole body rates of fat oxidation of 57 \(\mu\text{mol-kg}^{-1}\cdot\text{min}^{-1}\) for competitive endurance athletes (VO$_2\text{peak}$ 65 mL·kg$^{-1}$·min$^{-1}$) cycling at 70% of VO$_{2\text{peak}}$ (234 W) after 5 d of a fat-rich diet (5). In close agreement with these values, the rates of fat oxidation for the subjects in the present investigation were 69 ± 25 \(\mu\text{mol-kg}^{-1}\cdot\text{min}^{-1}\) when cycling at 65% of VO$_{2\text{peak}}$ (232 ± 23 W). However, despite an increase in exercise intensity from 65% of VO$_{2\text{peak}}$ to 85% of VO$_{2\text{peak}}$ (323 ± 32 W), the rates of fat oxidation did not decline: at the higher power output, rates of fat oxidation still averaged 61 \(\mu\text{mol-kg}^{-1}\cdot\text{min}^{-1}\). This indicates that after the high-fat diet, subjects were better able to oxidize lipids during high-intensity exercise to compensate for their (presumably) low muscle glycogen stores. In support of this contention, blood glycerol concentration was higher during both cycling at 65% and 85% of VO$_{2\text{peak}}$ after 4 d of the high-fat diet compared with the high-carbohydrate diet. Unfortunately, without muscle biopsies and/or tracer techniques, it is impossible to determine the source(s) of the additional fat utilized during intense exercise after fat-adaptation.

Comparison of data from subjects in the current investigation with the integrated model presented by Brooks and Brooks (20) and Brooks et al. (21) suggests that the rate of fat oxidation is increased in response to a high-fat diet. This is consistent with the current findings, as the rates of fat oxidation were higher during both cycling at 65% and 85% of VO$_{2\text{peak}}$ after 4 d of the high-fat diet compared with the high-carbohydrate diet. Unfortunately, without muscle biopsies and/or tracer techniques, it is impossible to determine the source(s) of the additional fat utilized during intense exercise after fat-adaptation.

**Comparison of data from subjects in the current investigation with the integrated model presented by Brooks and Brooks (20) and Brooks et al. (21) suggests that the rate of fat oxidation is increased in response to a high-fat diet.**
Mercier (3) reveals a substantially larger contribution of fat to energy expenditure. There are several possible reasons for this discordance. First, the subjects in the present study were all highly trained competitive ultra-endurance athletes undertaking prolonged, strenuous training at the time of investigation (several competed in the Hawaii Ironman World Championships within weeks of completing this study). Apart from the investigations of Romijn et al. (21, 22), previous studies examining fuel metabolism have, for the most part, employed moderately trained individuals. Although the $\dot{V}O_2_{\text{max}}$ values of the subjects in the study of Burke et al. (5) were similar to those in the present experiment, $\dot{V}O_2_{\text{max}}$ values alone fail to provide valid information regarding the training status of an individual. Second, we have previously reported (5) that fat adaptation in concert with strenuous endurance training lowers resting muscle glycogen content to ~250 mmol·kg dry weight$^{-1}$. In that study, laboratory training sessions were not monitored and subjects had difficulty completing the prescribed sessions (5). On the contrary, subjects in the present investigation completed the prescribed training sessions, in the face of (presumably) very low preexercise muscle glycogen levels: the rates of substrate oxidation in the model presented by Brooks and Mercier (3) are from individuals tested in a glycogen-replete state. Taken collectively, we are confident that our data represent the upper limits to fat oxidation during strenuous exercise.

Given such high rates of fat oxidation after a high-fat diet as observed in the present study, it is interesting to speculate on the maximal intensity of exercise that can be sustained when fat is the major fuel. Although it has been suggested that the exclusive oxidation of fat cannot sustain exercise much above an intensity of 50% of $\dot{V}O_2_{\text{peak}}$ (8), such a hypothesis remains unproven. To the best of our knowledge, the only study to systematically investigate the effect of exercise intensity on substrate metabolism in well-trained male subjects was that of Romijn et al. (22). Using stable isotope techniques in association with indirect calorimetry, these workers observed that cycling at 65% of $\dot{V}O_2_{\text{peak}}$ elicited the highest rates of whole body fat oxidation (43 $\mu$mol·kg$^{-1}$·min$^{-1}$). Interestingly, in that study (22), exercise at 25% and 85% of $\dot{V}O_2_{\text{peak}}$ resulted in similar rates of total fat oxidation (27–30 $\mu$mol·kg$^{-1}$·min$^{-1}$).

In the present investigation, one subject failed to complete the laboratory interval training session on d-4 of the high-fat diet. The RER value for this subject at the end of the fourth repetition at 86% of $\dot{V}O_2_{\text{peak}}$ was 0.79 (~30% of energy from carbohydrate and 70% from fat). In order for this subject to complete the final four 5-min repetitions, the power output had to be dropped to an intensity corresponding to ~70% of $\dot{V}O_2_{\text{peak}}$. Astrand et al. (1) have previously reported that when untrained subjects commenced exercise with low starting muscle glycogen stores, they could only attain a workload that elicited ~85% of the $\dot{V}O_2_{\text{peak}}$ attained in the control condition. It is not entirely clear why the capacity to perform intense exercise declines when fat is the predominant fuel source. The availability of carbohydrate as glycogen in the working muscle may be important (3), as too is the fact that carbohydrate is needed to maintain tricarboxylic acid cycle intermediates at a level needed to support the oxidative capacity of the muscle (23). However, it is likely that the main reason why carbohydrates are the preferred fuel during intense exercise is that the energy derived per unit of oxygen consumed is greater than fat (8).

Although subjects in the present study were able to complete the prescribed training while consuming a high-fat diet, this was associated with an increase in perceived effort. Both on-road cycling and all other nonlaboratory training sessions were characterized by significantly higher ratings of perceived training effort when subjects consumed the high-fat compared to the high-carbohydrate diet. Ratings of perception of effort of the legs were significantly higher during the interval training after 4 d of the high-fat diet compared with all other days.

In an effort to assess the impact of dietary changes on training and daily life, the POMS inventory was administered to subjects at the end of each day of the current investigation. The global POMS score was significantly higher on d-4 of the high-fat diet compared with all other days for both dietary interventions. Only one other study has administered the POMS inventory to “athletes” while consuming a high-fat diet and attempting to maintain their normal training schedule. In that investigation, Keith et al. (15) reported an elevation in total mood score but no difference for the fatigue component of the inventory when their female subjects con-
sumed 7 d of a low-carbohydrate diet versus a high-carbohydrate diet part. Of the reason for the difference in results between the current study and that of Keith et al. (15) could be that the subjects in our study were all highly trained athletes who were working toward competitive goals and who maintained their normal training intensity. On the other hand, the “athletes” (\( \text{VO}_{2\text{peak}} = 55 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \)) in the study of Keith et al. (15) may have reduced the intensity of their low training volume (~110 km \cdot wk^{-1}) and exercised at the same perception of effort on both high and low-carbohydrate diets.

One practical conclusion from the results of the present study is that highly trained and motivated competitive endurance athletes can perform both intense interval training and prolonged, submaximal endurance sessions while consuming a high-fat, low-carbohydrate diet for 4 d. However, compared with an isonitrogenous high-carbohydrate diet, general training sessions and high-intensity laboratory workouts were associated with increased ratings of perceived effort. Therefore, when undertaking specialized training and nutritional practices (i.e., a taper), we recommend that athletes work to objective measures of training intensity (i.e., power output) rather than subjective factors (i.e., ratings of perception of effort). Furthermore, athletes should be aware that if they undertake a short-term period of nutritional periodization, they experience a general increase in fatigue and a reduced feeling of well-being.

Perhaps the more interesting conclusion from the present study is that after short-term adaptation to a high-fat, low-carbohydrate diet, highly trained competitive athletes were able to complete intense aerobic exercise. Cycling at the high absolute (~325 W) and relative (i.e., 86% of \( \text{VO}_{2\text{peak}} \)) intensities after fat-adaptation elicited rates of fat oxidation that are among the highest reported in the literature (i.e., >60 \( \mu \text{mol} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \)) for that power output. However, fat oxidation alone cannot sustain exercise at power outputs requiring >60–65% of \( \text{VO}_{2\text{peak}} \), even in highly trained athletes adapted to a high-fat diet.

This study was funded by a grant from Nestlé Australia made to the Department of Sports Nutrition at the Australian Institute of Sport. We thank Dr. Inigo Mujika, Fabien Gargano, Robyn Hunter, and Bronwyn Jones for excellent technical assistance.

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