Endurance in high-fat-fed rats: effects of carbohydrate content and fatty acid profile

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Recent studies have provided a conflicting picture when the effect of dietary fat on endurance performance was investigated in humans. Findings in six studies include increased endurance performance within 1- to 2-wk adaptation (17, 25), maintenance of endurance performance after 4-wk adaptation (11, 27), and decreased endurance performance after both 2- and 7-wk adaptation to a fat-rich compared with a carbohydrate-rich diet (10, 28). In contrast to this varied response, studies in rats have demonstrated a more uniform, distinct positive effect of a fat-rich diet on endurance performance (18, 24, 32). A major difference between the studies in rats and humans is the content of carbohydrate supplied in the fat-rich diets. In most human studies, the fat content is within a range of 45–70% total energy content in the diet (E%) fat, which is an achievable fat intake for a free-living person when the effect of dietary fat on endurance performance is considered. In contrast, most animal studies have used fat-rich diets in which the fat content was ~75–85 E% and the carbohydrate content was <1 E% (18, 24, 32). Thus it is difficult to evaluate whether the observed positive effect of a fat-rich diet on endurance in rats is due to the increased supply of dietary fat or is in fact also caused by the almost total absence of dietary carbohydrates. It is therefore of interest to study whether endurance performance in rats is enhanced after adaptation to a diet rich in fat containing 15 E% carbohydrate, similar to that given in human studies.

Another difference between animal and human studies is the fatty acid profile of the fat-rich diets; most studies of high-fat-fed rats have used a saturated-fat diet containing mainly edible tallow or lard, whereas studies in humans have used fat-rich diets containing a mixed fatty acid profile. It might be that the fatty acid profile of the diet influences endurance performance because studies have shown that different types of fatty acids are oxidized differently. Leyton et al. (19) demonstrated in rats that unsaturated fatty acids were preferentially oxidized compared with saturated fatty acids. Furthermore, Jones et al. (13) and Shimomura et al. (31) found that, at rest, the respiratory exchange ratio (RER) was lower after adaptation to a diet containing mainly unsaturated fatty acids compared with a diet containing mainly saturated fatty acids in humans and rats, respectively. Thus if the dietary fatty acid profile does influence both the rate and type of the fatty acids that are oxidized, then it is possible that a high-fat diet containing monounsaturated or polyunsaturated fatty acids could potentially lead to a sparing of muscle and liver glycogen compared with a high-saturated-fat diet and thus have a positive bearing on endurance performance.

The purpose of this study was therefore to compare the effects of two high-fat diets containing a high proportion either of saturated or monounsaturated fatty acids and 15% of calories as carbohydrate vs. a carbohydrate-rich diet on endurance performance and substrate utilization after a 4-wk adaptation to diet and/or training.

**METHODS**

Experimental design. The experiment was approved by the University of Wollongong Animal Experimentation Ethics Committee. Rats were randomly allocated to one of three dietary groups and consumed either a high-saturated-fat (Sat) diet, a high-monounsaturated-fat (Mono) diet, or a high-carbohydrate (CHO) diet. Each dietary group was randomly divided into two subgroups, one of which was trained and the other remaining sedentary. After 4 wk, rats from each...
group were either killed or subjected to an endurance test until exhaustion and then killed.

Animal care. Ninety-nine male Wistar rats (initial wt 240–270 g) were acquired from the Animal Resource Center (Perth, Australia) and housed three per cage at 21 ± 2°C (12:12-h light-dark cycle, 6 PM to 6 AM) for 1 wk before random allocation into three dietary groups. Each group was fed one of three diets for 4 wk. The diets were a high-carbohydrate diet (10 E% fat, 20 E% protein, 70 E% carbohydrate), a high-fat diet rich in saturated fatty acids (65 E% fat, 20 E% protein, 15 E% carbohydrate), and a high-fat diet rich in monounsaturated fatty acids (65 E% fat, 20 E% protein, 15 E% carbohydrate) (Table 1). Fatty acids in the diets were extracted, derivatized, and analyzed on a gas chromatograph as described (26), and selected dietary fatty acids are listed in Table 1. Rats were given free access to food and water. Body weight was recorded weekly throughout the experiment.

Training program. Each dietary group was randomly divided into two subgroups, one of which was trained for 4 wk (n = 19) and the other remaining sedentary (n = 14). Rats were trained in the hours before the dark cycle 6 days/wk on a custom-built, nine-lane motorized treadmill equipped with a rear electric shock grid. Training was initiated at 15 min at 20 m/min on a 0° incline, and, in the course of 2 wk, speed, incline, and duration were progressively increased such that the rats were running for 60 min at 28 m/min on a 5° incline at the end of the second week. Over the third week the incline was increased in two steps to 10°, and the final load, 60 min at 28 m/min on a 10° incline, was maintained until the experiment was terminated. To avoid possible effects of routine handling, sedentary rats were habituated to the treadmill 3 days/wk for 10 min at 0° incline and at the same speed as the trained rats. This training and handling program is similar to those used in other studies (3, 22). A total of seven animals, all trained, had to be excluded from the study because they missed >5% of the total training time, primarily because of injured feet and toenails.

Measurement of RER. In the week before the endurance testing, RER was measured for a randomized subsample (n = 6) of each group. The rats were fasted for 4–5 h and then ran at 28 m/min on a 10° incline. This load was similar to that in the endurance test. After 3–4 min of adaptation, exercise was initiated and oxygen consumption and carbon dioxide production were measured for at least 12 min. Rats ran on a custom-built, single-lane treadmill equipped with a rectangular perspex box positioned on the treadmill belt. Air was drawn from the front end at a constant rate (2.5 l/min) and passed through a drierite and silica column to absorb water. Flow was measured on a mass-flow controller (Teddyne Hastings). The fractions of oxygen and carbon dioxide were measured on an oxygen analyzer (S3A/11, Metek, Pittsburg, PA) and an infrared Beckmann LB2 analyzer system (Beckman). The flowmeter was controlled and calibrated by using a Singer DTM Flowmeter (American Meter Division), and the oxygen and carbon dioxide analyzers were regularly calibrated with gas samples of known composition, having an accuracy of ±0.2% and ±0.08% for O2 and CO2, respectively.

Exercise and tissue-sampling procedures. All rats were either trained or handled on the day before the endurance test and fasted for 5–6 h before the endurance test. Rats were randomly selected to be anesthetized either before the endurance test ("rested") or after the endurance test ("exhausted"). To minimize effects of diurnal variation, rested rats were anesthetized between 1230 and 1345. The endurance test was performed between 1300 and 1700 on a nine-lane treadmill at 28 m/min on a 10° incline. Rats were run until they could no longer maintain the pace of the treadmill. Exhaustion was defined as the inability for a rat to right itself when placed on its back and/or side, as described in previous studies (18, 32). If rats were not exhausted, exercise was continued until exhaustion occurred. To avoid bias, rat identification was concealed to the investigator assessing the exhaustion criteria in all rats.

Rested and exhausted rats were immediately anesthetized by intraperitoneal injection of Nembutal (pentobarbital sodium; 60 mg/100 g body wt). Red and white quadriceps were removed, frozen, and quickly stored in liquid nitrogen. The abdomen was then opened, and a piece of liver was excised and frozen. Blood from the abdominal aorta was drawn into a syringe and separated into two fractions; 1 ml was added to 2 ml 10% perchloric acid, spun at 3,000 rpm for 4 min, and the supernatant was stored for further analysis, and serum from the remainder was stored for later analysis. All tissue and blood were stored at −80°C until analyzed.

Biochemical analysis. Glucose and lactate concentrations in plasma were determined by an automated membrane-bound enzymatic assay on a 23A6 Glucose Analyzer and a 2700 Select Biochem Analyzer (Yellow Springs Instruments, Yellow Springs, OH), respectively. Free fatty acid (FFA) concentration in serum was analyzed with a commercially available enzymatic colorimetric method (NEFA C, Wako, Richmond, VA). Glycogen in muscle and liver was determined as glucose residues after hydrolysis in 1 M HCl at 100°C for 2 h (21). A small piece of muscle was freeze-dried and subsequently dissected free of all visible connective tissue, fat, and blood under a stereomicroscope. One portion was used to measure muscle triacylglycerol content as described by Kiens and Richter (14), and the other portion was used to determine muscle citrate synthase (CS) activity and β-hydroxyacyl-CoA dehydrogenase activity (HAD) fluorometri-

Table 1. Experimental diet composition

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>CHO</th>
<th>Mono</th>
<th>Sat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edible tallow</td>
<td>0</td>
<td>0</td>
<td>179</td>
</tr>
<tr>
<td>Coconut oil</td>
<td>0</td>
<td>0</td>
<td>179</td>
</tr>
<tr>
<td>Canola oil</td>
<td>42</td>
<td>400</td>
<td>42</td>
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<tr>
<td>Σ Fat, E%</td>
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<td>65</td>
<td>65</td>
</tr>
<tr>
<td>Σ Saturated FA*</td>
<td>20.4</td>
<td>12.7</td>
<td>42.9</td>
</tr>
<tr>
<td>Σ Monounsaturated FA*</td>
<td>26.2</td>
<td>48.4</td>
<td>29.2</td>
</tr>
<tr>
<td>Σ n-6 FA*</td>
<td>45.4</td>
<td>27.5</td>
<td>20.4</td>
</tr>
<tr>
<td>Σ n-3 FA*</td>
<td>4.5</td>
<td>7.7</td>
<td>3.6</td>
</tr>
<tr>
<td>Casein, g</td>
<td>124</td>
<td>208</td>
<td>208</td>
</tr>
<tr>
<td>Σ Protein, E%</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Sucrose, g</td>
<td>214</td>
<td>67</td>
<td>67</td>
</tr>
<tr>
<td>Starch, g</td>
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<td>134</td>
<td>134</td>
</tr>
<tr>
<td>Bran, g</td>
<td>51</td>
<td>51</td>
<td>51</td>
</tr>
<tr>
<td>Σ Carbohydrate, E%</td>
<td>70</td>
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<td>15</td>
</tr>
<tr>
<td>Mineral mix, t g</td>
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</tr>
<tr>
<td>Vitamin mix, t g</td>
<td>67</td>
<td>67</td>
<td>67</td>
</tr>
</tbody>
</table>

CHO, carbohydrate-rich; Mono, monounsaturated fat; Sat, saturated fat; FA, fatty acids; E%, % total energy content in the diet. The ingredient values are g/kg diet. FA composition values are % total sum of FA. Each diet was supplemented with 57 g of gelatin and 3 g of methionine/kg diet. FA extracted and analyzed on a gas chromatograph as described (26). †Supplied (per kg of vitamin mix): 1.8 g vitamin A acetate (500,000 IU/g); 0.125 g vitamin D concentrate (350,000 IU/g); 22 g α-tocopherol (250 IU/g); 45 g ascorbic acid; 5 g inositol; 75 g choline chloride; 2.25 g menadione; 5.0 g p-amino benzoic acid; 4.25 g niacin; 1.0 g riboflavin; 3.5 g pyridoxine HCl; 1.0 g thiamine HCl; 3.0 g d-calcium pantothenate; 0.020 g biotin; 0.09 g folic acid; and 0.0035 g vitamin B12. ‡Supplied (per kg of mineral mix): 30.5 g MgSO4 ·7H2O; 65.2 g NaCl; 105.7 g KCl; 200.2 g KH2PO4; 38.8 g MgCO3; 3H2O; 40.0 g FeC2H2O2 ·H2O; 512.4 g CaCO3; 0.8 g KI; 0.9 g NaF; 1.4 g CuSO4 ·5H2O; 0.4 g MnSO4; and 0.05 g Co(NO3)2 ·6H2O.
RESULTS

In this study, diet composition did not have an effect on exercise time to exhaustion in either sedentary or trained rats (Fig. 1). In the sedentary rats, mean run time was 50 ± 3 min (range 30–83 min), whereas in trained rats average run time was 153 ± 8 min (range 97–237 min), which was significantly longer (206%) compared with the sedentary rats. Because running performance in rats is significantly correlated with body mass (7, 33), the statistical evaluation of the running performance included body mass as a covariate to eliminate possible bias of body mass on the training effect. Before the experiment, body weight was identical in the six groups, averaging 264 ± 3 g. After 4 wk, body weight was significantly higher in the sedentary rats (362 ± 6, 369 ± 7, and 386 ± 6 g) than in the trained rats (339 ± 6, 339 ± 4, and 338 ± 7 g) with CHO, Mono, and Sat diets, respectively. However, in contrast to training, diet composition did not affect body weight in either sedentary or trained rats. Under the housing conditions that were applied in this study, food intake could not be estimated precisely, but body weight increased similarly with the three different diets in, respectively, sedentary and trained groups, suggesting that adequate amounts of the diet were available to meet energy requirements.

RER measured during the initial 12 min of exercise was significantly affected by diet composition but not training (Fig. 2). Rats that were fed the CHO diet had a significantly higher RER than rats fed the fat-rich diets. Furthermore, there was also a significantly lower RER in rats fed the Mono diet compared with rats fed the Sat diet (P < 0.05). Muscle glycogen content was significantly higher in the trained than the sedentary rats before exercise, but no difference was present in the exhausted rats. There was no significant effect of diet composition on glycogen levels in either red or white quadriceps muscle (Table 2). In the rested rats, liver glycogen content was significantly higher in the CHO-fed rats than in the fat-fed rats, whereas in the exhausted rats there was no effect of diet composition. In the trained rats, liver glycogen was higher in the trained than the sedentary rats (P < 0.05, Table 2). Neither diet composition nor training affected muscle triacylglycerol concentration, although in both rested (P = 0.08) and exhausted rats (P = 0.09) the P value for a diet effect approaches the level of significance. However, in exhausted rats there was a significant training-diet interaction, whereby sedentary rats on the Sat diet and trained rats on the Mono and CHO diets had lower triacylglyceryl levels than sedentary rats on the CHO diet (Table 2).

CS activity was significantly affected by both training and diet composition; thus rats adapted to the Mono diet had significantly higher CS activity than rats on the CHO and Sat diets. A significant effect of diet composition, but not training, was found on HAD activity (Table 3), whereby rats that consumed the carbohydrate-rich diet had a lower HAD activity than rats that consumed the fat-rich diets. GS was significantly affected by diet at both low and high (P = 0.06) glucose 6-phosphate concentrations, whereby the Mono-fed rats had higher GS values than Sat- and CHO-fed rats. There was no effect of training on GS at either glucose 6-phosphate concentration, and the fractional activity of GS was not affected by either diet composition or training (Table 3).
Dietary Fat and Endurance Performance

The two main findings of this study were that endurance performance was not enhanced after adaptation to a high-fat diet containing 15 E% carbohydrate compared with a carbohydrate-rich diet in either trained or sedentary rats. This is in fact consistent with a study by Tollenaar (35), in which sedentary rats showed no difference in endurance but not exercise endurance.

Adaptation to high-fat diets has consistently been shown to change substrate utilization toward higher fat oxidation during exercise (10, 12, 27). In rats and humans, several mechanisms have been demonstrated that could account for this change: a decreased muscle glycogen utilization during exercise (18, 24, 27, 32), an increased plasma FFA concentration during exercise (6, 16, 32), and/or an increased β-oxidative capacity after the diet adaptation (8, 10, 24, 32). These dietary fat-induced changes toward glycogen sparing and/or higher fat oxidation during exercise are generally considered to favor endurance performance capacity in both humans and rats (10, 17, 18, 32) and as such have been the object of several studies.

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performance when tested weekly over 7 wk while consuming diets containing 20, 40, and 70 E% fat. This contrasts with the large increases in endurance performance after adaptation to fat-rich diets by both trained (18, 32) and sedentary rats (24, 32) and trained dogs (15), as previously found by others. In an application of an overall perspective to the studies of high-fat-diet adaptation in rats, it seems clear that, compared with a carbohydrate-rich diet, enhanced performance was demonstrated when the fat-rich diets contained no carbohydrates (18, 24, 32), in contrast to the similar performance observed in the present study and the study by Tollenaar (35), in which the carbohydrate content in the fat-rich diet was 15 or 17 E%, respectively. This suggests that the inclusion of a moderate amount of carbohydrate (15–20 E%) in a fat-rich diet ameliorates the fat-diet-induced performance increase. When rats were fed a carbohydrate-free, fat-rich diet, preexercise muscle and liver glycogen values were higher after the carbohydrate-rich than after the fat-rich diet in both sedentary (6, 24) and trained animals (18, 32), contrasting again with the findings of the present study and the study by Gleson and Waring (9), in which no differences in preexercise muscle glycogen and smaller differences in liver glycogen were observed between a carbohydrate-rich diet and a fat-rich diet containing some carbohydrate. Reynolds et al. (30) demonstrated that, in dogs maintained on either a fat-rich diet (60 E% fat, 15 E% carbohydrate) or a carbohydrate-rich diet (15 E% fat, 60 E% carbohydrate) and trained for 8 wk, there was no decrease in either preexercise muscle glycogen stores (semitendinosus) or more importantly in muscle glycogen breakdown during a standard aerobic test. This suggests that the inclusion of a moderate amount of carbohydrate in a fat-rich diet attenuates the decrease in liver glycogen storage and totally abolishes the decrease in muscle glycogen storage and possibly utilization during exercise that is observed with fat-rich carbohydrate-free diets. This could be an explanation for the observed lack of a fat-diet-induced increase in endurance performance in rats in the present study. Lapachet et al. (18) demonstrated that, after an 8-wk consumption of a fat-rich diet, percent body fat in male rats was 2% higher compared with a carbohydrate-rich diet; however, despite the change in body composition, endurance performance was highest after the fat-rich diet. We did not measure body composition, but it is possible that a 4-wk adaptation to a fat-rich diet did induce higher body fat content and that this could partly explain a lack of a fat-diet-induced increase in endurance. There are, at present, insufficient data to discuss whether the carbohydrate content of the fat-rich diet would also play a crucial role in humans. The study by Phinney et al. (27) demonstrates that very marked metabolic changes were present after a 4-wk adaptation to a carbohydrate-free fat-rich diet (eucaloric ketogenic diet). However, the relevance of such dietary conditions to the human condition is, in any case, questionable.

Effects of fatty acid composition on endurance and substrate utilization. There is only limited evidence as to how a difference in dietary fatty acid composition will affect endurance and substrate utilization during exercise. Ayre and Hulbert (2) demonstrated that endurance performance, measured in vitro in rat extensor digitorum longus muscle after intermittent low-frequency stimulation, was lower after 9 wk of adaptation to a diet rich in n-6 fatty acids compared with a diet rich in n-3 fatty acids; both diets contained 10% wt/wt fat. Studies have demonstrated that cardiovascular function is affected by the dietary fatty acid profile, most likely through an effect on heart muscle; however, the mechanism behind these changes has not been explained (5, 20). In this study there was no effect of dietary fatty acid composition on endurance performance in trained or sedentary animals, but substrate oxidation was affected by dietary fatty acid composition. RER values measured during the initial phase of exercise show that fat oxidation was significantly higher during the initial phase of exercise after the Mono diet compared with the Sat diet (Fig. 2). It is not clear whether this difference in substrate oxidation persisted throughout the endurance bout; however, when substrate recruitment is calculated over the exercise bout...
(preexercise values minus values at exhaustion divided by average running time), the total glycogen (muscle and liver) breakdown in sedentary rats was 3.95 ± 0.27 and 6.28 ± 0.46 mmol·kg⁻¹·wt⁻¹·min⁻¹ and in trained rats was 1.99 ± 0.15 and 2.22 ± 0.17 mmol·kg⁻¹·wt⁻¹·min⁻¹ in Mono- and Sat-fed rats, respectively. In contrast, triacylglycerol breakdown (preexercise values minus values at exhaustion) in sedentary rats was 4.6 ± 3.1 and 2.9 ± 2.1 mmol/kg dry wt and in trained rats was 7.6 ± 2.9 and 1.0 ± 3.2 mmol/kg dry wt in Mono and Sat rats, respectively. Thus total glycogen breakdown was higher and muscle triacylglycerol breakdown was lower in the Sat compared with the Mono rats, which provides support for a difference in substrate recruitment and most likely oxidation. The finding of a significantly higher total GS activity in Mono compared with Sat rats, as well as a lower serum FFA and plasma glucose in the rested Mono rats compared with Sat rats, indicates that dietary fatty acid composition did have a differential impact on the metabolic adaptations. However, despite the positive changes that occurred in Mono rats, such as a higher fat oxidation and a lower total glycogen utilization, this did not lead to enhanced endurance. It is not clear why there was no benefit in performance from these adaptations, but it is tempting to speculate that the adaptations induced in the enzymes responsible for the transport and oxidation of fat were insufficiently increased to accommodate the higher fatty acid flux and utilization.

The finding of a lower RER during the initial phase of exercise after the Mono diet compared with the Sat diet is in accordance with the studies that have demonstrated preferential mobilization (29) and oxidation (13, 19, 31) of unsaturated vs. saturated fatty acids. Several mechanisms have been put forward to explain the increased oxidation of unsaturated to saturated fatty acids. This could be mediated by an increase in the solubility of lipids in circulating lipoproteins, and thereby an enhanced enzyme-substrate contact, due to the unsaturation of the lipids (4, 7). Another potential mechanism is an increased lipoprotein lipase (LPL) activity in heart and skeletal muscle after adaptation to an unsaturated-fat vs. a saturated-fat diet, as suggested by Shimomura et al. (31). Melin and co-workers (23) demonstrated that chylomicrons containing long-chain polyunsaturated fatty acids (20:4 and 20:5) were less susceptible to recruitment by LPL. However, canola oil, the fat source of the Mono diet in the present study, primarily contains oleic (18:1), linoleic (18:2), and α-linolenic (18:3) fatty acids, thus fatty acids that are susceptible to recruitment by muscle LPL. In accordance with Shimomura et al (31), both mechanisms, an enhanced enzyme-substrate contact and an increased muscle LPL activity, would lead to an increased delivery of unsaturated fatty acids to the muscle sarcolemma and, most probably, an increased fat oxidation, as indicated by the lower RER values in the initial phase of exercise in animals fed the Mono diet.

Summary. In the present study a fat-rich diet with 15 E% of carbohydrate did not affect exercise performance differently from a carbohydrate-rich diet. However, the data suggest a shift toward faster fat utilization during exercise in the high-fat-fed sedentary and trained rats, the magnitude of which was dependent on the type of dietary fat.

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