Fat Metabolism During Exercise: A Review

Part III: Effects of Nutritional Interventions

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Jeukendrup AE, Saris WHM, Wagenmakers AJM. Fat Metabolism During Exercise: A Review – Part III: Effects of Nutritional Interventions. Int J Sports Med 1998; 19: 371 – 379

By changes in nutrition it is possible to manipulate fat oxidation. It is often theorized that increasing fat oxidation may reduce glycogen breakdown and thus enhance performance. Therefore, the effects of acute, short-term and long-term fat feeding have been subjects of investigation for many years. Ingestion of long-chain triacylglycerols (LCT) during exercise may reduce the gastric emptying rate and LCT will appear in the plasma only slowly. Medium-chain triacylglycerols (MCT) do not have these disadvantages and they are rapidly oxidized. However, the contribution of MCT to energy expenditure is only small because they can only be ingested in small amounts without causing gastrointestinal distress. So at present, fat supplementation in the hours preceeding to or during exercise (either long chain or medium chain triacylglycerols) cannot be recommended. High-fat diets and fasting have been suggested to increase fatty acid availability and spare muscle glycogen resulting in improved performance. Both fasting and short term high-fat diets will decrease muscle glycogen content and reduce fatigue resistance. Chronic high-fat diets may provoke adaptive responses preventing the decremental effects on exercise performance. However, at present, there is little evidence to support this hypothesis. Also from a health perspective, caution should be exercised when recommending high-fat diets to athletes.

■ Key words: High-fat diets, medium-chain triacylgycerols, MCT, fasting, caffeine, carnitine.

Introduction

This is the last of three parts of a review on fat metabolism during exercise. In part I (44) the importance of fat as a substrate during exercise has been outlined and it was described how fat is mobilized from adipose tissue and several aspects of fat metabolism within the muscle were discussed. Part II (45) focused on the interaction between carbohydrate and fat metabolism and the regulation of substrate utilization. In addition the effects of exercise intensity and training were discussed. Here (in part III) we will discuss the short-term and long-term effects of nutrition on fat metabolism as well as the effect of nutritional supplements on fat utilization and exercise performance.

Nutrition and Substrate Utilization during Exercise

Fat supplementation

Fat supplementation during exercise is usually regarded as undesirable since endogenous fat stores are very large and there may not be a need to supplement additional fat. In addition there is the risk that ingestion of long chain triacylglycerols leads to an increase of the adipose tissue compartment and increased body weight. Arguments that have been put forward in favour of fat supplementation are mainly based on a possible "glycogen sparing effect" and subsequent performance improvement. Fat supplements can be ingested as either long chain triacylglycerols or medium chain triacylglycerols.

Ingestion of long chain triacylglycerols during exercise

Nutritional fats include triacylglycerols, phospholipids and cholesterol of which triacylglycerols and possibly phospholipids can contribute to any extent to energy provision during exercise (see Part I and II of this review [44,45]). In contrast to carbohydrates, nutritional fats reach the circulation only slowly since they are potent inhibitors of gastric emptying (33). Furthermore, the digestion in the gut and absorption of fat are also rather slow processes compared to the digestion and absorption of carbohydrates. Bile salts, produced by the liver and lipase secreted by the pancreas are needed to split the long chain triacylglycerols (LCT) into glycerol and 3 long chain fatty acids (LCFA) or monoacylglycerol and 2 fatty acids. The fatty acids move into the intestinal mucosa cells and are reesterified in the cytoplasm to form long chain triacylglycerols (LCT) (2). These LCTs will be encaptured by coat of proteins (chylomicrons) to make them water soluble (64). These chylomicrons are then released through the interstitial compartment into the lymphatic system which ultimately drains in the systemic circulation. Exogenous fat enters the systemic circulation much slower than carbohydrates which are absorbed as glucose (or to minor extents as fructose or galactose) and directly

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enter the main circulation through the porta vein. Long chain dietary fatty acids typically enter the blood 3 – 4 hours after ingestion (17,23).

Also important is that these long chain fatty acids enter the circulation in chylomicrons and it is generally believed that the rate of breakdown of chylomicron-bound triacylglycerols by musle during exercise is relatively low and contributes only minimally to energy expenditure (49). It has been suggested that one of the roles of these triacylglycerols in chylomicrons is the replenishment of intramuscular triacylglycerols stores after exercise (65).

Summing up: Long chain triacylglycerols ingestion during exercise is not desirable because of the following reasons: 1. It slows gastric emptying, 2. the LCT only slowly appear in the systemic circulation, and 3. LCT enter the systemic circulation in chylomicrons which are believed to be no major fuel during exercise but may serve to replenish intramuscular TG stores after exercise.

Ingestion of medium chain triacylglycerols

Although fat in general is slowly hydrolyzed and absorbed this also depends on the chain length of the fatty acids. In contrast to the long chain triacylglycerols, short and medium chain triacylglycerols (MCT) are more rapidly absorbed and they are not transported by chylomicrons but directly enter the systemic circulation through the portal vein. Consequently they will enter the systemic circulation more rapidly.

With the introduction of medium chain fatty acids (MCFA) in enteral and parenteral nutrition, its possible role in sports nutrition became subject of investigation. MCFA contain 8 or 10 carbons, whereas LCFA contain 12 or more carbons. Unlike most LCT, MCT are liquid at room temperature. This is in part the result of the small molecular size of MCT. MCT are more polar and therefore better soluble in water. This greater water solubility and smaller molecular size has consequences at all levels of metabolism. MCT are more rapidly digested and absorbed in the intestine than LCT. Furthermore MCFA follow the portal venous system and enter the liver directly while LCFA follow the slow lymphatic system (2,30,35). It has been suggested that MCT may be a valuable exogenous energy source during exercise in addition to carbohydrates (42). Also it has been suggested that MCT ingestion may improve exercise performance by elevating plasma fatty acid levels and sparing muscle glycogen (89) since it has been observed that increased availability of plasma fatty acids reduced the rate of muscle glycogen breakdown and delayed the onset of exhaustion (13,29,34,73,92).

MCT added to CHO drinks did not inhibit gastric emptying (4). In fact the drinks with MCT emptied faster from the stomach than an isocaloric CHO drink. In a subsequent study the oxidation rates of orally ingested MCT were investigated (42). Eight well-trained athletes cycled 4 times 180 min at 50% W_{max} (57% $\dot{V}O_2$max). Subjects ingested either CHO, CHO + MCT, or MCT. During the second hour (60 – 120 min period) the amount of MCT oxidized was 72% of the amount ingested with CHO + MCT whereas during the MCT trial only 33% was oxidized. It was concluded that more MCT is oxidized when ingested in combination with CHO. Data confirmed the hypothesis that oral MCT might serve as an energy source in addition to glucose during exercise since the metabolic availability of MCT was high during the last hour of exercise with oxidation rates being as high as 70% of the ingestion rate. However, the maximal amount of oral MCT that could be tolerated in the gastrointestinal tract is small (about 30 g) and this limited the contribution of oral MCT to total energy expenditure to values between 3 and 7%. We also investigated MCT oxidation in CHO + MCT supplements under conditions where the reliance on blood substrates is maximal such as in a glycogen depleted state (43). Subjects exercised to exhaustion the evening before the experiment in which MCT oxidation was determined during exercise at 57% $\dot{V}O_2$max. Although total fat oxidation was markedly increased, MCT oxidation increased only marginally. The contribution of MCT to total energy expenditure was still small (about 6 – 8%) (43).

It has also been suggested that elevating plasma fatty acid levels by infusing intralipid and heparin will spare muscle glyogen and improve exercise performance (13,92). In theory, MCT ingestion could also be a way to elevate plasma fatty acid levels. However, well-trained athletes who ingested CHO + MCT or CHO displayed no significant differences in glycogen breakdown between the trials nor in the respiratory exchange ratio during exercise (47). This may be attributed to the relatively small amount of MCT (about 30 grams) that can be tolerated in the gasto-intestinal tract. Van Zeyl et al. (89) suggested that the amount of MCT ingested was too small to exert positive effects on performance. Therefore they gave subjects 86 g of MCT during 2 hours of exercise followed by a 40 k time trial. They observed decreased glycogen breakdown and increased performance when a CHO + MCT drink was ingested. However, recently we performed a similar experiment in which subjects received 85 g of MCT in a MCT or CHO + MCT drink during 2 hours of cycling exercise at 60% $\dot{V}O_2$max (40). CHO + MCT did not improve performance compared to CHO or placebo ingestion (46). However, when only MCT was ingested, peformance measured with a reliable time trial protocol (41) was decreased which was related to gastrointestinal complaints reported by the subjects (40). It is not known why subjects in the study of van Zeyl (89) did not experience gastro-intestinal complaints after MCT feeding and in our study subjects did (40).

In conclusion: 1. MCT is rapidly emptied from the stomach, absorbed and oxidized and 2. the oxidation of exogenous MCT was enhanced when coingested with CHO. 3. Ingestion of 30 g MCT did not affect muscle glycogen breakdown and 4. contributed only 7% to energy expenditure. 5. Ingestion of larger amounts of MCT resulted in gastrointestinal distress. Therefore MCT does not appear to have the positive effects on performance that are often claimed.

Substances that are claimed to promote fat oxidation

Caffeine

Caffeine has been shown to stimulate the mobilization of fatty acid indirectly by increasing the circulating epinephrine levels or directly by antagonizing adenosine receptors that normally inhibit hormone sensitive lipase and fatty acid oxidation. Costill et al. (13,19,36) showed that caffeine ingested one hour prior to the start of an exercise bout increased plasma fatty

acid concentrations and improved performance. The improvement in performance was explained by the increased availability of fatty acids, which would lead to a suppression of carbohydrate metabolism and consequently to a decreased glycogen utilization. However, the results of studies investigating the ergogenic effects of caffeine and the possible role of fatty acids are contradictory. Several studies showed increased plasma fatty acid concentrations but did not see any effect on fat oxidation (50,85) or performance (18,72,85). Other studies did not observe any effect on either fat metabolism or performance (7,86,95). Recent studies showed large improvements in performance with high doses of caffeine (24,25,67,81) and these improvements were not always accompanied by change in plasma fatty acid or fatty acid oxidation. For example Pasman et al. (67) studied subjects who exercised at 80% $\dot{V}O_2$max until exhaustion with caffeine doses ranging from 3 to 9 $mg \cdot kg^{-1}$. Time to exhaustion increased by 10–20% but this could not be explained from the increased availability of fatty acids. It is concluded that, although caffeine stimulates fatty acid mobilization, the ergogenic effect of caffeine cannot be explained by the increased availability of fatty acids (19,25). For details on caffeine metabolism and the ergogenic effects of caffeine we refer to several recent reviews (11,14,78,80,84, 88,94).

Carnitine

Carnitine is produced endogenously in hepatic tissue, kidney and the brain but is also exogenously derived from the diet (mainly from red meat). L-carnitine is not produced in the muscle and the muscle carnitine concentration is therefore dependent on the endogenous synthesis and the diet, while there is also a daily loss of L-carnitine through urine and faeces. In normal conditions (healthy men), there is a balance between the excretion and the synthesis and exogenous supply of L-carnitine. As discussed in part I (44), the main role of L-carnitine is its role in the transport of fatty acids across the mitochondrial inner membrane (9). The importance of carnitine in energy metabolism has lead to the belief that L-carnitine supplements may increase fat oxidation, thereby reducing the reliance on endogenous carbohydrate stores which theoretically could improve endurance performance. L-carnitine supplements were introduced as ergogenic acids after rumours were spread that the Italian soccer team, that became World Champion in 1982, had used L-carnitine supplements. Ingestion of L-carnitine has been observed to increase plasma L-carnitine concentratons but muscle carnitine uptake was not affected by the increased plasma carnitine concentration (79). This may be explained by the fact that the plasma carnitine concentration is about 100 times lower than the muscle carnitine concentration and thus any uptake of L-carnitine must occur against a large concentration gradient. Even the ingestion of a large oral dose of L-carnitine (3) or even infusion of L-carnitine (Rademaker, Rademaker, Saris and Wagenmakers; unpublished findings) may not result in a significant change in the muscle L-carnitine concentration. Consequently no difference was found in muscle carnitine concentration after L-carnitine ingestion (3,91). Although few investigations reported effects on heart rate, maximal oxygen uptake and performance (57), the majority of the studies could not find any effect of either orally or intravenously administered L-carnitine on substrate utilization or performance (8,27,79,87,96) (for review see [93] and [26]).

Nutritional Regimens and Fat Oxidation

Fasting

Fasting has been proposed as a way to increase fat utilization, spare muscle glycogen and improve exercise performance. In rats short-term fasting increases plasma epinephrine and norepinephrine concentrations, stimulates lipolysis and the concentration of circulating plasma fatty acids. This in turn increases fat oxidation and "spares" muscle glycogen leading to a similar (52) or even increased running time to exhaustion in rats (16). In humans, fasting also results in an increased concentration of circulating catecholamines, increased lipolysis, increased concentration of plasma fatty acids (15) and a decreased glucose turnover (51). Muscle glycogen concentration was unaffected by fasting (51,56). However, fasting will result in decreased liver glycogen stores as indicated by decreased Ra glucose during exercise after 3.5 days of fasting compared to the post absorptive state (51). Although it has been reported that fasting had no effect on endurance at low exercise intensities (45% $\dot{V}O_2$max), Zinker et al. (97) observed a 38% decreased performance with fasting at 79–86% $\dot{V}O_2$max. Loy et al. (56) reported a 15–63% decreased performance with fasting at 79–86% $\dot{V}O_2$max, and Gleeson et al. (22) reported a decreased performance at 100% $\dot{V}O_2$max (Table **1**). This decreased performance was not reversible by carbohydrate ingestion during exercise (74).

Although, it may be argued that the observed effects in most of these studies may be due to the fact that in the control situation the last meal was provided 3 hours before the exercise to exhaustion (Table **1**), and thus it may have been an effect of the pre-exercise feeding improving endurance capacity instead of decreased performance during fasting. However, also the studies comparing a 12 h fast to a more prolonged fast show decreased performance (51,59,97) and thus it seems justified to conclude that fasting decreases endurance capacity.

In summary, fasting increases the availability of lipid substrates resulting in increased oxidation of fatty acids at rest and during exercise. However, since the glycogen stores are not maintained, fatigue resistance and exercise performance are impaired.

Effects of short-term high-fat diet

A change in the carbohydrate-fat content of nutrition can cause a shift in substrate utilization. The ratio of energy providing substrates can be expressed as a food quotient (FQ) in analogy to RQ. A high-fat diet (low FQ), and concomitant low-carbohdydrate diet, leads to an increased contribution of fatty acids to total energy expenditure. A high-fat diet may lead to impaired performance as shown already in 1939 by Chirstensen and Hansen (10). In this early study, ingestion of a high-fat diet (> 90% of total energy intake) reduced time to exhaustion on a cycle ergometer by almost 60% compared to a high-CHO diet (10) (Table **2**). After consumption of a high-carbohydrate diet (high FQ) higher resting RQ values were observed whereas with a high-fat diet (low FQ) the RQ's were low. The intake of 100–200 g carbohydrate in the hours preceding exercise results is an increased carbohydrate oxidation at the cost of fat oxidation (1,10,62). With the reintroduction of the muscle biopsy technique in the late sixties, it was discovered that a

Table 1 The effects of prolonged fasting on endurance performance in men

Duration of fast	Subjects	Mode of exercise	Intensity	Time	% difference	Ref.
3 h	4 T	cycling	86% $\dot{V}O_2$max to exhaustion	115 min	−63%*	Loy et al., 1986
24 h	4 T	cycling	86% $\dot{V}O_2$max to exhaustion	42 min		
3 h	4 T	cycling	67% $\dot{V}O_2$max to exhaustion	201 min	−15%*	
24 h	4 T	cycling	66% $\dot{V}O_2$max to exhaustion	170 min		
3 h	6 T	cycling	78% $\dot{V}O_2$max to exhaustion	191 min	−26%*	Loy et al., 1986
24 h	6 T	cycling	80% $\dot{V}O_2$max to exhaustion	142 min		
3 h	6 T	cycling	68% $\dot{V}O_2$max to exhaustion	214 min	−21%*	
24 h	6 T	cycling	70% $\dot{V}O_2$max to exhaustion	168 min		
3 h	9 T	running	72% $\dot{V}O_2$max to exhaustion	82 min	−7%	Dohm et al., 1986
24 h	9 T	running	72% $\dot{V}O_2$max to exhaustion	76 min		
14 h	8 UT	cycling	45% $\dot{V}O_2$max to exhaustion	139 min	−15%*	Knapick et al., 1988
84 h	8 UT	cycling	45% $\dot{V}O_2$max to exhaustion	118 min		
12 h	5 UT	cycling	70% $\dot{V}O_2$max to exhaustion	120 min	−35%*	Maughan et al., 1988
36 h	5 UT	cycling	70% $\dot{V}O_2$max to exhaustion	78 min		
12 h	7 T	cycling	53% $\dot{V}O_2$max to exhaustion	144 min	−38%*	Zinker et al., 1990
36 h	7 T	cycling	54% $\dot{V}O_2$max to exhaustion	89 min		
4 h	6 UT	cycling	100% $\dot{V}O_2$max to exhaustion	212 s	−13%*	Gleeson et al., 1988
24 h	6 UT	cycling	100% $\dot{V}O_2$max to exhaustion	243 s		

$\dot{V}O_2$max = maximal oxygen uptake; T = trained, UT = untrained.
* indicates a significant difference between short-term and long-term fasting; $p < 0.05$.

Table 2 The effects of a short-term (< 7 days) high-fat diet on endurance performance

Species	Duration	fat%	CHO%	Exercise	Time	% difference	Glycogen	Ref.
Rat	1 week	75	0	35 m/min, 0% grade	45 min	+7%*	6.2	Miller et al., 1984
	1 week	11	68	5 m/min, 0% grade	42 min		7.3	
Human	1 day	68	16	Time to cycle 1600 kJ	139 min	−19%*	19.8	Startling et al., 1997
	1 day	5	83	ime to cycle 1600 kJ	117 min		11.8	
Human	3 days	94	4	176 W to exhaustion	88 min	−58%*	–	Christensen et al., 1939
	3 days	3	83	176 W to exhaustion	210 min		–	
Human	3 days	46	5	75% $\dot{V}O_2$max to exhaustion	57 min	−66%*	6.5	Bergström et al., 1967
	3 days	0	82	75% $\dot{V}O_2$max to exhaustion	167 min		32.9	
Human	3 days	–	< 10	70% $\dot{V}O_2$max to exhaustion	33 min	−58%*	–	Martin et al., 1978
		–	75	70% $\dot{V}O_2$max to exhaustion	78 min		–	
Human	5 days	69	5	25 min 65% $\dot{V}O_2$max	–	–	8.4	Jansson et al., 1982
	5 days	8	75	25 min 65% $\dot{V}O_2$max	–	–	7.1	
Human	7 days	50	38	75–80% $\dot{V}O_2$max to exhaustion	91 min	+20%*	–	Muoio et al., 1994
	7 days	15	73	75–80% $\dot{V}O_2$max to exhaustion	76 min		–	

Resting muscle glycogen is expressed in mg/kg and is measured in m. soleus in rats and m. vastus lateralis in humans. If applicable a conversion factor from dry muscle tissue to wet muscle tissue was applied. $\dot{V}O_2$max = maximal oxygen uptake; * indicates a significant difference in performance between the high-CHO and high-fat diet; $p < 0.05$.

high-fat, low-carbohydrate diet resulted in decreased muscle glycogen levels and this was the main factor causing lack of fatigue resistance during prolonged exercise (5, 6, 31, 32). Plasma fatty acid concentrations are elevated at rest and increase more rapidly when a low-carbohydrate diet is consumed (12, 58, 60). These changes in plasma fatty acid concentrations but also the increased plasma glycerol concentrations after a low-carbohydrate diet are indicative for increased lipolysis (15, 16, 51, 56, 97). Jansson and Kaijser reported that the uptake of fatty acids by the muscle during 25 min cycling at 65% $\dot{V}O_2$max was 82% higher in subjects receiving a high-carbohydrate diet (75%) for 5 days. Plasma fatty acids contributed 25% and 14% respectively to energy expenditure. Increased fatty acid concentrations in the blood after a period of carbohydrate restric-

tion will lead to an increased ketogenesis. After a few days of high-fat feeding the ketone body production increases 5-fold (20). The arterial concentration of ketone bodies may increase 10–20-fold (20). In the first phase of light to moderate exercise, ketone body concentrations usually decline and after 30–90 min they will increase again (20,51,97). However, the observed concentrations under those conditions are still higher after a high-fat diet compared to low-fat diets. Carbohydrate restricted diets may also lead to an increased breakdown of muscle TG (12,39,82).

In Table **2** the effects of short-term high-fat diets on exercise performance are listed. As discussed above the effect of short-term (3–7 das) high-fat feeding may decrease exercise time to exhaustion (5,10,21,31,32,58). To our knowledge, there is only one study that showed a beneficial effect of eating a slightly larger amount of fat. Muoio et al. (63) examined the effects of three moderate diets for 7 days in six runners. The percent energy contribution of carbohydrate, fat and protein were 61/24/14, 50/38/12 and 73/15/12, respectively. The authors conclude that the "high-fat" diet (38% fat) increases $\dot{V}O_2$max and running time to exhaustion. Furthermore the authors suggest that the mechanism by which performance is improved is through increased β-oxidation and fatty acid oxidation. However, no difference in fat oxidation (respiratory exchange ratio) was observed between the diets. The results may simply be explained by the fact that trials were not randomized, but were provided in the order low fat, medium fat, high fat. Therefore the results of Muoio et al. (63) should be interpreted with great caution.

An interesting study was recently performed by Starling et al. (83) who tried to manipulate intramuscular triacylglycerol stores by partially depleting these stores during a 120 min cycling bout at 65% $\dot{V}O_2$max which has been shown to result in relatively high rates of intramuscular triacylglycerol breakdown (75). Each subject then ingested a high-carbohydrate diet (83% of energy) or a high-fat diet (68% of energy) for 12 hours after this exercise bout. After a 12 hour fast these subjects then were biopsied and performed a time trial (equal to 1600 kJ). The intramuscular triacylglycerol concentrations measured from muscle biopsies dropped 6–11% during the exercise bout, although this decrease was statistically no significant. Ingstion of the high-fat diet increase in the intramuscular triacylglycerol stores by 21%. 24 hours after the ride, while they were still decreased when a high-carbohydrate diet was ingested. In parallel with these findings, muscle glycogen concentrations were higher after the high-carbohydrate diet, resulting in improved time trial performance compared to the high-fat diet.

In summary, short-term high-fat diets increase the availability of lipid substrates but reduced the storage of glycogen. As a result, although fat oxidation may be increased during exercise, fatigue resistance and exercise performance seem to be decreased.

Effects of a long-term high-fat diet

It has been suggested that a 5–7 day alteration in the dietary composition is an insufficient time to induce an adaptive response to the changed diet. Jansson and Kaijser concluded that a high-fat diet over a prolonged period resulted in a decreased utilization of carbohydrates and that this relative shortage of carbohydrates was compensated by an increased contribution of fat to energy metabolism (37,38). In rats it has been shown that adaptation to a high-fat diet leads to considerable improvements of endurance capacity (61,77). These adaptations can be attributed to the number of oxidative enzymes and a decreased degradation of liver glycogen during exercise (77). For instance, long-term high-fat diets have been shown to increase the concentrations of muscle 3-hydroxyacyl -CoA dehydrogenase in rats (54,61) and humans (48) and increase the concentration of intramuscular triacylglyerol (48), while muscle glycogen concentration typically decreases (Table **3**). These results suggest that after adaptation to a high-fat diet the capacity to oxidize fatty acids instead of carbohydrates is increased, because of an adaptation of the oxidative enzymes in the muscle cell.

Only few studies have looked at the effect of long-term high-fat diets on fat metabolism and exercise performance in humans. Phinney et al. (71) investigated exercise performance in obese subjects after 6 weeks of a high-fat diet (90 en% fat). Before and after the diet, the intention was to have subjects exercise at 75% $\dot{V}O_2$max until exhaustion. Subjects were able to exercise longer on the high-fat diet than they were on their normal diet while after the diet, fat became the main substrate. Results of this study, however, may have been influenced by the fact that these subjects were not in energy balance and lost 10.6 kg of body weight. So although there were no difference in the absolute $\dot{V}O_2$max before and after the dietary period, there were considerable differences in the relative intensity (after the high-fat diet exercise was performed at 60% $\dot{V}O_2$max while the exercise intensity was 76% $\dot{V}O_2$max before the diet).

The observed improvements in performance may have been an artefact rather than a positive effect of the adaptation period. Therefore Phinney et al. (69,70) performed a follow-up study in which trained subjects were studied before and after a 4 week high-fat diet (less than 20 g CHO per day). The diet drastically reduced the preexercise muscle glycogen concentration (143 ± 10 versus 76 ± 4 mmol glucose kg^{-1} wet weight muscle). However, no difference in the average time to exhaustion at 62–64% $\dot{V}O_2$max before and after the diet was found. However, the results are difficult to interpret because of the large variability of the subjects' performance times (times to exhaustion). One subject exercised 57% longer while other subjects showed no improvement or even decreased times to exhaustion. Also, the exercise intensity was relatively low and subject's reliance on carbohydrates during exercise at 62–64% $\dot{V}O_2$max was low. In such a situation reduced carbohydrate stores may not be limiting. It is possible that a higher exercise intensities performance woud have been impaired. The results of the study of Phinney (69,70 may be supportive of the view since the subjects with the highest R values (higher rates of CHO oxidation) showed a decreased performance whereas the subjects with the lowest R values displayed a increased time to exhaustion.

Nevertheless, it is remarkable that performance was not reduced in all subjects even though preexercise muscle glycogen levels were decreased by almost 50% and fat oxidation during exercise was markedly adaptations including a 44% increase of acyl-carnitine palmityol transferase (CPT) activity, and a 46% decrease of the hexokinase activity (70).

Table 3 The effects of long-term (> 7 days) high-fat diet on endurance performance

Species	Duration	fat%	CHO%	Exercise	Time	% difference	Glycogen	Ref.
Rat	4 weeks	78	1	28 m/min, 10% grade	115 min	+6%*	6.5	Conlee et al., 1990
	4 weeks	12	69	28 m/min, 10% grade	109 min		9.7	
Rat	5 weeks	75	0	35 m/min, 0% grade	47 min	+31%*	4.6	Miller et al., 1984
	5 weeks	11	68	35 m/min, 0% grade	36 min		6.7	
Rat	5 weeks	4	65	120 min, 20 m/min	–		17.8	Satabin et al., 1989
	5 weeks	30	5	120 min, 20 m/min	–		17.8	
Rat	8 weeks	79	0	29 m/min, 8% grade	356 min	+38%*	19.9	Lapacher et al., 1996
	8 weeks	10	69	29 m/min, 8% grade	258 min		21.2	
Rat	12 weeks	53	0	30 m/min, 10% grade	68 min	+62%*	25.8	Simi et al., 1991
	12 weeks	5	62	30 m/min, 10% grade	42 min		30.6	
Human	10 days	67	7	60% $\dot{V}O_2$max to exhaustion	80 min		–	Lambert et al., 1994
	10 days	12	74	60% $\dot{V}O_2$max to exhaustion	43 min	+88%*	–	
	10 days	67	7	90% $\dot{V}O_2$max to exhaustion	8.3 min		22.3	
	10 days	12	74	90% $\dot{V}O_2$max to exhaustion	12.5 min	-34%*	12.1	
Human	14 days	59	30	53% $\dot{V}O_2$max to exhaustion	270 min	+3%	–	Pruett et al., 1970
	14 days	9	87	51% $\dot{V}O_2$max to exhaustion	262 min		–	
	14 days	59	30	70% $\dot{V}O_2$max to exhaustion	162 min	-14%*	–	
	14 days	9	87	70% $\dot{V}O_2$max to exhaustion	189 min		–	
Human	28 days	85	2	64% $\dot{V}O_2$max to exhaustion	147 min	-3%	25.7	Phinney et al., 1983
	28 days	29	57	62% $\dot{V}O_2$max to exhaustion	151 min		13.7	
Human	49 days	62	21	70% $\dot{V}O_2$max to exhaustion	65 min	-36%*	20.2	Helge et al., 1996
	49 days	20	65	70% $\dot{V}O_2$max to exhaustion	102 min		26.6	

Resting muscle glycogen concentration is expressed in mg/g and is measured in m. soleus rats and m. vastus lateralis in humans. If applicable a conversion factor from dry muscle tissue to wet muscle tissue was applied. $\dot{V}O_2$max = maximal oxygen uptake; * indicates a significant difference in performance between the high-CHO and high-fat diet; $p < 0.05$.

Lambert et al. (53) fed 5 endurance trained cyclists for 14 days a somewhat less extreme high-fat diet or high-carbohydrate diet. The high-fat diet contained 67% fat and 7% CHO whereas the high-carbohydrate diet contained 74% CHO and 12% fat. In order to evaluate exercise performance three different exercise tests were performed: a Wingate test (sprint exercise), a cycling time to exhaustion test at a high intensity (90% $\dot{V}O_2$max) and an exhaustion test at a moderate intensity (60% $\dot{V}O_2$max). Muscle glycogen concentrations were 44% lower after the high-fat diet. No differences were found in sprint performance and time to exhaustion during high intensity exercise. However time to exhaustion during the moderate intensity exercise test was significantly longer (80 ± 8 min versus 43 ± 7 min). The high-fat diet also resulted in increased fat oxidation rates during exercise. Both the study of Lambert et al. (53) and Phinney et al. (70) used relatively low exercise intensities (60–65% $\dot{V}O_2$max) which are far below intensities in competition. So it is not clear how these results would translate into practical applications in training and competition.

Helge et al. (28) studied 20 untrained individuals who underwent an endurance training program for 7 weeks while ingesting a high-fat diet (62% fat; n = 10), or a high-carbohydrate diet (65% CHO; n = 10). The training resulted in an 11% increase in $\dot{V}O_2$max in both groups after 7 weeks, while also time to exhaustion at 80% $\dot{V}O_2$max was significantly improved in both groups. The increase, however, was more pronounced in the high-CHO group than in the high-fat group. The time to exhaustion increased from 35 min to 102 min with the high-CHO diet and only to 65 min in the high-fat group. Interestingly, after the seven weeks both groups received a high-carbohydrate diet and performance was maintained in the high-CHO group whereas the high-fat group improved (to 77 min) This, however, was still less than the performance of the high-CHO group. The authors, therefore, conclude that ingesting a high-fat diet during an endurance training program is detremental to performance. Because switching to a high-CHO diet after 7 weeks of a high-fat diet did not reverse the negative effects, they conclude that this negative effect on performances is not simply due to a lack of carbohydrate as a fuel but rather due to suboptimal adaptations to the training. The results of these studies have been summarized in Table **3**.

From a health perspective, eating large amounts of fat has been associated with the development of obesity and cardiovascular disease. If this is also the case for athletes has not yet been determined. To our knowledge there are no studies available at this point describing the effects of high-fat diets on cardiovascular risk factors in training athletes. Recently no changes were found in plasma LDL, HDL and total cholesterol levels in male and female runners with diets in the range of 17–40% fat (55,68). Although it is generally accepted that the risk of obesity and cardiovascular diseases increases with the consumption of high-fat diets in sedentary people. Regular exercise or endurance training seems to attenuate these risks (76). Exposure to high-fat diets has also been associated with insulin resistance and recently this has been linked to an effect of the intramuscular triacylglycerol pools on glucose uptake (66).

However, this observation was made in obese subjects and it is not clear whether these results can be extrapolated to athletes, especially since athletes seem to have larger intramuscular triacylglycerol stores and increased insulin sensitivity. In addition consumption of high-fat diets and training have been associated with a decreased immune function. Venkatraman et al. (90), however, could not find any deleterious effects of a 40% fat diet on several indicators of immune function in well-trained runners. As there is little information about the negative effects of high-fat diet on athletes and the effects of those diets on performance are unclear, we suggest that caution should be exercised when recommending high-fat diets to athletes.

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