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Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet

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Abstract. These studies investigated the effects of 2 weeks of either a high-fat (HIGH-FAT: 70% fat, 7% CHO) or a high-carbohydrate (HIGH-CHO: 74% CHO, 12% fat) diet on exercise performance in trained cyclists ($n=5$) during consecutive periods of cycle exercise including a Wingate test of muscle power, cycle exercise to exhaustion at 85% of peak power output [90% maximal oxygen uptake ($\dot{V}O_{2max}$), high-intensity exercise (HIE)] and 50% of peak power output [60% $\dot{V}O_{2max}$, moderate intensity exercise (MIE)]. Exercise time to exhaustion during HIE was not significantly different between trials: nor were the rates of muscle glycogen utilization during HIE different between trials, although starting muscle glycogen content was lower [68.1 (SEM 3.9) vs 120.6 (SEM 3.8) $mmol \cdot kg^{-1}$ wet mass, $P<0.01$] after the HIGH-FAT diet. Despite a lower muscle glycogen content at the onset of MIE [32 (SEM 7) vs 73 (SEM 6) $mmol \cdot kg^{-1}$ wet mass, HIGH-FAT vs HIGH-CHO, $P<0.01$], exercise time to exhaustion during subsequent MIE was significantly longer after the HIGH-FAT diet [79.7 (SEM 7.6) vs 42.5 (SEM 6.8) min, HIGH-FAT vs HIGH-CHO, $P<0.01$]. Enhanced endurance during MIE after the HIGH-FAT diet was associated with a lower respiratory exchange ratio [0.87 (SEM 0.03) vs 0.92 (SEM 0.02), $P<0.05$], and a decreased rate of carbohydrate oxidation [1.41 (SEM 0.70) vs 2.23 (SEM 0.40) g $CHO \cdot min^{-1}$, $P<0.05$]. These results would suggest that 2 weeks of adaptation to a high-fat diet would result in an enhanced resistance to fatigue and a significant sparing of endogenous carbohydrate during low to moderate intensity exercise in a relatively glycogen-depleted state and unimpaired performance during high intensity exercise.

Key words: High-fat diet – Carbohydrate – Fat metabolism – Exercise performance – Fatigue

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Introduction

There is evidence that adaptation to a high carbohydrate diet prior to exercise (Bergstrom et al. 1967; Saltin and Karlsson 1971) and carbohydrate ingestion during exercise (Coggan and Coyle 1991) enhances submaximal endurance performance (of more than 2 h duration) during exercise at a moderately-high intensity (more than 70% maximal oxygen consumption, $\dot{V}O_{2max}$). Moreover, short-term exposure to diets which are very low in carbohydrate has been shown to impair resistance to fatigue during exercise of both high and low intensity (Bergstrom et al. 1967; Christensen and Hansen 1939; Galbo et al. 1979; Maughan and Poole 1981). As a result, endurance athletes are routinely advised to ingest high carbohydrate diets (more than 70% by energy) during training, and to ingest carbohydrate during competition.

In practice, however, it has been found that the habitual diets of endurance-trained athletes suggest that the nutrient composition of these diets is seldom in excess of 55% carbohydrate by energy (Coetzer et al. 1993; van Erp-Baart et al. 1989). Moreover, carbohydrate loading prior to exercise does not result in a sparing of endogenous carbohydrate stores during exercise, but only "spares fat" (Bosch et al. 1993). Similarly, it has been found that it does not spare muscle glycogen stores (Bosch et al. 1994; Coyle et al. 1986) and carbohydrate ingestion during exercise only slows the rate of liver glycogen breakdown (Bosch et al. 1994).

Under euglycaemic conditions, the rate at which ingested carbohydrate can be utilized by the working muscle has appeared to be limited to 1 $g \cdot min^{-1}$ (Hawley et al. 1994). Hence, despite the ingestion or infusion of carbohydrate, it has been shown that fatigue develops as the rate of fat oxidation during prolonged exercise continues to rise with increasing exercise duration to compensate for declining muscle glycogen utilization (Bosch et al. 1993; Coggan and Coyle 1987; Hawley et al. 1994).

A number of attempts have therefore been made to

increase the availability of free fatty acids (FFA) for oxidation during exercise, in order to decrease the rates of muscle glycogen depletion (Costill et al. 1977; Hargreaves et al. 1991; Jenkins et al. 1988; Rennie et al. 1976). However, the procedures involved in artificially increasing plasma FFA concentrations are too invasive to be of any practical value to athletes.

Accordingly, others have examined whether chronic exposure (greater than or equal to 4 weeks) to a high fat diet might increase the capacity of the muscle to oxidize fat and thereby, spare muscle glycogen utilization during exercise (Conlee et al. 1990; Miller et al. 1984; Phinney et al. 1983; Simi et al. 1991). Such studies have shown that adaptation to a high-fat diet in rats (Conlee et al. 1990; Miller et al. 1984; Simi et al. 1991) and dogs (Hammel et al. 1977; Kronfeld et al. 1973) causes a marked improvement in endurance during exercise of moderate-to-high intensity lasting from 60 to 90 min (or approximately 120 min when rats were carbohydrate-fed after fat adaptation, when compared to exercise after adaptation to a high carbohydrate diet only).

Similar studies in humans ingesting a high (more than 60% by energy) fat diet for more than 2 weeks have demonstrated that submaximal endurance performance (less than 70% maximum) is unaffected in exercise lasting between 2.5 to 3 h despite apparently much lower rates of carbohydrate oxidation (Phinney et al. 1983; Pruett 1970). However, the patterns of fuel utilization in the study by Phinney et al. (1983) cannot be reliably determined because the extremely low (less than 2% by energy) carbohydrate content of the diets induced a marked ketosis. Significant rates of ketone body production and oxidation may lower the respiratory exchange ratio (Schutz and Ravussin 1980), thereby, underestimating carbohydrate oxidation, and thus, may preclude the use of gas exchange to calculate the relative rates of fat and carbohydrate oxidation.

Therefore, the aim of the present study was to determine whether adaptation to a high-fat diet, sufficient to alter muscle carbohydrate concentrations and rates of carbohydrate and fat oxidation during exercise, but not sufficient to induce ketosis, influences the patterns of fuel utilization and performance during high- and low-intensity submaximal exercise in endurance-trained human athletes.

Methods

Subjects. Five endurance-trained male cyclists volunteered for the study. All the subjects were cycling between 100 and 120 km each week, and had a mean $\dot{V}O_{2max}$ of 4.2 (SEM 0.3) $l \cdot min^{-1}$. Individual $\dot{V}O_{2max}$ values and other subject characteristics are given in Table 1. Each subject was informed as to the nature of the study and informed, written consent was obtained prior to the trials. All experimental procedures were approved by the Ethics and Research Committee of the Faculty of Medicine of the University of Cape Town Medical School.

Measurement of $\dot{V}O_{2max}$ and peak sustained power output. The $\dot{V}O_{2max}$ and peak sustained power output (PPO) were determined during a progressive exercise test, performed on a Godart electronically braked cycle ergometer (Bilthoven, Holland) modified with toe clips and racing handle bars. Each subject began cycling at an exercise intensity of 3.3 $W \cdot kg^{-1}$ body mass, and this was increased by 50 W after the first 150 s. Thereafter, the exercise intensity was increased by 25 W every 150 s until the subjects felt exhausted. Exhaustion was defined as more than a 10% reduction in pedalling frequency and/or an inability to maintain the same exercise intensity. The PPO was determined according to the procedure of Kuipers et al. (1985) as modified by Hawley and Noakes (1992).

Table 1. Subject characteristics

Subject	Age (years)	Mass (kg)	Fat (%)	Maximal O_2 uptake ($l \cdot min^{-1}$)
PB	20	68.7	12.0	3.69
DS	24	69.5	12.0	3.52
DT	22	74.0	12.0	4.17
CZ	24	78.0	10.0	5.11
GW	21	74.0	13.4	4.54
Mean	22.0	72.8	11.9	4.21
SEM	0.8	1.7	0.5	0.3

$$PPO\,(W) = \text{final exercise intensity completed (W)} + [(t \cdot 150^{-1}) \times 25\ W]$$

where t is the number of seconds completed in the final exercise intensity.

During the PPO estimations, expired gas was sampled continuously for the determination of oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$). The subjects inspired air through a Hans Rudolph 2700 one-way valve (Vacumed, Ventura, Calif.) connected to a Mijnhardt dry gas meter. Expired gas was passed via a condensation coil through a 15-l baffled mixing chamber to Ametek S-3A/I O_2 and CD-3 CO_2 analysers (Thermox Instruments, Pittsburgh, Pa.). Prior to each test, analysers were calibrated using room air and analytical grade gases of known composition (16% O_2, 5% CO_2 and 79% N_2). The gas meter was calibrated with a 3-l syringe. Instrument outputs were processed by an on-line IBM personal computer which calculated inspired ventilation ($\dot{V}_i$, in litres per minute), $\dot{V}O_2$, and $\dot{V}CO_2$ using conventional equations (Conzolazio et al. 1963). Maximal rates of $\dot{V}O_2$ were used to adjust the exercise intensities in the subsequent performance trials described below.

Dietary manipulations. Prior to the performance trials, subjects underwent two randomly-assigned, 2-week periods of either a high fat diet (70% fat by energy, HIGH FAT) or an equal energy, high carbohydrate diet (70% by energy, HIGH CHO), separated by a minimum of 2 weeks of an ad libitum or normal diet. The HIGH FAT consisted of 67.3 (SEM 1.8)% fat, 7.1 (SEM 2.2)% carbohydrate and 25.5 (SEM 2.5)% protein (by energy). The HIGH CHO comprised 73.6 (SEM 5.0)% carbohydrate, and 12.0 (SEM 6.7)% fat, and 13.5 (SEM 1.6)% protein (by energy). To aid adherence to dietary manipulations, the appropriate foods were purchased for the subjects and palatable menus were provided.

During the 2 weeks of dietary manipulation preceding the exercise performance trials, the subjects were asked to maintain regular training, but to refrain from training on the day preceding the performance trials. In general, the dietary manipulation was well-tolerated, although subjects reported individual and day-to-day variation in their ability to maintain the amount of training and its intensity. All performance trials were conducted in the morning, after an overnight fast.

Experimental trials. Upon arrival at the laboratory, the subjects were weighed and skinfold thicknesses were measured for the determination of body composition (Durnin and Wormesley 1974).

Each subject then underwent a test designed to predict maximal velocity (v_{max}) and force of contraction (P_0) of the leg muscles (modified from Nadeau and Brassard 1983). In this test, the subjects completed a series of 5-s exercise periods on a friction-braked, Monark cycle ergometer (Varberg, Sweden) at maximal cadence against randomly ordered loads of 19.6, 29.4, 49.0, 58.8, and 68.8 N. Each 5-s period was followed by a 1-min rest. The v_{max} and P_0 were estimated from the x and y intercepts of the linear regression between force (N) and pedal cadence ($^\circ \cdot s^{-1}$), calculated from the maximal number of pedal revolutions completed during each 5-S exercise interval.

After a 10-min rest, the subjects were then asked to perform a 30-s Wingate test for muscle power on the Monark cycle ergometer (Ayalon et al. 1974). In this test, the subjects pedalled against 0 resistance for a 2-min warm-up, after which they pedalled as fast as possible for 30 s against a load rapidly adjusted to $0.8\,N \cdot kg^{-1}$ body mass. The effects of HIGH FAT and HIGH CHO on maximal power output (highest power output during a 5-s sampling period during the 30 s test) were compared.

After completing the Wingate test, the subjects rested for 30 min. A cannula was inserted into a forearm vein for serial sampling of blood for subsequent analyses of blood glucose and lactate concentrations, plasma glycerol, FFA, and β-hydroxybutyrate concentrations. The subjects then cycled until they felt exhausted at an intensity corresponding to 85% of their previously-determined PPO or approximately 90% of their $\dot{V}O_{2max}$. This exercise period was ended when the subjects' pedal cadence fell by 10%. Throughout this period, $\dot{V}O_2$, $\dot{V}CO_2$, and respiratory exchange ratio (R) were measured continuously and venous blood samples were obtained at 5-min intervals and at exhaustion.

Immediately prior to the high intensity exercise period and at exhaustion, muscle biopsies were obtained from the vastus lateralis muscle of the same leg using the percutaneous needle biopsy method of Bergstrom (1962), as modified by Evans et al. (1982). These samples were rapidly dissected free of connective tissue, frozen in liquid nitrogen and stored at -80° C for later analysis of muscle glycogen content. Blood was sampled via an indwelling cannula from a forearm vein. Patency of the cannula was maintained by slowly infusing saline (sodium chloride, 0.9%).

Following the high-intensity exercise period, the subjects rested for 20 min during which venous blood was sampled every 5 min for subsequent analyses of metabolites as described previously. Thereafter, the subjects cycled to exhaustion at an intensity which corresponded to approximately 50% of their previously determined PPO or 60% of their $\dot{V}O_{2max}$. In this exercise test, venous blood samples were taken at the beginning and at the end of the exercise period, and $\dot{V}O_2$ and $\dot{V}CO_2$ were measured after 20 min and 40 min of exercise.

Analytical procedures. Venous blood samples (5 ml) were divided into three aliquots. From the sample 1 ml was placed in an ice-cold tube containing potassium oxalate and sodium fluoride, centrifuged at 4° C for 15 min and the supernatant was stored frozen for subsequent analysis of plasma glucose concentrations using the glucose oxidase method (Glucose Analyser 2, Beckman Instruments, Fullerton, Calif.). Another 1-ml aliquot was deproteinized with 1-ml ice-cold 0.6 N perchloric acid, and the supernatant was used for the enzymatic determinations of blood lactate (Gutman and Wahlefeld 1974) and β-hydroxybutyrate (Williamson and Mellanby 1974) concentrations. The remaining blood sample was allowed to clot, was centrifuged, and the serum was stored for later analyses of FFA and glycerol concentrations. Serum FFA concentrations were determined using an enzymatic colorimetric assay (Boehringer Mannheim Biochemica, Mannheim, Germany, cat no. 1082 914). Serum glycerol concentrations were enzymatically determined using an adaptation of the methods of Eggstein and Kuhlmann (1974) and Weiland (1974). Muscle glycogen content was measured according to the technique of Passoneau and Lauderdale (1974).

Differences in blood metabolite concentrations, or muscle glycogen content, were compared between the two feeding trials and with time using two-way analyses of variance. An α level of $P<0.05$ was considered to be statistically significant. Where significant F ratios were found, the Bonferroni posthoc analysis was performed to determine which means were significantly different. Changes in endurance time were compared with a paired Student's t-test using two-tailed values of P.

Results

Effects of dietary manipulation on maximal muscle power

There was no effect of nutrient composition of the diet on muscle power. Estimated v_{max}, P_0 during 5-s cycle exercise periods at workloads ranging from 20 to 70 N, and maximal power output during a 30-s Wingate test were similar after 2 weeks of either HIGH FAT or HIGH CHO, under conditions of either dietary fat or dietary carbohydrate adaptation (Table 2).

Effect of dietary manipulation on metabolism and performance during steady-state high intensity exercise

During high-intensity exercise (90% $\dot{V}O_{2max}$, HIE), steady-state $\dot{V}O_2$, R, heart rate, ventilation ($\dot{V}_E$) and mean performance times to exhaustion were also not significantly different in the HIGH-FAT and HIGH-CHO trials (Table 3).

In contrast, the starting muscle glycogen content was significantly lower in the HIGH-FAT trial than in

Table 2. Estimated maximal velocity (v_{max}) and force of contraction (P_0) values and maximal power output measured during the 30-s Wingate test in response to 2 weeks of either high-fat (HIGH-FAT) or high-carbohydrate (HIGH-CHO) feeding in trained cyclists

		v_{max} ($^\circ \cdot s^{-1}$)	P_0 (kp)	Maximal power output (W)
HIGH-FAT	mean	1241	16.7	862
($n=5$)	SEM	108	1.6	94
HIGH-CHO	mean	1216	16.7	804
($n=5$)	SEM	88	1.5	65

Table 3. Effects of dietary manipulation on submaximal heart rate, oxygen uptake, minute ventilation, and performance time to exhaustion during cycling exercise at 85% peak power output

	HIGH-FAT ($n=5$)		HIGH-CHO ($n=5$)	
	mean	SEM	mean	SEM
Time to exhaustion (min)	8.3	2.3	12.5	3.8
Heart rate (beats·min^{-1})	186	3	181	2
Oxygen uptake (l·min^{-1})	3.62	0.27	3.63	0.39
Ventilation (l·min^{-1})	120.9	5.9	122.7	6.3
Respiratory exchange ratio	1.07	0.04	1.15	0.03

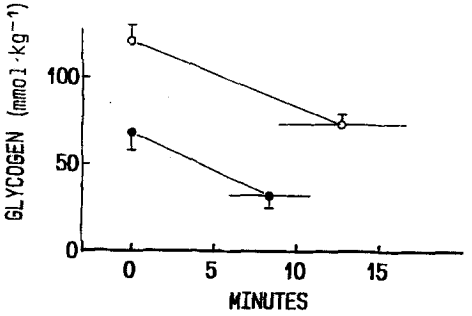

Fig. 1. Muscle glycogen content before and after cycling exercise at 85% of peak power output following 2 weeks of dietary manipulation, ingesting a HIGH-FAT (●) or HIGH-CHO (○) diet. Initial glycogen content was significantly lower after 2 weeks of a HIGH-FAT diet ($P<0.01$) when compared to 2 weeks of adaptation to a HIGH-CHO diet, although the rate of muscle glycogen depletion with exercise was not different among trials

the HIGH-CHO trial [68.1 (SEM 3.9) vs 120.6 (SEM 3.8) mmol·kg^{-1} wet mass, $P<0.01$, Fig. 1], and remained lower at exhaustion. At exhaustion, in the HIGH-CHO trial, muscle glycogen content was the same as the starting muscle glycogen content in the HIGH-FAT trial (Fig. 1). The overall rates of muscle glycogen utilization during HIE in the HIGH CHO and HIGH FAT trials were similar [4.9 (SEM 1.1) vs 5.3 (SEM 0.9) mmol·kg wet mass^{-1}·min^{-1}, respectively].

Effects of dietary manipulation on metabolism and endurance during subsequent moderate intensity exercise

Despite the much lower starting leg muscle glycogen content in the subjects on the HIGH-FAT trial than in the subjects on the HIGH-CHO diet (Fig. 1), the mean exercise time to exhaustion during moderate intensity exercise (MIE) was nearly twofold greater after HIGH-FAT than after HIGH-CHO [79.7 (SEM 7.6) min vs 42.5 (SEM 6.8) min, $P<0.01$, Table 4]. This improvement in endurance was associated with a lower $\dot{V}_i$ and R, as well as lower calculated rates of carbohydrate oxidation after HIGH-FAT. While approximately 2.23 (SEM 0.14) g of carbohydrate were oxidized each minute during steady-state MIE during the HIGH-CHO trial, only 1.41 (SEM 0.25) g of carbohydrate were oxidized per minute during the HIGH-FAT trial.

Table 4. Effects of dietary manipulation on submaximal heart rate, oxygen uptake, minute ventilation, substrate utilization, respiratory exchange ratio and performance time to exhaustion during moderate-intensity cycling at 50% of peak power output

	HIGH-FAT ($n=5$)		HIGH-CHO ($n=5$)	
	mean	SEM	mean	SEM
Time to exhaustion (min)	79.7	7.6	42.5**	6.8
Heart rate (beats·min^{-1})	142	7	143	8
Oxygen uptake (l·min^{-1})	2.28	0.06	2.38	0.05
Ventilation (l·min^{-1})	50.4	1.5	59.2*	0.8
CHO oxidation (g·min^{-1})	1.41	0.70	2.23	0.40
Fat oxidation (g·min^{-1})	0.60	0.12	0.32	0.07
Respiratory exchange ratio	0.87	0.03	0.92*	0.02

* $P<0.05$, ** $P<0.01$

Blood metabolite concentrations during the successive periods of HIE and MIE

Plasma glucose concentrations during the HIGH-FAT and HIGH-CHO trials were not significantly different. At no time during either the HIE or MIE periods did the mean plasma glucose concentration fall below 3.9 mmol·l^{-1} (Fig. 2).

There were also no differences in the change in blood lactate concentration during HIE and MIE between the HIGH-FAT and HIGH-CHO trials. Blood lactate concentrations in both HIE trials increased to over 10 mmol·l^{-1} at exhaustion (Fig. 2). However, the rate of reduction in blood lactate concentrations during the 20-min rest following HIE was greater after HIGH-FAT than after HIGH-CHO (Fig. 2, $P<0.01$).

Serum FFA and glycerol concentrations during successive steady-state HIE and MIE to exhaustion are given in Fig. 3. As expected, initial serum FFA concentrations were significantly higher after HIGH-FAT than after HIGH-CHO ($P<0.01$). Serum FFA concentrations immediately post-HIE were also greater in the

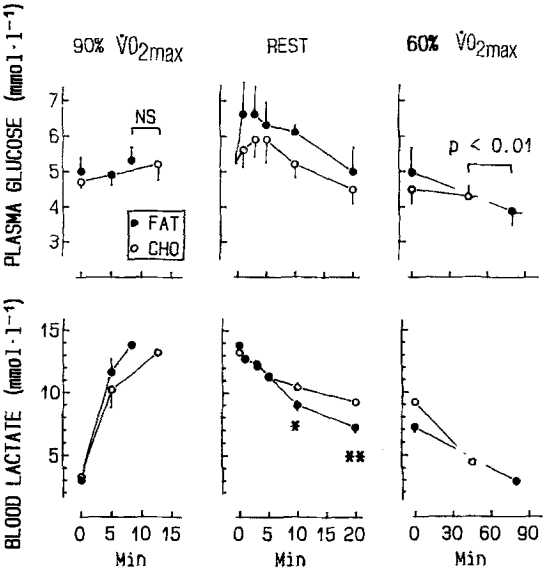

Fig. 2. Changes in plasma glucose concentrations during high intensity and moderate intensity exercise were not significantly different between the HIGH-FAT trial and HIGH-CHO trial. Blood lactate concentrations rose to over 10 mmol·l^{-1} at exhaustion following high-intensity exercise, and were significantly lower during the 20-min rest which separated the high-intensity exercise from the moderate-intensity exercise ($P<0.01$). $\dot{V}O_{2max}$, maximal oxygen uptake

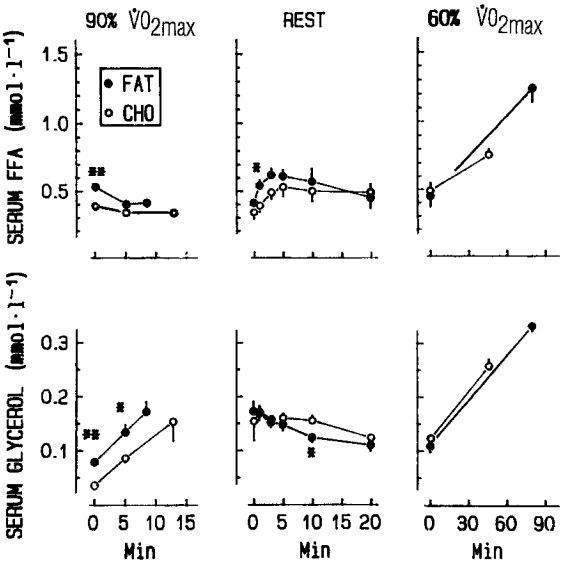

Fig. 3. Serum free fatty acid (*FFA*) concentrations were higher after tests of muscle power and prior to the high intensity exercise period in the HIGH-FAT trial compared to the HIGH-CHO trial ($P<0.01$). In addition, *FFA* concentrations remained higher in the postexercise recovery period when subjects were fat-adapted ($P<0.05$). Similarly, serum glycerol concentrations were higher during high-intensity exercise during the HIGH-FAT trial, than during the HIGH-CHO trial ($P<0.01$) and lower in recovery when the subjects were fat-adapted ($P<0.05$). $\dot{V}O_{2max}$, maximal oxygen uptake

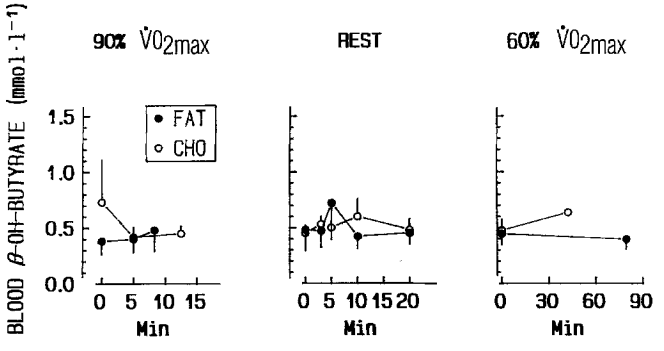

Fig. 4. Blood β-hydroxybutyrate (β-OH-butyrate) concentrations were no different between trials and did not increase significantly with exercise or in the postexercise recovery period. $\dot{V}O_{2max}$, maximal oxygen uptake

subjects during the HIGH-FAT trial ($P<0.05$). However, there were no differences between trials in serum FFA concentrations during subsequent MIE.

Serum glycerol concentrations were higher throughout HIE in the HIGH-FAT trial, than in the HIGH-CHO trial ($P<0.01$, Fig. 3). However, the rates of serum glycerol accumulation in the two HIE trials were not different. Serum glycerol concentrations also rose similary during MIE in both the HIGH-FAT and HIGH-CHO trials. The only difference between trials in serum glycerol concentrations was during the recovery period following HIE. Serum glycerol concentrations were lower in the HIGH-FAT trial than the HIGH-CHO at 10 min postexercise ($P<0.05$).

Blood β-hydroxybutyrate concentrations between trials and with time were also similar (Fig. 4). The subjects did not become ketotic in either trial.

Discussion

The first important finding of this study was that the exercise performance of the subjects adapted to 2 weeks of a high-fat diet was not significantly impaired during supra-maximal and high intensity exercise (Wingate test and steady-state exercise at greater than 90% $\dot{V}O_{2max}$, Tables 2, 3). Secondly, endurance was significantly enhanced during subsequent prolonged exercise at approximately 60% $\dot{V}O_{2max}$ (Table 4). This improvement in endurance occurred, despite a mean starting muscle glycogen content which was two-fold lower than in the carbohydrate-adapted trial (Fig. 1).

Hence, chronic adaptation to a high-fat, low-carbohydrate diet appears to be fundamentally different to an acute lowering of body glycogen stores which has been found to impair performance during exercise of both high and low intensity (Bergstrom et al. 1967; Christensen and Hansen 1939; Galbo et al. 1979; Maughan and Poole 1981). This potential to adapt to a high-fat, low-carbohydrate diet is consistent with studies that have shown that rats adapted to high-fat diets over 4 to 5 weeks had increased endurance performance during steady-state submaximal exercise lasting approximately 1 h (Miller et al. 1984; Simi et al. 1991), or more than 2 h [after carbohydrate loading following fat adaptation (Conlee et al. 1990].

It has been suggested that possible mechanisms which may be responsible for the attenuation of carbohydrate oxidation following chronic ingestion of a high-fat diet, and the associated improvement in endurance performance may include an increased storage of triglyceride in the muscle (Conlee et al. 1990), an increased activity of carnitine-palmityl transferase (Fisher et al. 1983), and an increased activity of 3-hydroxacyl-coenzyme A dehydrogenase, relative to an increase in citrate synthase activity in the skeletal muscle mitochondria (Miller et al. 1984; Simi et al. 1991). These adaptations to a chronic exposure to high-fat or low CHO feeding may „retool" the working muscle mitochondria and increase their capacity for fat oxidation. Moreover, chronic exposure to a high-fat, low carbohydrate diet has been shown to decrease muscle hexokinase activity (Fisher et al. 1983), to reduce glucose uptake by the muscle and to increase tissue insulin resistance (Beck-Nielsen et al. 1978), all of which would be expected to attenuate the rate of endogenous carbohydrate oxidation.

The improvements in endurance performance associated with dietary fat adaptation in the present study contrast with the finding of Phinney et al. (1983). These investigators have shown that exercise performance at 64% $\dot{V}O_{2max}$, lasting between 2 and 2.5 h, was similar in both fat-adapted and carbohydrate-adapted conditions. Differences between our studies and those

of Phinney et al. (1983) may be attributed, in part, to the HIE that preceded the MIE in this study. The mean starting muscle glycogen content after prior HIE in the fat-adapted state in the present study was lower than that found even at the end of MIE in the study by Phinney et al. (1983). Thus, dietary fat adaptation may be more likely to have an effect when body glycogen stores are low.

Another difference between our studies and those of Phinney et al. (1983) is that their 5-week fat adaptation diet was ketogenic, whereas our 2-week fat adaptation diet did not raise circulating β-hydroxybutyrate concentrations (Fig. 4). Blood β-hyroxybutyrate concentrations were threefold higher in the fat-adapted condition in the study by Phinney et al. (1983) than in the present study.

A third difference between our studies and those of Phinney et al. (1983) is in the rates of CHO oxidation during prolonged, moderate exercise. Even after dietary fat adaptation, rates of CHO oxidation in the present study were still over fivefold higher than in subjects exposed to a ketogenic, high-fat diet for a period of 5 weeks (1.41 g $CHO \cdot min^{-1}$ vs 0.25 g $CHO \cdot min^{-1}$; Phinney et al. 1983). This may have been due, in part, to HIE which preceded MIE. At exhaustion after HIE, mean blood lactate concentrations exceeded 14 $mmol \cdot l^{-1}$, and were still greater than 5 $mmol \cdot l^{-1}$ just prior to starting MIE. Moreover, the blood lactate concentrations during the 20 min rest between HIE and MIE was significantly lower during the HIGH-FAT trial than in the HIGH-CHO trial. Thus, the differences in the rates of lactate disappearance would suggest that in a relatively glycogen-depleted state, lactate may have been oxidized and thus, contributed to the overall rate of carbohydrate oxidation under the HIGH-FAT conditions.

However, it is also possible that the overall rates of carbohydrate oxidation were underestimated in the study by Phinney et al. (1983). The respiratory quotient associated with both ketogenesis without concomitant oxidation and gluconeogenesis from amino acids with the retention of glucose have been stated to be 0 and 0.36, respectively by Schutz and Ravussin (1980). Significant rates of ketogenesis, ketone body oxidation and gluconeogenesis may explain why the calculated rate of blood glucose oxidation after fat adaptation, based on ^{13}C-glucose enrichment of blood and breath CO_2, of 5.1 mg $CHO \cdot kg^{-1} \cdot min^{-1}$ exceeded the total carbohydrate oxidation (0.36 vs 0.25 g $CHO \cdot min^{-1}$; Phinney et al. 1983) estimated from gas exchange data.

Phinney et al. (1983) have suggested that the extreme conservation of carbohydrate oxidation during exercise would limit the intensity of exercise which could be performed by the subjects. This suggestion is not supported by the results of the present study, in which HIE performance was not significantly different in the carbohydrate versus the fat-adapted state. In the present study, blood glucose concentrations were maintained throughout HIE and MIE, subjects were not ketotic, and R indicated that carbohydrate oxidation accounted for over 50% of the overall substrate oxidized during MIE (Fig. 2).

One of the limitations in the present study was HIGH FAT was also relatively higher in protein content, when compared to the equal energy HIGH CHO. The de-amination of excess protein may have increased the availability of carbohydrate from the diet, and therefore, would have diluted the effect of the HIGH FAT, apparently low carbohydrate diet.

Thus, the results of the present study would suggest that 2 weeks of adaptation to a high-fat diet is sufficient to alter endogenous carbohydrate stores and relative rates of substrate oxidation, in the absence of marked ketosis. Submaximal exercise performance is enhanced under these conditions, especially when preceded by HIE, which partially depletes muscle glycogen stores. These results do not suggest that fatigue is dissociated from muscle glycogen depletion, but rather, that fatigue in the glycogen-depleted state may be delayed in subjects with mitochondrial adaptations favouring an increased capacity for fat oxidation. Further work, however, is required to see if such adaptations improve endurance under conditions of more competitive, higher intensity (more than 70% of maximum), prolonged exercise, without the complications of prior exercise, or in ultra-endurance sport, in which both liver glycogen and muscle glycogen stores may become depleted.

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