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Fat adaptation followed by carbohydrate loading compromises high-intensity sprint performance

L. Havemann,[1] S. J. West,[1] J. H. Goedecke,[1] I. A. Macdonald,[2] A. St Clair Gibson,[1] T. D. Noakes,[1] and E. V. Lambert[1]

[1]*University of Cape Town/Medical Research Council Research Unit for Exercise Science and Sports Medicine, Department of Human Biology, University of Cape Town, South Africa; and* [2]*School of Biomedical Sciences, University of Nottingham Medical School, Queen's Medical Centre, Nottingham, United Kingdom*

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Havemann, L., S. J. West, J. H. Goedecke, I. A. Macdonald, A. St Clair Gibson, T. D. Noakes, and E. V. Lambert. Fat adaptation followed by carbohydrate loading compromised high-intensity sprint performance. *J Appl Physiol* 100: 194-202, 2006. First published September 1, 2005; doi:10.1152/japplphysiol.00813.2005.—The aim of this study was to investigate the effect of a high-fat diet (HFD) followed by 1 day of carbohydrate (CHO) loading on substrate utilization, heart rate variability (HRV), effort perception [rating or perceived exertion (RPE)], muscle recruitment [electromyograph (EMG)], and performance during a 100-km cycling time trial. In this randomized single-blind crossover study, eight well-trained cyclists completed two trials, ingesting either a high-CHO diet (HCD) (68% CHO energy) or an isoenergetic HFD (68% fat energy) for 6 days, followed by 1 day of CHO loading (8–10 g CHO/kg). Subjects completed a 100-km time trial on *day 1* and a 1-h cycle at 70% of peak oxygen consumption on *days 3*, *5,* and *7*, during which resting HRV and resting and exercising respiratory exchange ratio (RER) were measured. On *day 8*, subjects completed a 100-km performance time trial, during which blood samples were drawn and EMG was recorded. Ingestion of the HFD reduced RER at rest (P 0.005) and during exercise (P 0.01) and increased plasma free fatty acid levels (P 0.01), indicating increased fat utilization. There was a tendency for the low-frequency power component of HRV to be greater for HFD-CHO (P 0.056), suggestive of increased sympathetic activation. Overall 100-km time-trial performance was not different between diets; however, 1-km sprint power output after HFD-CHO was lower (P 0.05) compared with HCD-CHO. Despite a reduced power output with HFD-CHO, RPE, heart rate, and EMG were not different between trials. In conclusion, the HFD-CHO dietary strategy increased fat oxidation, but compromised high intensity sprint performance, possibly by increased sympathetic activation or altered contractile function.

muscle recruitment; rating of perceived exertion; heart rate variability; fat oxidation; endurance exercise

FATIGUE DURING ENDURANCE EXERCISE has been associated with, among other things, a depletion of muscle glycogen stores (2, 21). In an attempt to delay the onset of fatigue during endurance exercise, various nutritional strategies have focused on optimizing muscle glycogen stores before exercise and/or "sparing" muscle glycogen stores during exercise. A more recent nutritional strategy aimed at achieving this encompasses 5–6 days of fat loading, followed by 1 day of carbohydrate (CHO) loading before the event (3, 4, 6). This strategy has been shown to increase fat oxidation at rest and during exercise, in the fasted (3) and nonfasted state (4, 6), and even when CHOs are ingested during exercise (4, 6). This strategy has also been shown to increase muscle glycogen stores and reduce muscle glycogen utilization during exercise (3). However, despite this muscle glycogen-sparing effect, overall improvements in performance have not been demonstrated (3, 4, 6).

The effects of this particular dietary strategy on performance have only been tested under time-trial conditions (25 min to 1 h) after prolonged submaximal steady-state exercise [2–4 h at 65–70% of peak oxygen consumption ($\dot{V}O_{2\ peak}$)] (3, 4, 6). The effects of 5–6 days of fat loading, followed by 1 day of CHO loading, have not been investigated during exercise that simulates race conditions, which includes high-intensity (85% $\dot{V}O_{2\ peak}$) sprints. Because glycogen is the predominant fuel during high-intensity exercise (28), a nutritional strategy that not only stores muscle glycogen but also promotes glycogen sparing would most likely benefit endurance exercise that includes high-intensity exercise bouts. However, factors other than muscle glycogen content may also have an effect on exercise performance after a high-fat diet (HFD).

In fact, the ingestion of a HFD has been shown to increase sympathetic activation during exercise (17, 20, 29). Sasaki et al. (29) demonstrated an increase in sympathetic activation during exercise with 7 days of high-fat feeding (50% fat energy), which was associated with muscle glycogen depletion in the working muscle. Conversely, Helge et al. (17) demonstrated increased sympathetic activation during exercise with prolonged high-fat intake that persisted despite muscle glycogen restoration. Ingestion of the HFD was also associated with increased effort perception (18) and reduced endurance exercise capacity, possibly as a consequence of the increased sympathetic activation (17). Moreover, an increased effort perception has also been reported during high-intensity (85% $\dot{V}O_{2\ peak}$) sprint bouts (16.0 1.3 vs. 13.8 1.8) (33) and during submaximal exercise at intensities between 30 and 90% $\dot{V}O_{2\ peak}$ (14.3 2.5 vs. 12.6 2.2) (27) after 3 days of high-fat compared with 3 days of high-CHO feeding. The coupling between the sympathetic activation with high-fat intake and the increase in effort perception during exercise requires further investigation.

The decrement in performance with increased sympathetic activation associated with high-fat feeding may be related to alterations in central drive. Using microneurography, Seals and Enoka (30) found that increased muscle sympathetic activation

Address for reprint requests and other correspondence: J. H. Goedecke, UCT/MRC Research Unit for Exercise Science and Sports Medicine, Dept. of Human Biology, Univ. of Cape Town, PO Box 115, Newlands 7725, South Africa (e-mail: juliag@sports.uct.ac.za).

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during fatiguing isometric handgrip exercise was associated with increased electromyograph (EMG) activity. To our knowledge, no other studies have investigated the effect of dietary manipulation, in particular high-fat feeding, on neural recruitment strategies, central regulation and "awareness of fatigue" during exercise. Therefore, the aim of the present study was to investigate the effect a HFD followed by 1 day of CHO loading on substrate utilization, heart rate variability as a proxy measure of sympathetic activation, effort perception, muscle recruitment, and performance during a 100-km cycling time trial, including high-intensity sprints, simulating race situations.

METHODS

Subjects and preliminary testing. Eight endurance-trained male cyclists participated in this study, which was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town. All subjects were free from known metabolic conditions and were currently not taking any medications for chronic conditions such as high blood pressure or stimulants for conditions such as asthma. The subjects were informed of the nature of the study and written, informed consent was obtained before the start of the study. Body weight and height were measured to the nearest decimal place. The percent body fat was determined from measurements of skinfold thickness, using the equations of Durnin and Womersley (10). The characteristics of the subjects are summarized in Table 1.

$\dot{V}_{O_2\ peak}$ and peak power output (W_{peak}) were measured on an electronically braked cycle ergometer (Lode, Groningen, The Netherlands) modified with toe clips and racing handlebars as described by Hawley and Noakes (16). Work rates were started at 3.33 W/kg body mass and increased first by 50 W and then by 25 W every 150 s until the subject was exhausted. W_{peak} was defined as the highest exercise intensity the subject completed for 150 s (in W), plus the fraction of time spent in the final workload. During the progressive exercise test, ventilation volume, oxygen uptake ($\dot{V}_{O_2}$) and carbon dioxide production ($\dot{V}_{CO_2}$) were measured over 15-s intervals using a breath-by-breath Oxycon Alpha analyzer (Jaeger-Mijnhardt, Bunnik, The Netherlands). Heart rate was recorded continuously by means of a Polar heart rate monitor (Polar Electro, Kempele, Finland). Before each test, the gas meter was calibrated with a Hans Rudolph 3-liter syringe (Vacumed, Ventura, CA), and the analyzers were set with room air and a 4% carbon dioxide-96% nitrogen gas mixture. W_{peak} values were used to set the work rates in the experimental trials to correspond to 63% of each subject's W_{peak} (70% $\dot{V}_{O_2\ peak}$).

The subjects were further instructed to complete a 3-day dietary record consisting of 2 week days and 1 weekend day. These dietary records were analyzed with the Food Finder 3 program (Medtech, Medical Research Council, Tygerberg, South Africa) to determine the subjects' self-reported energy intake and macronutrient consumption. This dietary information was used as a guideline to devise the two experimental diets. To aid adherence to the diets, subjects were also required to indicate their food preferences.

Table 1. *Subject characteristics*

Characteristic	Mean (SD)	Range
Age, yr	26.0 (3.3)	22.0–32.0
Weight, kg	81.3 (9.6)	74.0–100.0
Height, m	1.80 (0.10)	1.65–1.91
Body fat, %	14.0 (2.8)	8.90–18.1
$\dot{V}_{O_2\ peak}$, ml kg ^{1} min ^{1}	57.8 (5.5)	51.1–67.2
W_{peak}, W	361 (36)	290–419

Values are means (SD) for 8 subjects. $\dot{V}_{O_2\ peak}$, peak oxygen uptake; W_{peak}, peak power output.

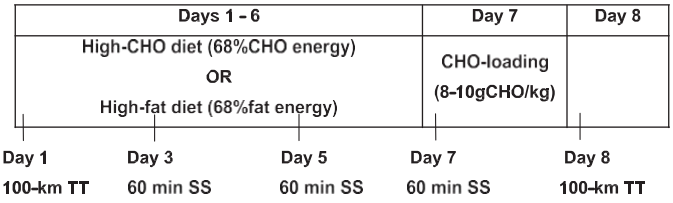

Fig. 1. Summary of diet and testing protocol. CHO, carbohydrate; SS, steady-state cycle at 63% of peak power output (W_{peak}); TT, 100-km time trial.

Study design. Each subject completed two trials in a randomized, single-blind, crossover design with a 2-wk washout period separating each trial. Each trial consisted of an 8-day diet, training, and testing period (Fig. 1). During the trials, subjects reported to the laboratory on *days 1, 3, 5, 7*, and *8* to undertake supervised training and testing.

Dietary manipulations. Subjects were required to ingest either a HFD (68% energy from fat) for 6 days followed by 1 day of CHO loading (90% energy from CHO), or an equal-energy CHO diet (68% energy from CHO) for 6 days followed by 1 day of CHO loading (90% energy from CHO). A registered dietician formulated individualized menus. To control dietary intake, all the meals were prepacked and provided for the subjects together with a diary to record any deviations from the diet. Efforts were made to blind the diets by covertly manipulating the macronutrient compositions of the diets.

Exercise training sessions. On *days 1, 3*, and *5*, subjects reported to the laboratory after a 10- to 12-h fast and completed an exercise training session. On the first day of training (*day 1*), subjects completed a 100-km familiarization time trial on their own bicycles mounted on a Kingcycle trainer (EDS Portaprompt, High Wycombe, UK). The calibration and reliability of the Kingcycle has been described in detail previously (25). The time trial included five 1-km sprint distances after 10, 32, 52, 72, and 99 km, as well as four 4-km sprint distances after 20, 40, 60, and 80 km during which subjects were requested to cycle "as fast as possible." The familiarization time trial also served as a screening trial to see whether the subjects were adequately trained to complete the trial.

On *days 3* and *5*, subjects completed a 60-min steady-state cycle at 70% $\dot{V}_{O_2\ peak}$ on a lode bike, maintaining their cadence at 90 rpm. The steady-state training sessions on *days 3* and *5* were undertaken to ensure consistency in the subjects' training during the trial, as well as monitor their physiological and metabolic responses to the dietary interventions. During the steady-state cycle, heart rate was recorded continuously by means of a Polar heart rate monitor (Polar Electro) and $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values were measured for 4–5 min every 15 min (15, 30, 45, and 60 min), using the online computerized system (Oxycon Alpha Analyzer, Jaeger-Mijnhardt). Rate of perceived exertion (RPE) scores were also recorded at 15-min intervals, using the validated Borg 6–20 RPE scale (9). Printed scale instructions together with a verbal explanation of how the scale works, were given to the subjects before the trial to familiarize them with the operation of the scales.

Before exercise on all 3 days, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values were measured while the subject was seated in a resting position for 15–20 min to determine the resting respiratory exchange ratio (RER), using the online computerized system, as previously described. Heart rate variability (HRV) was also recorded before exercise using a heart rate monitor (Body IQ, Cape Town, South Africa). HRV has been implicated as an indirect measure of autonomic nervous system activation (23). During the HRV test, heart rate measurements were recorded while subjects breathed rhythmically (12 breaths/min) for 5 min of supine lying, followed by 5 min of standing. Power spectrum analysis for low frequency (LF) (indicative of sympathetic activation) and high frequency (HF) (indicative of parasympathetic activation) was per-

formed based on the HRV interval, using MATLAB software (The MathWorks). The natural logarithm of LF and HF power, as well as the ratio of LF power to HF power was calculated from the power spectrum values.

Experimental trials. On *day 7*, subjects reported to the laboratory after a 10- to 12-h overnight fast. HRV, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values were measured at rest as described earlier. Subjects then completed a 60-min steady-state cycle at 70% $\dot{V}_{O_{2\ peak}}$, during which heart rate was recorded continuously and $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ values were measured for 4–5 min every 15 min (15, 30, 45, and 60 min). Blood samples were drawn at rest and at 15-min intervals (15, 30, 45, and 60 min) during the constant-load exercise for the subsequent analysis of plasma glucose, lactate, free fatty acids (FFA) and catecholamine concentrations (see *Blood sampling and analysis*). In addition, electromyography (EMG) amplitude (*EMG measurements*) and RPE were recorded at 15-min intervals.

On *day 8*, CHO-loaded subjects reported to the laboratory after a 10- to 12-h overnight fast. HRV, $\dot{V}_{O_2}$, and $\dot{V}_{CO_2}$ values were measured at rest as described earlier. After a 5-min warm-up, subjects completed a 100-km performance time trial. During the 100-km time trial, a blood sample was drawn at rest and again immediately before the 1-km sprints at 32, 52, 72, and 99 km for the subsequent analysis of plasma glucose, lactate, FFA, and catecholamine concentrations. EMG amplitude was recorded during the midpoint of each 1-km sprint (10.5, 32.5, 52.5, 72.5, and 99.5 km), each 4-km sprint (22, 42, 62, and 82 km), and at three nonsprint distances (5, 55, and 95 km) during the 100-km time trial. Power output and heart rate were measured continuously throughout the trial. RPE was recorded immediately before and after every sprint. The only feedback the subjects received during the 100-km time trial was their elapsed distance. During both trials, subjects ingested a 10% glucose polymer solution at regular intervals (200 ml every 20 min) to maintain plasma glucose concentrations.

Isometric maximal voluntary contraction. Before the exercise trial on *days 7* and *8*, subjects' peak isometric force was assessed on the lower right limb on a Kin-Com isokinetic dynamometer (Chattanooga Group, Chattanooga, TN). The subject's hips and upper body were firmly strapped to the seat. The arm position for each test was standardized with each subject crossing his arms over his chest. All isometric tests were conducted at 60° knee flexion, with the limb being in full extension at 0°. The angle of 60° flexion has been shown to be the angle of maximal isometric force generation (34). The standardized warm-up included two isometric contractions of the knee extensors at 50% followed by two contractions at 85% of each subject's subjective maximum. The isometric test included four maximum voluntary contractions (MVC) of 5 s each separated by 5-s intervals. Subjects were verbally motivated to encourage them to achieve their maximum potential. EMG amplitude of the vastus lateralis was recorded during the MVC isometric force test, and the MVC with the highest mean force was used for subsequent analyses. The purpose of the MVC was to allow measurement of muscle recruitment patterns during the subsequent cycling trials to be expressed as a percentage of the MVC. Normalizing each subject's EMG muscle activity relative to his own maximal activity also excluded confounding variables such as electrode positioning, skin impedance, and differences in percent body fat.

EMG measurements. Before the isometric MVC and the exercise trials, an EMG triode electrode (Triode MIEPO100, Thought Technology, Montreal, Canada) was placed over the belly of each subject's right vastus lateralis to measure muscle recruitment patterns during the isometric MVC, the steady-state cycle on *day 7* and the 100-km time-trial on *day 8*. The electrode positioning was standardized for each subject. The hair at the placement site was shaved off, and the skin was scraped using industrial sandpaper. An alcohol swab was used to remove any oil and dirt from the skin. To minimize interference, the electrode was taped onto the leg with self-adherent wrap and covered with cotton pads.

The triode electrode was attached to the muscle "belly" as described above and connected to a preamplifier. The amplifier was linked via fiber-optic cable to the Flexcomp/DSP EMG apparatus (Thought Technology) and host computer. EMG activity was sampled at 1,984 Hz, a high enough frequency for reliable data collection and quantitative data analyses (19). EMG signals from the electrode were band-pass filtered (20–500 Hz) and amplified using standard differential amplifiers (Thought Technology, common mode rejection ratio 103 dB at 1 kHz, input impedance 1 10^6 M ; adjustable gain up to 1,600). The sampled EMG was passed through a 50-Hz line filter to remove interference from electrical sources to yield raw data. Movement artifact was removed from the raw signals with a high-pass second-order Butterworth filter with a cut off frequency of 15 Hz. The means of the EMG signals were then removed, and the signals were full wave rectified. The signals were smoothed with a linear envelope using a low-pass second-order Butterworth filter with a cutoff frequency of 10 Hz. Filtering procedures were performed using MATLAB software (The MathWorks).

Of the four 5-s isometric maximal voluntary contractions from the right limb, EMG was only sampled from the contraction that yielded the greatest force output. The full 5 s of EMG data were sampled during this contraction. In a similar manner, 5 s of EMG were sampled from the 20 s of EMG data collected during the steady-state cycling trial every 15 min and during the 1-km, 4-km, and nonsprint distances of the 100-km time trial. The filtered EMG data was processed to yield mean amplitude data using specifically developed software (Mullany POO. [2.2] computer program, 2000; Ref. 24). EMG amplitude was calculated using the root mean square method.

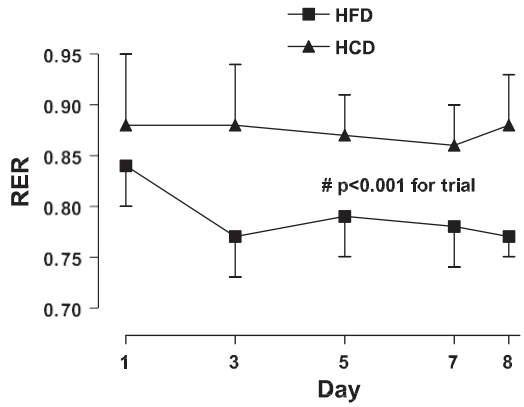

Fig. 2. Resting respiratory exchange ratio (RER) in response to the high-fat diet (HFD) and high-carbohydrate diet (HCD) interventions. Values are means (SD).

Table 2. *Mean dietary intake during dietary treatments*

		CHO			Fat			Protein		
	Energy, cal	g	g/kg	%Energy	g	g/kg	%Energy	g	g/kg	%Energy
HFD	3,560 (246)	150 (14)*	1.85 (0.10)*	16.8 (0.6)*	270 (18)*	3.33 (0.16)*	68.2 (0.6)*	134 (8)	1.65 (0.09)	15.0 (0.5)
HCD	3,550 (206)	602 (32)*	7.48 (0.46)*	67.8 (0.7)*	68 (6)*	0.83 (0.03)*	17.1 (0.6)*	134 (10)	1.66 (0.10)	15.1 (0.4)

Values are means (SD). CHO, carbohydrate; HFD, high-fat diet; HCD, high-carbohydrate diet. *Significant trial effect, (P 0.001).

Table 3. *Mean resting heart rate variability in response to the HFD and HCD*

	Day 1	*Day 3*	*Day 5*	*Day 7*	*Day 8*	*P* Value
LF supine						
HFD	6.65 (0.73)	6.31 (0.75)	6.67 (1.10)	6.12 (1.13)	6.21 (0.73)	0.056 trial
HCD	5.78 (0.59)	6.26 (0.92)	5.87 (0.70)	5.27 (0.92)	6.07 (1.15)	
LF standing						
HFD	6.90 (1.55)	7.07 (0.90)	7.68 (0.84)	7.19 (0.89)	7.14 (0.63)	NS
HCD	7.04 (1.11)	6.92 (1.03)	7.18 (1.06)	6.98 (0.97)	7.03 (0.93)	

Values are means (SD) expressed as the natural logarithm. LF supine, low-frequency supine; LF standing, low-frequency standing. NS, not significant.

Blood sampling and analysis. Venous blood samples (12 ml) were drawn during the steady-state cycle on *day 7* and during the 100-km time trial on *day 8* by inserting a flexible 20-gauge cannula into a forearm antecubital vein and attaching it to a three-way stopcock. The cannula was kept patent by flushing with 1 ml sterile saline after each blood sample. One aliquot (2 ml) was placed into a vacutainer containing potassium oxalate and sodium fluoride for subsequent analysis of glucose and lactate concentrations. Two aliquots (2 3 ml) were placed into vacutainers containing lithium heparin for analysis of plasma epinephrine and norepinephrine concentrations. The remaining aliquot (2 ml) was placed into a vacutainer containing gel and clot activator for determination of serum FFA (nonesterified) concentrations. All samples were kept on ice and then centrifuged at 3,000 rpm at 4°C for 10 min at the end of the trial. The supernatants were stored at 80°C (epinephrine and norepinephrine) and 20°C (glucose, lactate, insulin, and FFAs) for later analysis.

Plasma glucose concentrations were determined using the glucose oxidase method (Glucose analyzer 2, Beckman Instruments, Fullerton, CA). Plasma lactate and serum FFA concentrations were determined by spectrophotometric measurements (model 35, Beckman) using commercial kits (Lactate Pap, Bio-Merieux, Marcy-L'Etiole, France; and FFA Half-micro test, Boehringer, Mannheim, Germany). Plasma catecholamine concentrations were analyzed by high-performance liquid chromatography, according to the method described by Forster and MacDonald (12).

Statistical analysis. Values are presented as means (SD). An ANOVA with repeated measures and the Tukey post hoc analysis were performed using STATISTICA analysis software (version 6, Statsoft, Tulsa, OK). Statistical significance was accepted when *P* 0.05.

RESULTS

Training and dietary control. All subjects followed the experimental diets, ingested the food that was provided during both trials, and achieved the recommended target of fat and CHO intakes. The mean dietary intakes during both trials are presented in Table 2. According to design, there was a significant difference (*P* 0.001) between the CHO and fat contents of the HCD and HFD that were consumed. Although diets were blinded and covertly manipulated, subjects were able to distinguish that the diets were different but were unaware of their composition.

All the subjects attended all the training sessions, but two subjects only completed 45 min of the 60-min steady-state training session on *day 5*, after 4 days of high-fat intake. Although the remaining subjects successfully completed all the training sessions, four subjects experienced difficulties during the steady-state cycle on *day 3* and/or *day 5* on the HFD treatment, complaining of "tired" and "burning" legs or having difficulties in maintaining the training cadence at the defined workload.

Resting variables. The mean fasting RER was significantly lower with the HFD compared with the HCD trial (*P* 0.001). RER decreased over 6 days of the high-fat intake (0.84 0.04 *day 1* to 0.78 0.04 *day 7*) and remained low (0.77 0.02) on *day 8* despite 1 day of CHO loading on *day 7* (Fig. 2). In contrast, RER in the HCD trial did not change significantly over the 8 days.

The mean normalized HRV values for LF, reflecting sympathetic modulation for supine and standing, are presented in Table 3. No significant differences between trials were demonstrated for HF or LF-to-HF ratio (data not shown), but there was a tendency toward a significant trial effect (*P* 0.056) for the LF supine values (Table 3). No significant differences in mean resting RPE or heart rate were found between the two diet treatments (data not shown).

RER, heart rate, and RPE during steady-state training rides. Mean exercising variables, including heart rate, RER, and RPE, measured at 15-min intervals during the 60-min steady-state cycle on *days 3*, *5*, and *7*, are presented in Table 4. Mean exercising RER on *days 3*, *5,* and *7* was significantly lower on the HFD compared with the HCD (*P* 0.05). Conversely, mean exercising heart rate was significantly higher in response to the HFD treatment (*P* 0.05) during the three steady-state rides. There was a tendency (*P* 0.063) for mean effort perception during the three training rides to be higher when ingesting the HFD compared with the HCD.

Experimental steady-state cycle on day 7. Tables 5 and 6 summarize the metabolic responses during the steady-state cycle on *day 7*, after 6 days of high-fat intake. Mean RER was significantly lower (*P* 0.01) on the HFD compared with HCD. Heart rate increased during the exercise bout (*P* 0.05) and was significantly higher for HFD (*P* 0.05) compared

Table 4. *Mean exercising values during the 60-min steady-state cycle on days 3, 5, and 7 in response to the HFD and HCD treatments*

	Day 3	*Day 5*	*Day 7*	*P* Value
RER				
HFD	0.85 (0.04)	0.86 (0.03)	0.87 (0.03)	
HCD	0.93 (0.04)	0.92 (0.01)	0.93 (0.02)	0.05 for trial
Heart rate, beats/min				
HFD	153.0 (5.73)	152.0 (6.34)	151.0 (8.61)	
HCD	149.0 (7.33)	146.0 (7.23)	147.0 (8.19)	0.05 for trial
RPE				
HFD	14.0 (1.17)	13.9 (1.21)	12.7 (0.58)	
HCD	13.2 (0.96)	12.6 (1.09)	12.6 (0.74)	0.063 for trial

Values are means (SD). RER, respiratory exchange ratio; RPE, rating of perceived exertion.

Table 5. *Mean exercising values during the 60-min steady-state cycle on day 7 in response to the HFD and HCD treatments*

	15 min	30 min	45 min	60 min	*P* Value
RER					
HFD	0.89 (0.03)	0.86 (0.03)	0.84 (0.02)	0.85 (0.03)	0.005 for trial
HCD	0.96 (0.03)	0.91 (0.02)	0.91 (0.02)	0.91 (0.02)	0.001 for time
Heart rate, beats/min					
HFD	149 (9)	152 (7)	156 (7)	158 (7)	0.05 for trial
HCD	143 (11)	148 (8)	151 (6)	154 (8)	0.001 for time
RPE					
HFD	12 (2)	13 (1)	14 (1)	14 (2)	
HCD	11 (2)	12 (1)	13 (1)	13 (1)	0.001 for time
Normalized EMG amplitude, %					
HFD	36 (13)	29 (7)	27 (9)	25 (9)	
HCD	37 (8)	32 (1)	35 (13)	32 (8)	0.01 for time

Values are means (SD). EMG, electromyograph.

with HCD. Despite the increase in heart rate after HFD, mean RPE was not different between trials. Similarly, there was no significant difference in normalized EMG amplitude in response to the two dietary interventions. The EMG amplitude did, however, decrease significantly during the exercise bout (P 0.01) during both trials.

Euglycemia was maintained during the steady-state cycle during both trials, and plasma glucose concentrations were not different between the HFD and HCD. An interaction effect was demonstrated for plasma lactate concentrations (P 0.05) in response to the dietary interventions, with the mean plasma lactate response being significantly lower (P 0.01) after HFD compared with HCD. In contrast, plasma FFA concentrations were significantly higher (P 0.001) at rest and during the steady-state cycle after the HFD compared with HCD. Plasma catecholamine concentrations were not different between trials.

Metabolic and performance data during 100-km time trial. Circulating blood levels, obtained immediately before the 1-km sprints at 10, 32, 52, 72, and 99 km during the 100-km time trial, are summarized in Table 7. Plasma glucose concentrations were not significantly different between the HFD-CHO and HCD-CHO and subjects remained euglycemic throughout the 100-km time trial after both diet treatments. Plasma FFA concentrations increased significantly during both trials (P 0.001), but they were not different in response to the two diet interventions. Similarly plasma lactate concentrations increased during both trials (P 0.001) and there was a tendency (P 0.069) for the levels to be higher after the HCD-CHO compared with the HFD-CHO. Plasma catecholamine concentrations also increased significantly during both trials (P 0.001) but were not different between the two dietary treatments.

Overall 100-km time trial performance was not significantly different between trials (P 0.23); however, mean performance time was 3 min 44 s slower on the HFD-CHO compared with the HCD-CHO (Fig. 3). Performance of three of the eight subjects improved on the HFD-CHO, with no order effect observed (P 0.28). Variables recorded during the 4-km sprints are summarized in Table 8. No between-trial differences were demonstrated during the 4-km sprints. Mean power output and sprint time recorded during 4-km sprints decreased significantly over time during both trials (P 0.01). Conversely, RPE recorded immediately after 4-km sprints increased similarly over time (P 0.001) in both trials.

In contrast to the overall and 4-km performance, mean power output recorded during the high-intensity 1-km sprints was significant lower (P 0.05) after the HFD-CHO com- pared with the HCD-CHO treatment (P 0.05 time trial; Fig. 4). Consequently, 1-km sprint times tended to be slower (P 0.07) after the HFD-CHO compared with the HCD-CHO. Mean heart rate was similar for both treatments (Fig. 5*A*). RPE

Table 6. *Mean circulating blood concentrations during the 60-min steady-state cycle on day 7 in response to the two dietary treatments*

	15 min	30 min	45 min	60 min	*P* Value
Plasma glucose, mmol/l					
HFD	3.9 (0.61)	4.3 (0.86)	4.3 (0.70)	4.0 (0.52)	NS
HCD	4.5 (0.78)	4.5 (0.78)	4.4 (0.78)	4.5 (0.91)	
Plasma lactate, mmol/l					
HFD	2.5 (0.94)	2.55 (1.05)	2.56 (0.98)	2.57 (0.90)	0.05 time trial
HCD	4.01 (1.28)	4.26 (1.31)	3.88 (1.28)	3.85 (1.31)	
Serum free fatty acids, mmol/l					
HFD	0.27 (0.09)	0.37 (0.11)	0.41 (0.12)	0.47 (0.12)	0.005 for trial
HCD	0.20 (0.07)	0.24 (0.10)	0.29 (0.12)	0.35 (0.15)	0.001 for time
Plasma epinephrine, nmol/l					
HFD		0.99 (0.22)	1.15 (0.28)	1.27 (0.27)	0.001 for time
HCD		1.02 (0.08)	1.14 (0.22)	1.15 (0.08)	
Plasma norepinephrine, nmol/l					
HFD		8.42 (2.43)	8.81 (2.34)	10.36 (2.48)	0.001 for time
HCD		8.10 (1.20)	8.55 (2.25)	9.48 (1.96)	

Values are means (SD).

Table 7. *Circulating blood concentrations during the 100-km time trial in response to the HFD-CHO and HCD-CHO treatments*

	10 km	32 km	52 km	72 km	99 km	*P* Value
Plasma glucose, mmol/l						
HFD-CHO	4.3 (0.47)	4.4 (0.70)	4.5 (0.66)	4.2 (0.35)	4.3 (0.43)	NS
HCD-CHO	4.4 (1.23)	4.7 (0.75)	4.7 (0.83)	4.4 (0.76)	4.4 (0.70)	
Serum free fatty acids, mmol/l						
HFD-CHO	0.31 (0.09)	0.28 (0.14)	0.35 (0.23)	0.46 (0.29)	0.78 (0.38)	
HCD-CHO	0.28 (0.12)	0.25 (0.12)	0.30 (0.18)	0.38 (0.16)	0.77 (0.35)	0.001time
Plasma lactate, mmol/l						
HFD-CHO	1.18 (0.26)	4.69 (2.26)	4.51 (2.04)	4.04 (1.83)	2.95 (0.98)	0.069 trial
HCD-CHO	1.58 (0.37)	5.35 (3.45)	5.15 (3.33)	5.00 (2.87)	4.23 (2.19)	0.001time
Plasma epinephrine, nmol/l						
HFD-CHO	0.25 (0.04)	0.91 (0.30)	1.10 (0.31)	1.46 (0.65)	2.86 (1.58)	
HCD-CHO	0.21 (0.05)	0.95 (0.38)	1.20 (0.58)	1.58 (0.28)	3.61 (1.49)	0.001time
Plasma norepinephrine, nmol/l						
HFD-CHO	1.68 (0.49)	11.40 (6.13)	12.25 (6.93)	14.01 (7.33)	15.42 (8.48)	0.001time
HCD-CHO	1.90 (0.53)	12.72 (7.99)	12.88 (6.17)	14.56 (4.98)	19.69 (7.47)	

Values are means (SD). HFD-CHO, 6-day high-fat diet 1 day carbohydrate loading; HCD-CHO, 6-day high-carbohydrate diet 1-day carbohydrate loading.

recorded immediately after the 1-km sprints rose progressively over time (*P* 0.01) but was not different between treatments (Fig. 5*B*). Normalized EMG amplitude measured during the 1-km sprints was also similar for both treatments (Fig. 5*C*).

DISCUSSION

In this study, we examined the effects of 6 days of a high-fat intake, followed by 1 day of CHO loading, on substrate utilization, HRV, effort perception, muscle recruitment, and performance during endurance exercise. The study is unique in that it is the first study to investigate the effect of high-fat feeding, followed by CHO loading, on endurance exercise, including high-intensity sprints that simulate actual race situations. It was hypothesized that the potential glycogen-sparing effect of this dietary strategy (3) would be most beneficial for exercise that included high-intensity sprint bouts, where muscle glycogen is the predominant fuel (28). However, in contrast to our hypothesis, the HFD-CHO strategy actually compromised high-intensity 1-km sprint performance (Fig. 4). This is a novel finding and, to our knowledge, has not previously been reported.

The ingestion of a HFD for 6 days resulted in a shift in substrate metabolism toward a greater reliance on fat and a reduction in CHO oxidation. The increase in fat oxidation in the present study persisted despite 1 day of CHO loading on *day 7* as demonstrated by the lower resting RER (0.77 0.02 vs. 0.88 0.05, Fig. 2) and higher circulating FFA (Table 7) during exercise after HFD-CHO compared with HCD-CHO on *day 8*. These findings are consistent with the findings of Burke et al. (3, 4) and Carey et al. (6), who also demonstrated an increase in fat oxidation with short-term high-fat feeding that persisted even after restoration of CHO stores. Burke et al. (3) demonstrated that 1 day of rest and CHO loading was sufficient to restore muscle glycogen levels to above baseline levels in both dietary treatments (470 24 to 554 45 mmol/kg dry wt after HFD-CHO; 470 24 to 608 51 mmol/kg dry wt after HCD-CHO). Although muscle glycogen was not measured in our study, it is assumed that muscle glycogen levels were restored on *day 8* as a similar dietary strategy was used to that of Burke et al. (3) in which muscle glycogen levels were measured directly. The increase in fat oxidation with this dietary regime can therefore not be explained by low glycogen stores (36), and it may be related to changes in insulin sensitivity (13), increased fatty acid uptake into the muscle (5), and changes in skeletal muscle enzyme activities that favor fat oxidation (11, 13). In addition to an increase in fat oxidation, Burke et al. (3) have shown that the ingestion of a HFD-CHO resulted in a significant reduction in muscle glycogen utilization (100 mmol/kg dry wt) during a 120-min cycle at 70% maximal oxygen consumption with the HFD-CHO compared with the HCD-CHO dietary strategy.

Ingestion of a HFD for 6 days was associated with a significant increase in heart rate, as well as a tendency toward a higher effort perception during training on *days 3* and *5*. Six of the eight subjects complained of fatigue and difficulty in maintaining the defined workload during the steady-state cycle, with two subjects failing to complete the 60-min training session. Burke et al. (3) reported similar subject complaints while training on a HFD. The increased effort perception and heart rate may be attributed to low glycogen stores and an

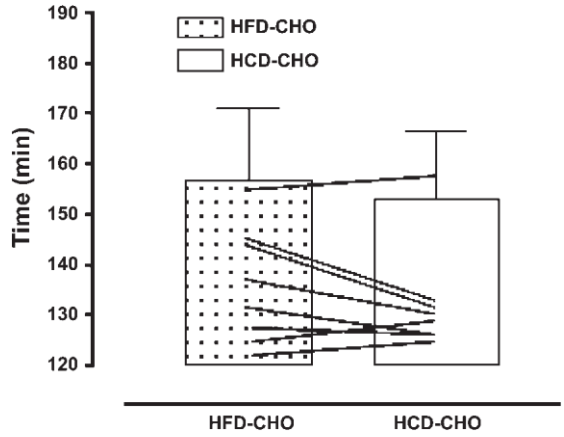

Fig. 3. Overall 100-km time trial performance in response to the 6-day HFD 1-day CHO loading (HFD-CHO) and 6-day HCD diet 1-day CHO loading (HCD-CHO) interventions. Solid lines, individual performance changes. Values are means (SD).

Table 8. *Variables measured during the 4-km sprints in response to the HFD-CHO and HCD-CHO treatments*

	20 km	40 km	60 km	80 km	*P* Value
Power, W					
HFD-CHO	289 (50)	291 (50)	279 (50)	268 (48)	0.01 time
HCD-CHO	308 (56)	308 (61)	305 (62)	295 (55)	
Sprint time, s					
HFD-CHO	336 (26)	338 (26)	340 (24)	347 (29)	0.05 time
HCD-CHO	327 (27)	330 (31)	328 (28)	335 (31)	
Heart rate, beats/min					
HFD-CHO	166 (6)	166 (8)	167 (6)	168 (7)	NS
HCD-CHO	166 (7)	166 (9)	164 (10)	166 (9)	
RPE					
HFD-CHO	16.6 (2.07)	17.6 (1.30)	18.4 (1.60)	18.8 (1.04)	0.001 time
HCD-CHO	15.8 (2.66)	17.3 (2.05)	17.5 (2.14)	18.3 (1.75)	
Normalized EMG amplitude, %					
HFD-CHO	33.8 (12.6)	31.33 (11.0)	31.2 (13.4)	32.1 (11.7)	NS
HCD-CHO	31.3 (10.0)	27.7 (6.2)	31.2 (5.3)	29.4 (8.3)	

Values are means (SD).

increase in sympathetic activation (Tables 3 and 4) (17, 29). This has practical implications for athletes ingesting a low-CHO/high-fat diet, for example the Atkins diet, in terms of their ability to train at high intensities. Moreover, athletes that rely on heart rate to set their training loads may fail to achieve a desired power output and hence training stimulus.

Although the HFD-CHO dietary strategy was associated with an increase in fat oxidation and an apparent sparing of muscle glycogen stores on *day 8*, overall 100-km time trial (156 min 54 s for HFD-CHO vs. 153 min 10 s for HCD-CHO) and 4-km sprint performance times after the two dietary treatments were not significantly different. Similarly, Burke et al. (3) and Carey et al. (6) demonstrated no overall improvements in performance during a 7 kJ/kg time trial (lasting 25 min) after a 2-h submaximal steady-state cycle (3) or a 1-h time trial after 4 h of constant-load exercise (6). However, in both studies, there were individual differences in performance. In the first study, time trial time was 8% faster in five of the seven subjects during the HFD-CHO compared with the HCD-CHO trials (3). Similarly, Carey et al. demonstrated improved performance in five of the seven subjects after the HFD. However, these studies did not simulate race conditions where high-intensity sprint bouts (90% of W_{peak}) are integral to performance. Mean power output during the 25-min time trial in the two studies of Burke et al. (3, 4) after fat adaptation were 281 W (76% of W_{peak}) and 302 W (76% of W_{peak}), respectively, and mean power output during the 1-h time trial of Carey et al. (6) was 312 15 (77.4% of W_{peak}). The present study is the first study that included high-intensity sprints (mean power output during 1-km sprints 90% of W_{peak}) with endurance exercise, simulating race conditions. In contrast to the original hypothesis, we found that HFD-CHO dietary strategy actually compromised high-intensity 1-km sprint power output. Power output during the 1-km high-intensity sprints was even compromised in the three subjects whose overall 100-km time-trial performance was improved on the HFD-CHO dietary strategy.

We postulated that this reduced performance might be related to changes in sympathetic activation associated with high-fat feeding, as we demonstrated an increase in LF power spectrum for HRV, suggestive of increased sympathetic activation after high-fat intake that persisted after 1 day of CHO loading. HRV has previously been shown to be a noninvasive, practical, and reliable measure of sympathetic modulation (14). In addition, heart rate was similar during the 1-km sprints (Fig. 5*A*) despite reduced 1-km power output after the HFD-CHO diet, suggesting increased sympathetic activation during the HFD-CHO trial. Previous research (29) has demonstrated an increase in sympathetic activation during exercise, as measured by plasma norepinephrine levels, with 7 days of high-fat feeding, which was associated with low muscle glycogen stores. However, Helge et al. (17) demonstrated that the increase in sympathetic activation during exercise with high-fat feeding persisted despite the restoration of muscle glycogen stores. An increase in sympathetic activation in response to a high-fat intake, as suggested by findings in the present study, has previously been associated with increased effort perception (17, 27, 33). The present study showed similar RPEs immediately after the 1-km sprints for both trials, despite reduced power output in the HFD-CHO trial, indicative of increased effort perception for less work produced (Fig. 5*B*).

Similarly, EMG amplitude was similar between treatments, and it failed to track the change in power output during the 1-km sprints. This suggests that the decrease in power output during the HFD-CHO trial was associated with a relative

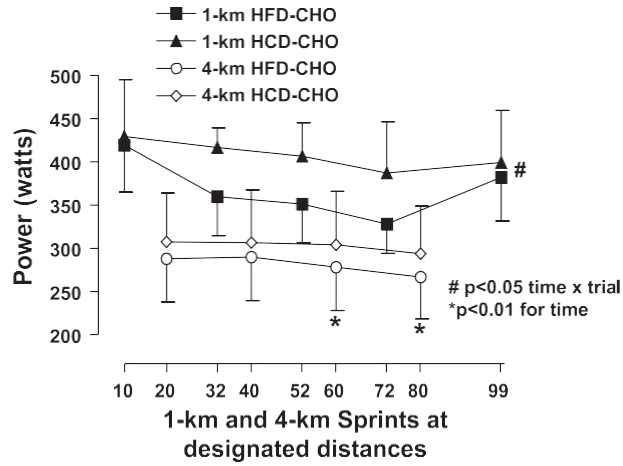

Fig. 4. Power output during the 1-km and 4-km sprints in response to the HFD-CHO and HCD-CHO interventions. Values are means (SD).

increase in muscle recruitment (Fig. 5*C*), indicating that subjects recruited a greater number of motor units for less power produced. In contrast to our findings, St Clair Gibson et al. (31) reported no effect of dietary manipulation on neuromuscular activity during a self-paced 100-km time-trial and showed similar reductions in power output and EMG activity during the 1-km or 4-km sprints between the placebo and CHO-loading exercise trials. It is not obvious how high-fat feeding increased muscle recruitment during the present study, but it may be related to the increased sympathetic activation associated with high-fat intake. It has previously been shown that an increase in sympathetic activation is associated with increases in EMG activity; however, this measure was during static and not dynamic exercise (30). It may also be possible that the increased muscle recruitment was a result of the development of peripheral fatigue due to altered contractile function and/or metabolic substrate perturbations (15).

Ingestion of the HFD-CHO may have compromised the ability to oxidize the available glycogen at a sufficient rate to fuel the high-intensity sprint bouts. The 1-km sprints were performed at an intensity of 90% of W_{peak}, during which muscle glycogen is the predominant fuel source (28). In contrast, power output during the 4-km sprints was performed at a lower intensity ($78-84\%$ of W_{peak}) and was not affected by the high-fat intake. Therefore, the glycogen-sparing effect of the HFD-CHO strategy, which was thought to be beneficial for endurance performance, may in fact compromise high-intensity sprint performance. This may possibly be mediated by changes in pyruvate dehydrogenase (PDH) activity (7, 26). Indeed, studies investigating the effects of a high-fat intake for between 3 days and 3 wk on PDH activity demonstrated a decrease in the active form of PDH, suggesting reduced glycogenolysis and reduced CHO oxidation (7, 26). Furthermore, preliminary data from Stellingwerff et al. (32), using a similar HFD-CHO strategy to the present study, also demonstrated a decrease in mean active PDH activity during steady-state exercise. Further studies, however, are required to examine this hypothesis.

There was an increase in power output during the final 1-km sprint in both the HFD-CHO and HCD-CHO trials, which is indicative of a reserve capacity. This suggests the presence of a pacing strategy (35), even though subjects were verbally encouraged to exercise as hard as possible during each sprint. The EMG amplitude of the HFD-CHO and HCD-CHO trials did not track the increase in power output during the last 1-km sprint. In this trial, muscle recruitment was only measured from the vastus lateralis muscle. However, it is possible that the muscle recruitment of the entire quadriceps femoris was altered as subjects fatigued, and the maintenance or increase in power output during the last high-intensity sprint was the result of motor unit rotation and/or substitution (37) or additional recruitment of non measured synergistic muscles (1, 8).

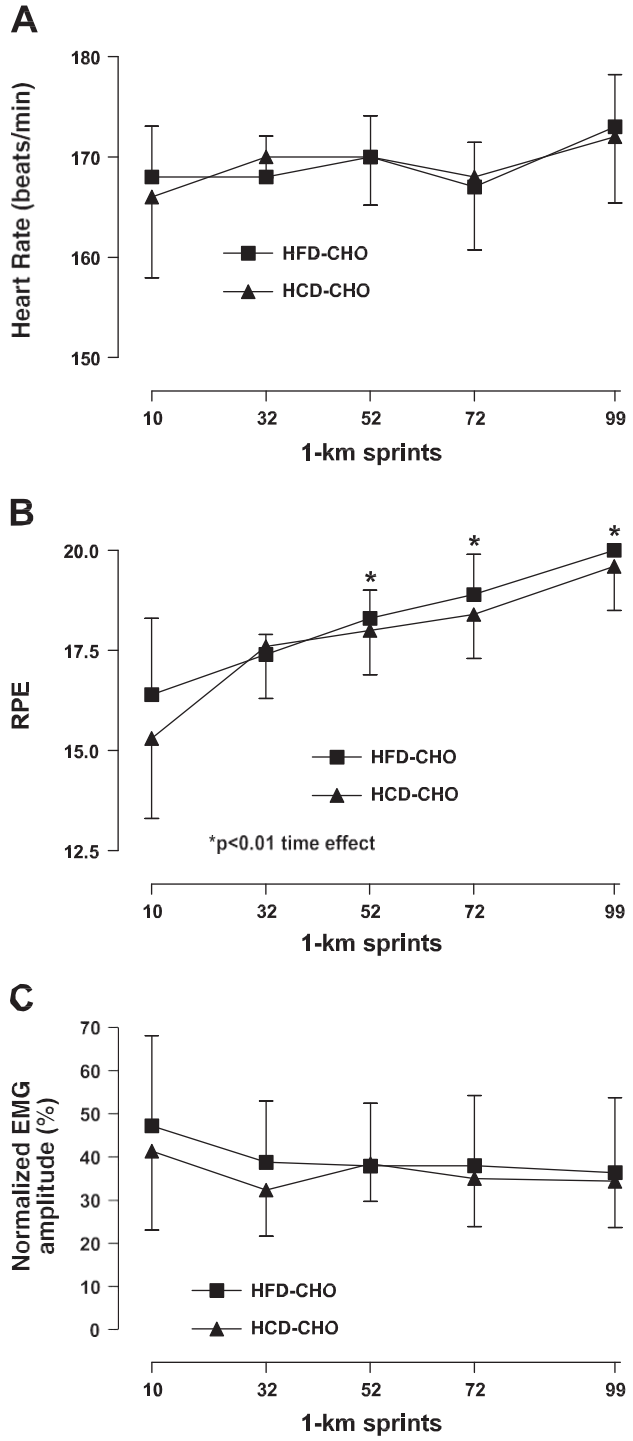

Fig. 5. Heart rate (A), ratings of perceived exertion (RPE; *B*), and normalized EMG amplitude (*C*) during the 1-km sprints in response to the HFD-CHO and HCD-CHO interventions.

In conclusion, ingestion of a HFD for 6 days, followed by 1 day of CHO-loading, increased fat oxidation, but it reduced high-intensity sprint power performance, which was associated with increased muscle recruitment, effort perception, and heart rate. The mechanisms associated with the decrement in performance are not clear, but they could possibly be related to increased sympathetic activation or altered contractile function and/or the inability to oxidize the available CHO during the high intensity sprints. Further research is required to investigate mechanisms associated with high-fat feeding and compromised high-intensity exercise performance.