Dietary Composition Influences Short-term Endurance Training-Induced Adaptations of Substrate Partitioning During Exercise

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The purpose of the current study was to examine the influence of dietary composition on short-term endurance training–induced adaptations of substrate partitioning and time trial exercise performance. Eight untrained males cycled for 90 min at ~54% aerobic capacity while being infused with [6,6\(^2\)H]glucose before and after two 10-d experimental phases separated by a 2-week washout period. Time trial performance was measured after the 90-min exercise trials before and after the 2nd experimental phase. During the first 10-d phase, subjects were randomly assigned to consume either a high carbohydrate or high fat diet while remaining inactive (CHO or FAT). During the second 10-d phase, subjects consumed the opposite diet, and both groups performed identical daily supervised endurance training (CHO+T or FAT+T). CHO and CHO+T did not affect exercise metabolism. FAT reduced glucose flux at the end of exercise, while FAT+T substantially increased whole body lipid oxidation during exercise and reduced glucose flux at the end of exercise. Despite these differences in adaptation of substrate use, training resulted in similar improvements in time trial performance for both groups. We conclude that (a) 10-d high fat diets result in substantial increases in whole body lipid oxidation during exercise when combined with daily aerobic training, and (b) diet does not affect short-term training-induced improvements in high-intensity time trial performance.

Key Words: physical exertion, [6,6\(^2\)H]glucose, high fat diet, high carbohydrate diet

Introduction

The balance of carbohydrate and lipid oxidation, substrate partitioning, during exercise is primarily influenced by exercise intensity (4). As exercise intensity...
increases, carbohydrate sources support a greater portion of total energy flux, while
the lipid contribution decreases. Substrate partitioning during exercise is also influ-
enced by secondary factors including endurance training and diet.

Endurance training results in the decreased oxidation of carbohydrate and
increased oxidation of lipid during exercise at the same absolute intensity. The
reduction of carbohydrate oxidation is a function of reduced muscle glycogenolysis
and glucose flux (3, 13). The increase in lipid oxidation is due to the increased
oxidation of plasma free fatty acids (26), other plasma triglycerides, or intramuscu-
lar triglyceride (34) in inactive (2) or active skeletal muscle. Substrate partitioning
during exercise at the same relative intensity following endurance training, on the
other hand, is relatively unchanged (3, 13). These metabolic changes appear to be
characterized by a period of rapid adaptation followed by more gradual changes
with continued training. Studies using 7–10 d of intensive endurance training have
demonstrated reduced muscle glycogenolysis (36) and glucose flux (31) and in-
creased whole body lipid oxidation (34) during exercise. While the mechanisms for
these early adaptations to training are not yet clear, they represent the foundation for
further adaptations with continued training.

Chronic dietary manipulations of several days or more may influence sub-
strate partitioning during exercise by altering substrate availability in addition to
inducing adaptations in skeletal muscle enzyme activities (18). High fat diets con-
sumed for 5–28 d increase whole body lipid oxidation during exercise in trained
subjects, likely as a result of increased lipid availability and decreased carbohydrate
availability (15, 28, 35). However, it appears that high fat diets also influence
substrate partitioning by promoting adaptations in key regulatory enzymes involved
in the oxidation of long-chain fatty acids (11, 15, 18). It is possible that these
adaptations in enzyme activity are partly responsible for the preservation of high fat
diet–induced increases in whole body lipid oxidation during exercise following the
restoration of carbohydrate availability (5, 6).

The fact that endurance training and dietary manipulations both individually
influence substrate partitioning during exercise raises the possibility that interac-
tions of these independent factors may exist. Several studies have examined the
influence of dietary composition on training capability and performance of trained
subjects (28, 35, 41). However, the use of trained subjects does not improve our
understanding of the influence of dietary composition on training-induced adapta-
tions in substrate partitioning during exercise. To date, this issue has only been
examined by Helge et al. in which healthy sedentary males were fed either high
carbohydrate or high fat diets while undergoing identical endurance training pro-
grams for either 4 wk (20) or 7 wk (19). At workloads corresponding to ~80% of
pretraining aerobic capacity (VO₂peak), high carbohydrate–fed subjects exhibited
unchanged whole body substrate oxidation after 2, 4, and 7 wk of training while high
fat-fed subjects demonstrated substantial increases in whole body lipid oxidation
after 4 and 7 wk of training. After 7 wk of training, these changes in whole body
substrate oxidation were coupled with equally reduced rates of muscle glycogenolysis
during exercise in both groups, suggesting that plasma glucose flux may have
increased in high carbohydrate–fed subjects and decreased in high fat–fed subjects.
This was not confirmed with tracer-derived measures of glucose kinetics. The lack
of postexercise biopsies at 2 and 4 wk of training did not allow for a detailed
examination of the interaction of training and diet at these time points. Despite
identical training programs, those fed the high fat diet showed an equal improvement in endurance performance after 4 wk, but an attenuated improvement after 7 wk (+68%), compared to those fed the high carbohydrate diet (+191%).

In summary, long- and short-term training and chronic dietary manipulations influence substrate partitioning during exercise. While a detailed examination of the interaction of training and diet over 7 wk has been completed (19), little is known of the interaction of short-term training and diet. Additionally, the only studies to examine changes in exercise performance with short-term training have used time-to-exhaustion tests (16, 20) that have been shown to be less reliable than time trial performance tests in familiarized trained subjects (24). Therefore, the purpose of the current study was to examine whether dietary composition influences short-term endurance training–induced adaptations of substrate partitioning and time trial exercise performance in previously untrained subjects. It was hypothesized that (a) a 10-d high fat diet alone would increase whole body lipid oxidation and reduce plasma glucose and estimated muscle glycogen use during 90 min of moderate intensity exercise, (b) the changes in substrate partitioning induced by the 10-d high fat diet would be magnified when combined with 10 d of endurance training, due to an additive effect of the high fat diet and training, (c) substrate partitioning during exercise would be unaltered by consumption of a high carbohydrate diet alone or in combination with 10 d of endurance training, and (d) subjects consuming the high carbohydrate diet would demonstrate greater improvements in time trial exercise performance after 10 d of training than those consuming the high fat diet.

**Methods**

**Subjects**

Eight healthy, nonsmoking, weight-stable, untrained males (age, 27 ± 2 yr; weight, 77.9 ± 2.8 kg; body fat, 15.6 ± 2.0%; VO$_{2\text{peak}}$, 45.2 ± 2.3 ml · kg$^{-1}$ · min$^{-1}$; lactate threshold, 54 ± 1% of pretraining VO$_{2\text{peak}}$) were recruited for the study. Subjects were considered untrained if they engaged in less than 2 h · wk$^{-1}$ of aerobic activity for at least 1 y and had an aerobic capacity of less than 55 ml · kg$^{-1}$ · min$^{-1}$. To reduce the chance of including a subject with abnormal glucose kinetics, only subjects with normal body composition (≥20% body fat), normal 8-h fasting blood glucose (<110 mg · dl$^{-1}$), and no family history of non-insulin dependent diabetes mellitus were enrolled. The procedures and risks were thoroughly explained to the subjects, and their written informed consent was obtained. The study was approved by the Biomedical Sciences Institutional Review Board of The Ohio State University.

**Experimental Design**

Subjects underwent two 10-d experimental phases during which they were fed either a high carbohydrate or high fat diet in randomized order. The compositions of the habitual and experimental diets are detailed in Table 1. Substrate partitioning during 90 min of moderate intensity exercise was assessed during isotope trials before and after each experimental phase. The first 10-d intervention was designed to examine the influence of dietary composition alone on substrate partitioning during exercise. This was followed by a 2-wk washout period to minimize any effects of the previous experimental diet (D.M. Medeiros, personal communication, April 1997) during which subjects consumed their habitual diets and remained
inactive. The second 10-d intervention phase was designed to examine the influence of dietary composition on short-term endurance training–induced adaptations in substrate partitioning during exercise and time trial exercise performance. The inability to washout the effects of prior training prevented the use of a crossover approach in which subjects would train while consuming both experimental diets in a randomized order. Thus, subjects completed only two of four possible experimental treatments (CHO or FAT, CHO+T or FAT+T), and the diet only and diet + training phases were analyzed separately.

**Preliminary Testing**

All exercise testing and training sessions were performed on an electronically braked bicycle ergometer (Lode Excalibur Sport, Groningen, Netherlands). Aerobic capacity was assessed by progressively increasing an initial workload of 50 W by 25–50 W every 2 min until voluntary exhaustion. Due to the novelty of this task to inactive subjects, two aerobic capacity tests separated by at least 3 d were performed at baseline to assure a true maximal effort. Expired gases were analyzed as described in the Respiratory Gas-Exchange Measurements section, and heart rate was measured by telemetry (Polar, Finland). True VO$_{2\text{peak}}$ was defined as the achievement of at least two of the following three criteria: (a) RER $\geq$ 1.10, (b) plateau in VO$_2$ despite increases in workload, and (c) maximum heart rate within 10% of the age predicted value. Aerobic capacity measurements were repeated before and after the diet + training phase to assess training induced changes. Lactate threshold was assessed 48 h after the determination of VO$_{2\text{peak}}$. Subjects reported to the laboratory 3–4 h fasted, well hydrated (500 ml of water within 3 h of testing), and abstained from caffeine consumption for 8 h and rigorous exercise for 48 h prior to the test. A Teflon catheter (21 gauge, 1.25 in.; Angiocath, Becton Dickinson, Franklin Lakes, NJ, USA) was secured in an antecubital or forearm vein, and subjects rested in a seated position for 5–10 min before the resting blood sample was taken on the bicycle ergometer. Subjects initially exercised for 6 min at a workload corresponding to approximately 30–40% VO$_{2\text{peak}}$. The workload was progressively increased by 25 W every 6 min until five stages were completed. Blood drawn at the end of each stage was assayed for lactate concentration, and lactate threshold was defined as the point at which lactate concentration was 1 mM above a line fit through the lactate values of the resting and first or second workloads (8). This information was used to set the workload during the isotope trials to one that corresponded with each subject’s lactate threshold. Workloads were set relative to lactate threshold rather than VO$_{2\text{peak}}$ to minimize intersubject variability in cardiac and metabolic responses to exercise (1). Lactate threshold measurements were repeated before and after the diet + training phase to assess training-induced changes. Following the determination of residual volume by nitrogen dilution, body density was measured by underwater weighing, and body composition was calculated using the Siri equation (42). Subjects completed a 4-d dietary recall (3 weekdays and 1 weekend day) at baseline to document habitual daily energy intake and composition.

**Subject Preparation**

Subjects were instructed to consume a mixed diet containing at least 250 g carbohydrate · d$^{-1}$ for 3 d prior to both experimental phases to assure similar and adequate
Table 1 Habitual and Experimental Dietary Composition

<table>
<thead>
<tr>
<th>Variable</th>
<th>Habitual</th>
<th>CHO</th>
<th>FAT</th>
<th>CHO+T</th>
<th>FAT+T</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kcal · d⁻¹</td>
<td>2298 ± 197</td>
<td>2760 ± 93</td>
<td>2904 ± 76</td>
<td>3253 ± 99*</td>
<td>3193 ± 118*</td>
</tr>
<tr>
<td>Carbohydrate (g · d⁻¹)</td>
<td>313.4 ± 43.4</td>
<td>490.7 ± 20.2</td>
<td>208.0 ± 3.5†</td>
<td>582.0 ± 18.7</td>
<td>230.8 ± 8.0†</td>
</tr>
<tr>
<td>Carbohydrate (%E)</td>
<td>53.8 ± 4.1</td>
<td>70.7 ± 1.7*</td>
<td>28.3 ± 0.3*†</td>
<td>71.0 ± 0.0*</td>
<td>28.5 ± 0.3*†</td>
</tr>
<tr>
<td>Carbohydrate (g · kg⁻¹ · d⁻¹)</td>
<td>4.2 ± 0.6</td>
<td>6.8 ± 0.2*</td>
<td>2.7 ± 0.0†</td>
<td>7.2 ± 0.1*</td>
<td>3.1 ± 0.1†</td>
</tr>
<tr>
<td>Fat (g · d⁻¹)</td>
<td>71.8 ± 10.1</td>
<td>44.3 ± 1.9</td>
<td>187.3 ± 4.7*†</td>
<td>51.8 ± 1.0</td>
<td>204.3 ± 8.0*†</td>
</tr>
<tr>
<td>Fat (%E)</td>
<td>28.0 ± 3.6</td>
<td>13.7 ± 0.9*</td>
<td>57.7 ± 0.3*†</td>
<td>13.8 ± 0.3*</td>
<td>57.0 ± 0.4*†</td>
</tr>
<tr>
<td>Protein (g · d⁻¹)</td>
<td>102.8 ± 12.7</td>
<td>104.3 ± 3.8</td>
<td>105.0 ± 4.1</td>
<td>121.0 ± 4.3</td>
<td>113.5 ± 4.3</td>
</tr>
<tr>
<td>Protein (%E)</td>
<td>17.4 ± 1.5</td>
<td>14.7 ± 0.9</td>
<td>14.3 ± 0.3</td>
<td>14.5 ± 0.3</td>
<td>13.5 ± 0.3</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE. CHO and FAT N = 3 per group; CHO+T and FAT+T N = 4 per group. %E = percent of total energy intake. *Significantly different than Habitual, p < .05; †Significantly different than corresponding CHO group, p < .05.
endogenous carbohydrate stores. In addition, subjects recorded items consumed the evening before their baseline isotope trial and repeated this meal the evening before their second baseline isotope trial. In preparation for each isotope trial, subjects were instructed to not exercise for 24 h, avoid caffeine for 8 h, and consume at least 500 ml of water within 3 h of testing. Subjects reported to the laboratory 6 h after consuming a small standardized meal (4.2 kcal · kg⁻¹; ~290 kcals, 66% carbohydrate, 15% fat, 19% protein) and began the exercise protocol following a 2-h resting infusion (i.e., 8-h fasted prior to exercise) for each isotope trial. Small meals of this type more closely resemble preexercise eating habits than overnight 12 h fasting.

**Glucose Tracer Infusion Protocol**

Prior to isotope infusion, Teflon catheters (21 gauge, 1.25 in.; Angiocath, Becton Dickinson, Franklin Lakes, NJ, USA) were placed in veins of the left and right forearms for blood collection and isotope infusion, respectively. The right forearm was covered with a flexible heating pad for the arterialization of venous blood and was secured with a large elastic bandage. This adapted technique was used recently (32), and pilot data indicated that it resulted in appropriate skin temperatures (40–45 °C) for arterialization of venous blood. A blood sample was taken before infusion to determine background isotopic enrichment. Subjects then received a primed (17 µmol · kg⁻¹) continuous (0.22 µmol · kg⁻¹ · min⁻¹) infusion of sterile, pyrogen-free [6,6²H]glucose (99% enriched; Cambridge Isotopes, Cambridge, MA, USA) that was maintained with a calibrated autosyringe (PHD 2000, Harvard Apparatus, South Natick, MA, USA). The glucose tracer solution was dissolved in 0.9% sterile saline by a licensed pharmacist and was infused through a 0.2-µm filter. To assure isotopic equilibrium, this infusion was continued for 2 h of rest. Pilot data indicated that isotopic equilibrium was attained within 75 min at rest. The tracer infusion rate was doubled to 0.44 µmol · kg⁻¹ · min⁻¹ to maintain an appropriate isotopic enrichment during exercise as confirmed by pilot data.

**Isotope Trial**

All isotope trials consisted of 90 min of continuous exercise at the same absolute workload corresponding to subjects’ pretraining lactate thresholds (~54% VO₂peak). Subjects did not complete familiarization trials of this 90 min exercise task to minimize acute training effects prior to the training phase of the study. During exercise, subjects were encouraged to consume 1200–1600 ml of water to maintain euhydration and were cooled by a fan. Ambient conditions remained constant across all trials (23.5 ± 0.3 °C, 744 ± 1 mm Hg, 62 ± 2% relative humidity).

**Blood Sampling**

After a background blood sample was taken immediately before the start of infusion, blood samples (10 ml) were drawn at 15-min intervals at the end of rest (~30, –15, and 0 min) and throughout exercise (15, 30, 45, 60, 75, and 90 min). The catheter was maintained patent by intermittent flushing with heparinized sterile saline. Blood samples were distributed into EDTA tubes for the assessment of hematocrit and glucose isotopic enrichment, into sodium fluoride–potassium oxalate tubes for glucose and lactate, and into serum separation tubes for non-esterified fatty acids.
(NEFA), glycerol, and insulin. Hematocrit was measured to assess changes in plasma volume during exercise. EDTA and sodium fluoride–potassium oxalate tubes were kept on ice, while the serum separation tubes were allowed to clot at room temperature for at least 15 min. All tubes were then centrifuged (1500 × g, 15 min, 4 °C) for the separation of plasma and serum. All samples were stored at –80 °C until analysis.

**Respiratory Gas-Exchange Measurements**

Respiratory measurements were made between minutes 10–20, 40–50, and 80–90 of exercise. Expired gases were analyzed with a mixing chamber metabolic system (Ametek S-3A/I oxygen and CD-3A carbon dioxide analyzers, Paoli, PA, USA; Consentius/Parvomedics metabolic software, heated pneumotach and pressure transducer, Sandy, UT, USA) that was calibrated with gases of known concentration (National Bureau of Standards) and a 3-L syringe prior to each collection period. Calibration procedures were repeated after each collection period to assess analyzer drift and, when necessary, respiratory values were adjusted accordingly.

**Exercise Performance**

Exercise performance was assessed with a time trial test following the 90 min of exercise and 15 min of rest. Subjects completed a target amount of work in the fastest time possible on an electronically braked ergometer in pedal dependent mode. The target amount of work was based on 65% of a subject’s maximum workload ($W_{\text{max}}$), determined within 1 week of the beginning of the diet + training phase of the study, and an approximate target time of 25 min (1500 s; 24).

$$\text{Target Work (J)} = 0.65 \times W_{\text{max}} \times 1500$$

A performance task with a target time of ~20–30 min was chosen because pilot data indicated that untrained subjects could not maintain high power outputs (at or slightly above lactate threshold) for much more than 30 min following the 90-min steady state exercise task. The time it took a subject to complete the target amount of work was used as the measurement of exercise performance. The only feedback provided to the subjects during the time trial was elapsed distance and power output. Subjects were not told their performance times until they completed the study. Heart rate, power output, and accumulated work were recorded at 1-min intervals. Respiratory measures were not taken during the time trial to minimize disturbances to subjects’ pacing strategies. A familiarization time trial was completed after a previous lactate threshold test to minimize learning effects on pacing strategy. Only one familiarization time trial was completed to minimize acute training effects prior to the training phase of the study.

**Dietary Manipulation**

Subjects were fed 10-d high carbohydrate and high fat diets in randomized order during the two experimental phases (Table 1). The energy content of the diets was designed to match subjects’ individual daily energy needs using the World Health Organization metabolic equations (46) and an activity factor of 1.5. These figures were adjusted for exercise energy expenditure during training calculated from individual workload-$V_{\text{O2peak}}$ regression equations and changes in body weight. The
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diets were designed to be different in carbohydrate and fat content, but similar in protein and fiber content as well as the ratio between polyunsaturated and saturated fats. All prepared foods were weighed to the nearest gram. Both diets were similar to those used by Helge et al. (19, 20).

A 3-d menu that included breakfast, lunch, dinner, and snacks was repeated during each experimental phase. Subject’s preferred foods were included to the extent possible. Subjects were provided with food on a daily basis and were instructed to not deviate from the prescribed diets. Compliance was improved with the inclusion of extra snacks in case of hunger and a list of low calorie “free foods” that could be added to meals. Subjects were not allowed to consume alcohol or caffeine during the study. Subjects recorded food not provided to them and omissions from the prescribed diet in journals if necessary.

Endurance Exercise Training Program

All subjects completed identical 10-d bicycle ergometer training programs that were supervised by the primary investigator. The 10-d training protocol consisted of three workouts performed in the fed state: (a) 4 d of 45 min at 60–65% VO_{2peak}, (b) 2 d of 90 min at 55–60% VO_{2peak}, and (c) 4 d of 6 1 min intervals at 85–100% VO_{2peak} separated by 3 min of recovery. This training program resembles those that have resulted in adaptations in glucose kinetics in 10 d (31). During the 10-d training period the subjects did not perform any other form of physical training and their activities were limited to walking to and from classes. Subjects warmed-up and stretched before each exercise session. A day of rest was included after the last day of training to allow for adequate rest before the final isotope trial.

Analytic Techniques

Energy Expenditure and Whole Body Carbohydrate and Lipid Oxidation. Energy expenditure values were derived from respiratory data averaged over the last 8 min of each 10-min collection period. Total energy expenditure (TEE), whole body carbohydrate, and lipid oxidation were calculated using the following stochiometric equations (12, 45) assuming a whole body nitrogen excretion rate of 135 µg · kg⁻¹ · min⁻¹:

\[
\text{TEE (kcal · min}^{-1} \text{)} = 3.9 \text{VCO}_2 / \text{RER} + 1.11 \text{VCO}_2
\]

Carbohydrate oxidation (mmol · min⁻¹) = 25.196 VCO₂ - 17.749 VO₂ - 0.21349 n

Lipid oxidation (mmol · min⁻¹) = 1.9357 VO₂ - 1.9357 VCO₂ - 0.031978 n

where VO₂ and VCO₂ are expressed in L · min⁻¹ and n is the nitrogen excretion rate. Rates of carbohydrate and lipid oxidation were converted to body weight–relative units (µmol · kg⁻¹ · min⁻¹), and lipid oxidation was converted to fatty acid oxidation by multiplying by 3 (3 mol fatty acids/1 mol triglyceride). The total grams of carbohydrate and lipid oxidized during 90 min of exercise were quantified by calculating the area under the substrate oxidation curves (5). Carbohydrate oxidation was divided into other carbohydrate (muscle glycogen and lactate) and plasma glucose oxidation by assuming 100% oxidation of glucose Rₚ (25).
Isotopic Enrichment. Plasma samples were deproteinized and derivatized using an adapted technique (14) prior to analysis. Plasma samples (500 mL) were deproteinized with the addition of 860 mL of ice cold 0.3 N ZnSO$_4$ and 860 mL of ice cold 0.3 N Ba(OH)$_2$, vortexed, and placed in an ice bath for 20 min. Samples were then centrifuged (1500 × g, 20 min, 4 °C), and the resulting supernatant was lyophilized by vacuum centrifugation in 103 75 mm borosilicate tubes. Dried samples were then capped and stored at 4 °C until derivatization. The pentaacetate derivative of glucose was formed with the addition of 100 mL of a 2:1 mixture of acetic anhydride and pyridine and 60 min incubation at 60 °C. Samples were then partitioned with the addition of 1.5 ml of distilled water and 400 mL of dimethylchloride followed by centrifugation (1500 3 g, 10 min). The water phase was removed by suction, and the dimethylchloride phase was dried under a stream of N$_2$ gas. The dried pentaacetate glucose derivative was suspended in 50 mL of ethyl acetate and transferred to airtight chromatography vials with glass pipettes.

Glucose isotopic enrichment was measured by gas chromatography–mass spectrometry (Hewlett-Packard 5989A, Palo Alto, CA, USA). Duplicate 1 µL injections passed through a gas chromatograph (30 m × 0.25 mm DB-5 capillary column) and were then ionized in the mass spectrometer by electron impact ionization at an electron beam energy of 70 eV. Duplicate injections were used rather than duplicate samples due to pilot data indicating similar intra-assay coefficients of variation (CV) of 0.16–0.32%. The resulting data was processed by selected ion monitoring at mass-to-charge ratios (m/e) of 200, 201, and 202, and correction was made for the contribution of m/e 201 to the apparent enrichment of m/e 202 (45). Samples were analyzed in sets grouped by subject to reduce intra-subject variability. Within- and between-assay CVs averaged 1.3 and 5.3%, respectively, as determined by control specimens analyzed within each run.

Glucose Kinetics. Glucose rates of appearance ($R_a$) and disappearance ($R_d$) at rest were calculated using the steady state tracer dilution equation (45):

$$R_a (\mu\text{mol} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = R_d = F \cdot [(IE_i/IE_p) - 1]$$

where $F$ is the infusion rate of the isotope; $IE_i$ and $IE_p$ are isotopic enrichments of the infusate and plasma, respectively; and $-1$ accounts for the tracer’s contribution to the turnover rate of glucose. The glucose concentration of the infusate was determined within 24 h before each trial and was used with the subject’s weight to calculate $F$. The glucose concentration of the infusate was confirmed with the later analysis of a sample of the infusate taken immediately prior to infusion and stored at −80 °C.

Glucose kinetics during exercise were calculated using Steele’s non-steady state equations modified for use with stable isotope tracers (45):

$$R_a = \frac{F - V_d - \frac{C_m}{1+E} \frac{dE}{dt}}{1+E}$$

$$R_d = R_a - V_d \frac{dC_m}{dt} \left(1 + E\right) - C_m \frac{dE}{dt} \left(1 + E\right)^2$$
where $V_d$ is the effective volume of distribution, $E$ is the plasma isotopic enrichment, $C_m$ is the measured plasma concentration of the tracee, and $dE/dt$ and $dC_m/dt$ are maximum rates of change in enrichment and glucose concentration, respectively. The $V_d$ was assumed to be 100 ml · kg⁻¹.

**Substrates and Metabolites.** Plasma glucose, lactate, NEFA, and glycerol were all assayed spectrophotometrically in duplicate with a microplate reader using commercially available kits. Plasma glucose was measured using a hexokinase enzymatic kit (Sigma 16-UV, St. Louis, MO, USA) with intra- and inter-assay CV of 2.2 and 1.9%, respectively. Plasma lactate was measured using a lactate dehydrogenase enzymatic kit (Sigma 735, St. Louis, MO, USA) with intra- and inter-assay CV of 2.0 and 1.5%, respectively. Serum NEFA was measured using an enzymatic colorimetric kit (Wako NEFA-C, Neuss, Germany) with intra- and inter-assay CV of 3.1 and 2.5%, respectively. Serum glycerol was measured using a portion of a triglyceride kit (Sigma GPO Trinder, St. Louis, MO, USA) with intra- and inter-assay CV of 2.0 and 1.6%, respectively. Serum insulin was measured using a radioimmunoassay kit (ICN Pharmaceuticals, Costa Mesa, CA, USA) with an intra-assay CV of 3.5%.

**Statistical Analyses.** Due to the small sample sizes (3–4 subjects per group) and the inability to reliably test assumptions of normality and equality of variance, the data were analyzed by nonparametric statistics. The significance of between-group differences in subject characteristics and dietary composition was analyzed by Mann-Whitney U tests. Differences in aerobic fitness and time trial performance variables between groups and within groups over time were analyzed by Mann-Whitney U and Wilcoxon matched pairs signed-rank tests, respectively. Between-group, within-trial differences (i.e., CHO_pre vs. FAT_pre) were analyzed by repeated Mann-Whitney U tests, while within-group, between-trial differences (i.e., CHO_pre vs. CHO_post) were analyzed by repeated Wilcoxon matched pairs signed-rank tests. The Holm procedure (21) was used with the repeated tests to protect against Type I errors. Significance was set a priori at $p < .05$. All data are presented as mean ± SE.

**Results**

Due to the inability of some subjects to complete the 90-min exercise task of the baseline isotope trial, 6 subjects completed the diet-only phase (3 per group), while 8 subjects completed the diet + training phase (4 per group). The data of these two non-finishing subjects were not included in the analysis. Power calculations prior to the study using published short-term training glucose kinetics data (7, 31) indicated that 3–7 subjects per group were needed to attain an 80% chance of detecting significant differences.

**Subject Characteristics, Aerobic Capacity, and Time Trial Performance**

There were no significant group differences in age, body weight, body composition, $V_O^{2peak}$, or lactate threshold at baseline. Changes in $V_O^{2peak}$, lactate threshold, and time trial performance with training are depicted in Table 2. The CHO+T group significantly increased $V_O^{2peak}$, lactate threshold, and lactate threshold power output. The FAT+T group did not increase $V_O^{2peak}$, but did increase lactate threshold and lactate threshold power output. The average heart rate during 90 min of exercise
was not affected by either CHO or FAT (155 ± 5 bpm). The average heart rate during 90 min of exercise decreased with training for the CHO+T group (154 ± 7 vs. 140 ± 3 bpm, \( p = .034 \)) but not the FAT+T group (152 ± 7 vs. 146 ± 6 bpm). Both groups demonstrated a significant (\( p < .05 \)) ~11% decrease in time trial time and an increase in average time trial power output. Heart rate during the time trial did not change with training (168 ± 3 vs. 163 ± 1 bpm, CHO+T \(_{\text{pre}}\) vs. CHO+T \(_{\text{post}}\); 168 ± 5 vs. 172 ± 7 bpm, FAT+T \(_{\text{pre}}\) vs. FAT+T \(_{\text{post}}\)).

**Dietary Composition**

Analysis of subjects’ food diaries indicated that compliance was very high, as there was little variation from the designed diets. The compositions of the habitual and experimental diets are detailed in Table 1. Habitual diets of all subjects were grouped because there were no differences in these diets among the subjects. The daily energy intake for the experimental diets successfully maintained body weight in all groups. The average change in body weight during the diet only phase was 0.1 ± 0.3 kg while, during the diet + training phase, it was −0.2 ± 0.4 kg. Energy intake was increased by ~20% to compensate for increased energy expenditure from training. The lower energy intake of the habitual diet is likely due to under-reporting of energy intake and inaccuracy of the 4-d dietary recall. The composition of the experimental diets differed substantially from the habitual diet. The carbohydrate content of the CHO and CHO+T diets was 60% higher than the habitual diet. The FAT and FAT+T diets contained 147% more fat than the habitual diet. Protein content was similar for all diets.

**Respiratory Exchange Ratio, Whole Body Carbohydrate, and Lipid Oxidation**

The respiratory exchange ratio (RER), whole body carbohydrate, and lipid oxidation results (Figure 1, Table 3) reveal the typical increase in lipid oxidation during prolonged exercise. The CHO diet did not alter the pattern of substrate oxidation during exercise, as there were no significant differences between CHO\(_{\text{pre}}\) and CHO\(_{\text{post}}\) for RER, whole body carbohydrate, or lipid oxidation rates during exercise. The FAT diet tended to increase whole body lipid oxidation at the end of exercise but not significantly. There were no significant differences between CHO+T\(_{\text{pre}}\) and CHO+T\(_{\text{post}}\) for RER, whole body carbohydrate, or lipid oxidation rates during exercise. FAT+T increased oxidation of lipid and decreased oxidation of carbohydrate during exercise. The RER of FAT+T\(_{\text{post}}\) was significantly lower than FAT+T\(_{\text{pre}}\) (\( p = .003 \)) and CHO+T\(_{\text{post}}\) (\( p = .004 \)). Whole body carbohydrate oxidation of FAT+T\(_{\text{post}}\) was significantly lower than FAT+T\(_{\text{pre}}\) between minutes 10–20 and 40–50 (\( p < .025 \)). Whole body lipid oxidation for FAT+T\(_{\text{post}}\) was significantly higher than FAT+T\(_{\text{pre}}\) between minutes 10–20 and 40–50 of exercise (\( p < .025 \)). These changes represented a 190 and 64% increase in whole body lipid oxidation rates for FAT+T\(_{\text{post}}\) compared to FAT+T\(_{\text{pre}}\) at 10–20 and 40–50 min of exercise, respectively.

**Glucose Kinetics**

The tracer infusion protocol resulted in isotopic equilibrium by the end of the rest period and maintained steady enrichment during exercise (Figures 2A and 2B).
Table 2  Aerobic Capacity, Lactate Threshold, and Time Trial Performance Before and After the Diet + Training Phase

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>% Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CHO+T</td>
<td>FAT+T</td>
<td>CHO+T</td>
</tr>
<tr>
<td>VO_{2peak} (L · min(^{-1}))</td>
<td>3.5 ± 0.2</td>
<td>3.6 ± 0.4</td>
<td>4.0 ± 0.2*</td>
</tr>
<tr>
<td>(ml · kg(^{-1}) · min(^{-1}))</td>
<td>42.2 ± 1.4</td>
<td>48.2 ± 4.0</td>
<td>48.1 ± 1.6*</td>
</tr>
<tr>
<td>Lactate threshold (%) pretraining VO_{2peak}</td>
<td>52 ± 2</td>
<td>57 ± 2</td>
<td>62 ± 2*</td>
</tr>
<tr>
<td>Lactate threshold (W)</td>
<td>117 ± 7</td>
<td>130 ± 16</td>
<td>134 ± 9*</td>
</tr>
<tr>
<td>Time trial time (min)</td>
<td>26.1 ± 1.4</td>
<td>23.7 ± 1.3</td>
<td>23.1 ± 0.5*</td>
</tr>
<tr>
<td>Average time trial power output (W)</td>
<td>152 ± 13</td>
<td>165 ± 14</td>
<td>172 ± 8*</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE. N = 4 per group. *Significantly different than corresponding Pretraining value, p < .05.
Plasma glucose homeostasis was maintained during exercise, as there were no significant differences in the plasma glucose concentration between rest and exercise (5.0 ± 0.1 and 4.9 ± 0.2 mmol·L⁻¹, respectively). Resting glucose $R_a$ and $R_d$ were not different for either CHO or FAT (Figures 3A and 3C). In general, glucose $R_a$ and $R_d$ increased approximately threefold above the resting rates by the end of exercise.

Figure 1 — Respiratory exchange ratio during 90 min of exercise: A. before and after diet only phase ($N = 3$ per group); B. before and after diet + training phase ($N = 4$ per group). Values are means ± SE. §§Significantly different than FAT+T pre, $p = .003$; #significantly different than CHO+T post, $p = .004$. 

Plasma glucose homeostasis was maintained during exercise, as there were no significant differences in the plasma glucose concentration between rest and exercise (5.0 ± 0.1 and 4.9 ± 0.2 mmol·L⁻¹, respectively). Resting glucose $R_a$ and $R_d$ were not different for either CHO or FAT (Figures 3A and 3C). In general, glucose $R_a$ and $R_d$ increased approximately threefold above the resting rates by the end of exercise.
Table 3 Whole Body Carbohydrate and Lipid Oxidation During 90 Min of Exercise Before and After the Diet Only and Diet + Training Phases

<table>
<thead>
<tr>
<th>Variable/group</th>
<th>Pre-manipulation exercise time (min)</th>
<th>Post-manipulation exercise time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole body CHO oxidation (µmol · kg(^{-1}) · min(^{-1}))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>185.3 ± 29.2</td>
<td>162.0 ± 22.1</td>
</tr>
<tr>
<td>FAT</td>
<td>162.2 ± 9.9</td>
<td>132.7 ± 11.5</td>
</tr>
<tr>
<td>CHO+T</td>
<td>130.6 ± 6.5</td>
<td>118.3 ± 5.9</td>
</tr>
<tr>
<td>FAT+T</td>
<td>155.2 ± 5.6</td>
<td>138.2 ± 10.2</td>
</tr>
<tr>
<td>Whole body lipid oxidation (µmol · kg(^{-1}) · min(^{-1}))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>1.9 ± 0.2</td>
<td>9.7 ± 1.3</td>
</tr>
<tr>
<td>FAT</td>
<td>2.3 ± 1.2</td>
<td>9.4 ± 2.7</td>
</tr>
<tr>
<td>CHO+T</td>
<td>5.5 ± 0.2</td>
<td>9.4 ± 1.8</td>
</tr>
<tr>
<td>FAT+T</td>
<td>5.0 ± 2.9</td>
<td>12.9 ± 1.6</td>
</tr>
</tbody>
</table>

Note. Values are means ± SE. CHO and FAT \(n = 3\) per group; CHO+T and FAT+T \(n = 4\) per group. §Significantly different than corresponding value of FAT+T\(_{pre}\), \(p < .025\).
There was no effect of CHO on glucose $R_a$ or $R_d$ during exercise. The average glucose $R_a$ during 90 min of exercise was lower for FAT$_{post}$ compared to FAT$_{pre}$ (23.4 ± 1.9 vs. 20.4 ± 1.4 µmol · kg$^{-1}$ · min$^{-1}$, FAT$_{pre}$ vs. FAT$_{post}$) and was significant during the last 30 min of exercise ($p = .023$). The average glucose $R_d$ during 90 min of exercise was lower for FAT$_{post}$ compared to FAT$_{pre}$ (23.7 ± 1.4 vs. 20.7 ± 1.8 µmol · kg$^{-1}$ · min$^{-1}$, FAT$_{pre}$ vs. FAT$_{post}$) and was significant during the last 30 min of exercise ($p = .014$).

Resting glucose $R_a$ and $R_d$ were not different for either CHO+T or FAT+T (Figures 3B and 3D). In general, glucose $R_a$ and $R_d$ increased approximately threefold above resting values by the end of exercise. The average glucose $R_a$ and $R_d$ for CHO+T$_{post}$ tended to be lower than CHO+T$_{pre}$ during the last 30 min of exercise but did not reach significance ($p = .06–.10$). The average glucose $R_a$ and $R_d$ for FAT+T$_{post}$ was lower than FAT+T$_{pre}$ during the last 30 min of exercise ($p < .025$).
Carbohydrate Sparing

A summary of total substrate oxidation is presented in Figure 4. Ten days of high fat feeding alone resulted in the sparing of ~18 g of carbohydrate during 90 min of exercise when FAT\textsubscript{post} was compared to FAT\textsubscript{pre}. When the same 10-d high fat diet was combined with 10 d of endurance training, the shift towards greater lipid oxidation in FAT+T\textsubscript{post} resulted in the sparing of ~36 g of carbohydrate compared to FAT+T\textsubscript{pre} primarily due to a reduction in the oxidation of muscle glycogen and lactate.

Metabolites and Insulin

Hematocrit did not change significantly during exercise, and therefore no plasma volume adjustments were necessary for either the concentrations of plasma metabolites or insulin. Across all conditions, plasma lactate concentration increased significantly from rest to exercise (0.9 ± 0.1 vs. 3.3 ± 0.5 mmol · L\textsuperscript{-1}, p < .05). Plasma lactate concentration during exercise was lower for FAT\textsubscript{post} and FAT+T\textsubscript{post} (2.7 ± 0.5 and 2.2 ± 0.3 mmol · L\textsuperscript{-1}) compared to FAT\textsubscript{pre} and FAT+T\textsubscript{pre} (3.6 ± 0.9 and 3.3 ± 0.5 mmol · L\textsuperscript{-1}), respectively (p < .05). There were no significant between- or within-group differences in serum NEFA (395.1 ± 51.8 and 361.0 ± 58.6 µmol · L\textsuperscript{-1}, rest and...
exercise), glycerol (92.0 ± 8.7 and 204.1 ± 31.7 µmol · L⁻¹, rest and exercise), or insulin (479.0 ± 50.0 and 408.7 ± 43.1 pg · ml⁻¹, rest and exercise) concentrations for either diet only or diet + training phases.

**Discussion**

The purpose of this study was to examine the influences of dietary composition on changes in substrate partitioning during exercise and time trial exercise performance after 10 d of endurance training in previously untrained subjects. The diet only phase revealed that neither the 10-d high fat diet nor the 10-d high carbohydrate diet greatly altered exercise metabolism. Ten days of training while consuming a high fat diet resulted in a substantial increase in whole body lipid oxidation during exercise. In contrast, 10 d of training while consuming a high carbohydrate diet did
not alter exercise metabolism. Despite the differences in substrate partitioning resulting from the diets during 10 d of training, both groups equally improved high intensity time trial performance.

Consuming a high carbohydrate diet for 3–5 d increases muscle glycogen concentration and may increase the rate of muscle glycogen oxidation during exercise (39). While it is likely that the 10-d high carbohydrate diet of the current study resulted in increased carbohydrate availability, whole body carbohydrate oxidation during exercise did not increase (Table 3; Figures 1 and 4). This may be due to the fact that subjects were exercising at an intensity that resulted in high RER values (0.91–0.98) and demanded a large contribution of carbohydrate (~94% of TEE at minute 15 and ~70% of TEE at minute 90) that could not be further elevated by increased carbohydrate availability. While it may be surprising that subjects exercising at ~54% VO\textsubscript{2peak} would have such high RER values, it should be remembered that these subjects were untrained, and this exercise intensity represented ~100% of their pretraining lactate threshold. Alternatively, although the relative carbohydrate intake of the experimental diet was significantly higher than the habitual diet (71% vs. 54% CHO, \( p < .05 \)), it may have not been high enough to increase whole body carbohydrate oxidation during exercise. Consumption of an 88% carbohydrate diet for 7 d resulted in a 17% increase in whole body carbohydrate oxidation during 1 h of exercise at 67% VO\textsubscript{2peak} in trained cyclists compared to a 68% carbohydrate diet (9).

Despite consistent evidence that short-term endurance training reduces carbohydrate use and increases lipid use during exercise (31, 34, 36), the results of this study indicate that whole body carbohydrate and lipid oxidation during exercise are relatively unaltered when training while consuming a high carbohydrate diet for 10 d (Table 3; Figures 1 and 4). Similar effects occur after 2–7 wk of training on a high carbohydrate diet (19, 20). Consuming a high carbohydrate diet while training may produce a significant increase in the muscle glycogen concentration that increases glycolysis during exercise. Estimations of muscle glycogen and lactate oxidation during exercise suggest that these sources represented a larger percent of substrate oxidation at the end of exercise after the CHO+T treatment (56%) than before (45%). Therefore, training-induced adaptations that promote increased lipid oxidation may have been counteracted by the effects of increased carbohydrate availability, leaving exercise metabolism relatively unchanged after training while consuming the high carbohydrate diet.

The 10-d high fat diet alone did not affect whole body lipid oxidation during exercise, although the trend for an increase at the end of exercise approached significance (\( p = .054 \), Table 3). This lack of change in the untrained subjects of the current study and the increase in whole body lipid oxidation during exercise in trained athletes after 5–28 d high fat diets (5, 6, 15, 28, 35) suggests that adaptations to endurance training may be necessary for substantial high fat diet adaptation. The results of the diet + training phase of the current study support this suggestion. The source of the lipid used to increase whole body lipid oxidation is not clear, as high fat diets have variable effects on lipid availability. Some have found that high fat diets elevate serum NEFA concentration during exercise (5, 35), while others (6, 15, 28), including the current study, have not observed this effect. Intramuscular triglyceride concentration increases after 5-d (23) and 28-d high fat diets (20, 27). Using limb balance techniques, Jansson and Kaijser (23) present evidence that both serum NEFA and intramuscular triglyceride oxidation are increased during exercise after a 5-d high fat diet. Schrauwen et al. (38), on the other hand, used [U-13C]palmitate
infusions during 60 min of exercise at 50% \( \text{VO}_{2\text{peak}} \) before and after a 7-d high fat diet and concluded that the increase in whole body lipid oxidation during exercise following the high fat diet was due to the enhanced oxidation of intramuscular triglyceride and/or plasma very low–density lipoprotein triglyceride, as there was no change in plasma free fatty acid oxidation.

The trend for an increase in whole body lipid oxidation only at the end of exercise following the high fat diet alone (Table 3, Figure 1) suggests that reduced carbohydrate availability may have played a role in the increase in lipid oxidation. A 14-d high fat diet resulted in lower resting muscle glycogen concentrations when compared to a high carbohydrate diet (68.1 \( \pm \) 3.9 vs. 120.6 \( \pm \) 3.8 mmol \( \cdot \) kg\(^{-1} \) wet weight, respectively; 28). However, recent evidence suggests that high fat diet–induced increases in whole body lipid oxidation during exercise persist following the restoration of carbohydrate availability (5, 6). These persistent adaptations may include changes in the activities of key enzymes. It has been suggested that the flux of substrates associated with changing diets may alter exercise metabolism by promoting adaptation of muscle enzymes (18). Thus, high fat diets may promote lipid oxidation by influencing the activity of \( \beta \)-oxidative enzymes through chronic elevations in the oxidation of fatty acids. High fat diets consumed for 3–49 d have resulted in increased activity of CPT-I (11, 15) and the \( \beta \)-oxidative enzyme \( \beta \)-hydroxyacyl-CoA-dehydrogenase (18) and decreased activity of PDH (37). However, others have noted no change in \( \beta \)-hydroxyacyl-CoA-dehydrogenase following 15-d (15) and 28-d (27) high fat diets.

Training while consuming the high fat diet resulted in substantial increases in whole body lipid oxidation during exercise (Table 3, Figures 1 and 4). A 15% decrease in whole body carbohydrate oxidation throughout exercise and a significant reduction in plasma lactate concentration during exercise accompanied the increased lipid oxidation. The source of the increased lipid oxidation may be intramuscular triglyceride as found by others after only 5 d of training (34). If the diet only phase of the experiment was not included, one may conclude that the high fat diet was primarily responsible for the increase in whole body lipid oxidation during exercise following FAT+T. However, although it cannot be tested statistically with the current design, the more pronounced whole body lipid oxidation (37.9 \( \pm \) 8.9 vs. 18.7 \( \pm \) 4.9 g lipid oxidized during 90 min of exercise, FAT+T \( \text{post} \) vs. FAT \( \text{post} \)) and lower whole body carbohydrate oxidation (130.5 \( \pm \) 12.8 vs. 163.3 \( \pm \) 15.8 g carbohydrate oxidized during 90 min of exercise, FAT+T \( \text{post} \) vs. FAT \( \text{post} \)), compared to the high fat diet alone, suggests an interaction between the high fat diet and short-term training (Table 3, Figures 1 and 4). Similar interactions have been suggested by others using rat models, where increased oxidative enzyme activity and improved endurance performance from intensive endurance training, were further magnified when combined with a high fat diet (40). However, Helge et al. (19) demonstrated that even if such interaction effects exist in humans, improvements in endurance performance are attenuated after 7 wk of training while consuming a high fat diet. Alternatively, the more pronounced increase in whole body lipid oxidation during exercise, when the high fat diet was combined with training, may have been due to a more rapid adaptation in whole body lipid oxidation. Adaptation of whole body lipid oxidation, a matching of lipid oxidation to intake, with a 50% fat diet in sedentary subjects occurred in approximately 7 d, but this process was reduced to approximately 2–4 d when combined with physical activity (43).
As with whole body carbohydrate oxidation, the current study found that the high carbohydrate diet had no detectable influence on glucose flux during 90 min of exercise (Figures 3A and 3C). While the diet may have increased carbohydrate availability, the fact that the exercise intensity already demanded a large proportion of energy from carbohydrate might have prevented any further increases in the oxidation of carbohydrate. In addition, subjects were tested following an 8 h fast that may have lowered the liver glycogen concentration to pre-manipulation levels precluding any changes in glucose flux.

Marginal reductions in glucose flux were found at the end of exercise following the high fat diet (Figures 3A and 3C). Martin et al. (29) found that a 3-d high fat diet (~90% of kcals from fat) reduced the oxidation of plasma glucose by nearly 50% during 30 min of moderate intensity exercise compared with a 3-d high carbohydrate diet (~75% of kcals from carbohydrate). Consumption of high fat diets for a longer period seems to induce a similar response. A 28-d high fat diet (~85% kcals from fat, <20 g carbohydrate · d⁻¹) produced a 40% decrease in plasma glucose use during exercise (35), but the mechanism for these changes is unclear. There is little support for the existence of the glucose–fatty acid cycle in contracting skeletal muscle (10, 33). Instead, it is believed that increased lipid availability may reduce increases in allosteric activators of key glycolytic enzymes thereby decreasing muscle glycogen and plasma glucose oxidation (10, 33). Reduced glucose oxidation during exercise following a high fat diet may be due to impaired glucose transport (17). High fat diets consumed for 8 wk reduce insulin- and contraction-stimulated glucose transport in animals by impairing the GLUT-4 translocation process (17). It is possible that such a mechanism exists in humans who consume a high fat diet.

Training while consuming a high fat diet produced a significant reduction in glucose flux at the end of exercise (Figures 3B and 3D). The greater reduction in glucose R_d at the end of exercise for FAT+T (–26%) compared to FAT (–15%) lends further evidence of the interaction of the high fat diet and short-term training. This effect may be a reflection of recent findings that endurance training enhances the inhibitory effect of lipids on whole body glucose metabolism during exercise (30).

The few short-term training studies that have reported VO_2peak data found either no change (36) or a 9–11% increase in VO_2peak (44) when subjects trained daily for 10 d. The current study found a significant 14% increase in the VO_2peak of the CHO+T group and no change in the VO_2peak of the FAT+T group (Table 2). No plausible explanation could be found for this finding, although the degree of variability in responses of the FAT+T group could be a factor. Additionally, the pretraining VO_2peak of the FAT+T group tended to be higher (48.2 ± 4.0 ml · kg⁻¹ · min⁻¹), although not significantly, than the CHO+T group (42.2 ± 1.4 ml · kg⁻¹ · min⁻¹), potentially limiting their improvement in aerobic capacity with the short-term training protocol. Improvements in lactate threshold with 10 d of training have not been previously reported. This study indicates that the training protocol produced significant increases in the power output associated with lactate threshold, and this adaptation was likely responsible for the improved time trial performance in both groups. Before training, subjects were capable of maintaining a power output during the time trial that was ~35 W above their pretraining lactate threshold. After training, they were capable of maintaining a power output during the time trial that was ~38 W above their posttraining lactate threshold. Despite marked differences in dietary composition and training-induced adaptations in steady state substrate use, both
groups were equally capable of improving high intensity time trial performance. These results agree with those of Helge et al. (19, 20) who showed that consumption of a high fat diet while training does not attenuate improvements in endurance performance compared to a high carbohydrate diet until sometime between 4 and 7 wk. It must be pointed out that the reliability of time trial performance in untrained subjects is not known. Jeukendrup et al. (24) clearly demonstrated that time trial performance was more reliable than time-to-exhaustion performance in familiarized trained subjects. They reasoned that this might be due to the subjective nature of the endpoint of time-to-exhaustion tests compared to the discrete endpoint of time trials allowing for a greater influence of psychological factors such as monotony and boredom. Despite the fact that exercise performance of untrained subjects is less reliable than that of trained subjects (22), it seems likely that the psychological factors explaining the poor reliability of time-to-exhaustion performance in trained subjects would also influence untrained subjects.

The carbohydrate sparing effect of high fat diets has led to the investigation of their potential as an ergogenic aid. While there is limited evidence that high fat diets may improve endurance performance (28), most have found no beneficial effect (5, 6, 15, 19, 20, 35). Consumption of a high fat diet during 10 d of training resulted in the sparing of ~36 g of carbohydrate during 90 min of exercise compared to those fed a high carbohydrate diet (Figure 4). The fact that this sparing did not improve performance after 90 min of exercise is not surprising, given that the sparing of ~70–120 g of carbohydrate during 120–240 min of exercise did not improve performance in previous studies (5, 6). It is estimated that ~133 and 102 g of muscle glycogen and lactate were oxidized during 90 min of exercise by CHO+T and FAT+T subjects, respectively. It is likely that these amounts were not enough to significantly deplete muscle glycogen stores prior to the time trial thus minimizing the advantage of the carbohydrate sparing effect of the high fat diet.

In conclusion, 10-d high carbohydrate and high fat diets alone do not substantially alter substrate partitioning during 90 min of exercise in untrained subjects. Whole body lipid oxidation was substantially increased throughout exercise when the high fat diet was accompanied by daily endurance training for 10 d. High carbohydrate diets consumed during short-term endurance training eliminate the training-induced increase in whole body lipid utilization. Despite these differences in adaptation of substrate use, untrained subjects that consumed either high carbohydrate or high fat diets for 10 d while training were equally capable of improving high-intensity time trial performance. The results of this study compliment those of the 4–7 wk studies of Helge et al. (19, 20) to clearly demonstrate an interaction between diet and short-term training. The collective results of these studies underscore the importance of dietary controls in future short- and long-term training studies.

References


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