

# Effects of Dietary Fat on Postprandial Substrate Oxidation and on Carbohydrate and Fat Balances

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## Abstract

To study the effect of dietary fat on postprandial substrate utilization and nutrient balance, respiratory exchange was determined in seven young men for 1 h before and 9 h after the ingestion of one of three different breakfasts: i.e., bread, jam, and dried meat (482 kcal: 27% protein, 62% carbohydrate, and 11% fat); bread, jam, and dried meat plus 50 g of margarine containing long-chain triglycerides (LCT); or bread, jam, and dried meat plus 40 g medium-chain triglycerides (MCT) and 10 g LCT margarine (858 kcal: 15% protein, 35% carbohydrate, and 50% fat).

Plasma glucose concentrations peaked 45 min after the start of the meals. When compared with the low fat meal, the LCT margarine supplement had no effect at any time on circulating glucose and insulin concentrations, nor on the respiratory quotient. When MCTs were consumed, plasma glucose and insulin concentrations remained lower and plasma FFA concentrations higher during the first 2 h.

9 h after the breakfasts, the amounts of substrates oxidized were similar in each case, i.e., ~320, 355, and 125 kcal for carbohydrate, fat, and protein, respectively. This resulted in comparable carbohydrate (mean  $\pm$  SD =  $-22 \pm 32$ ,  $-22 \pm 37$ , and  $-24 \pm 22$  kcal) and protein balances ( $-7 \pm 9$ ,  $+7 \pm 7$ , and  $-8 \pm 11$  kcal) after the low fat, LCT- and MCT-supplemented test meals, respectively. However, after the low fat meal, the lipid balance was negative ( $-287 \pm 60$  kcal), which differed significantly ( $P < 0.001$ ) from the fat balances after the LCT- and MCT-supplemented meals, i.e.,  $+60 \pm 33$  and  $+57 \pm 25$  kcal, respectively. The results demonstrate that the rates of fat and of carbohydrate oxidation are not influenced by the fat content of a meal.

## Introduction

Long-term weight maintenance in normal and obese subjects requires that average daily energy intakes be commensurate with the average amounts of energy expended per day. Furthermore,

carbohydrate, protein, and fat oxidation must occur in proportions matching the relative contributions made by these nutrients in the diets, since otherwise accumulation and/or depletion of carbohydrate, protein, or fat would occur.

Nitrogen balance is known to be maintained on high or low, but adequate, protein intakes, which indicates that adjustment of protein oxidation to intake is effectively achieved. It is not well known how this is brought about, but it clearly appears to have a high priority in the regulation of the body's metabolism. In the case of carbohydrate, oxidation must obviously be attuned to intake, since the body's glycogen stores are small (1, 2) in relation to daily carbohydrate turnover. By regulating the mobilization, availability, and use of metabolic fuels, the rate of carbohydrate utilization is made to match carbohydrate intake well enough to prevent excessive exhaustion or accumulation of glycogen reserves. Maintenance of fat balance would of course be facilitated as well if fat oxidation tended to adjust itself to fat intake, as is the case for protein and carbohydrate. Not much is known about this phenomenon, however, and the purpose of the studies reported here was to assess to what extent the ingestion of fat may influence fat oxidation.

Studies of metabolic responses to nutrient intake are usually performed on fasting individuals, when absorption of previous meals has been completed and when acute variations in metabolic rates have subsided. However, the effect of fat intake on fat oxidation would be difficult to assess by feeding fat to fasting subjects, because fat is already the predominant substrate in the postabsorptive state and because its contribution to energy expenditure increases spontaneously as more time elapses since food was last consumed. Furthermore, it is of greater interest to study the metabolic response to fat when it is consumed with other nutrients, as occurs in daily life. Therefore, we determined (by indirect calorimetry) the rates of protein, carbohydrate, and fat oxidation (3) after breakfasts providing fixed amounts of carbohydrate and protein, without or with a supplement of fat, were ingested. Carbohydrate and protein were given in amounts equal to the approximation of carbohydrate and protein oxidation expected to occur during one day (i.e., from 8 a.m. to 5 p.m.) after a breakfast containing primarily carbohydrate. When fat was added, either in the form of long-chain triglycerides (LCT)<sup>1</sup> or medium-chain triglycerides (MCT), the meal provided carbohydrate, protein, and fat in proportions similar to the relative contribution made by these substrates to the fuel mixture oxidized in the premeal fasting state.

The composition of the metabolic fuel mix oxidized during the 9-h postprandial period was not altered by the addition of fat. The maintenance of fat balance, which is of course essential for weight maintenance, does not appear to be facilitated by

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1. Abbreviations used in this paper: BUN, blood urea nitrogen; LCT, long-chain triglyceride(s); MCT, medium-chain triglyceride(s); RQ, respiratory quotient.

metabolic regulatory effects similar to those involved in the maintenance of carbohydrate and protein balances.

## Methods

**Subjects.** Seven healthy male students whose physical characteristics are presented in Table I volunteered for this study. They were all known to have maintained stable body weights for several months before the study and had no family history of diabetes. The nature and purpose of the study were explained to each subject before he gave his consent to participate. During the initial week of testing, the body composition of all subjects was estimated by skinfold measurements (4). The subjects were instructed to consume an equilibrated diet providing at least three meals and 300 g carbohydrate per day and to avoid vigorous and/or sustained physical activity for the 3 d preceding the tests, so that their metabolic state would be within the usual range. As judged from the various base-line measurements obtained before the test meals were measured (Figs. 1 and 2), this appeared to be the case.

**Protocol.** The subjects spent the night preceding the tests in a room adjoining that in which the experiment was performed. Their last meal was consumed between 6 and 8 p.m., and included 4 dl of sugared fruit juice, providing 200 kcal of carbohydrate. No food or drink was allowed thereafter, except water. They were awoken at 6:30 a.m., and after they voided, an 18-gauge Teflon catheter was placed in a forearm vein for blood sampling and was kept patent with physiological saline. 15 min after the insertion of the catheter, continuous respiratory exchange measurements were begun using a ventilated hood system (5, 6). All measurements were obtained while the subjects were lying in hospital beds that could be adjusted for optimal comfort.

After 1 h of base-line measurements, the subjects ingested one of the three test breakfasts described in Table II over a 15–20-min period (time zero = start of meal). The hood was then replaced over the subjects' heads and respiratory exchange measurements were made continuously from 30 min until 9 h. The subjects could listen to the radio and/or read but were not allowed to fall asleep. They were requested to limit their movements to changing their position slightly. After 5 h the venous catheter was removed, urine was collected, and the subjects were allowed to leave the hood for a 20-min period, during which the gas analyzer's calibrations were verified.

Blood samples were obtained every 15 min for the first 2 h, and at 30-min intervals until 5 h had elapsed. A venipuncture was made to obtain an additional blood sample at the end of the study (9 h), when the urine was again collected. Blood glucose concentrations were determined by the glucose oxidase method (8) using a glucose analyzer (model II; Beckman Instruments, Inc., Fullerton, CA). Plasma insulin (9), FFA (10, 11), catecholamine concentrations (12, 13), blood urea nitrogen (BUN), and urinary nitrogen (Kjeldahl) were also determined.

The order of the meals for a given subject was randomly assigned, with at least 1 wk allowed between tests. With the exception of one subject, the three tests were performed within a 5-wk period.

**Data analysis.** The urinary nitrogen excretions over the 10-h collection period, corrected for changes in the body urea nitrogen pool (assuming a distribution volume of 0.6 liter/kg body wt [14]) (Table III), were used to calculate individual rates of protein oxidation. The non-protein respiratory quotients (RQs) were obtained by subtracting from the respiratory exchange the contribution made by oxidation of protein,

Table II. Nutrient and Energy Contents of the Three Breakfasts

Breakfast	Kilocalories	Percent
Control (low fat, 482 kcal)*		
Carbohydrate	300	62
Protein	129	27
Lipid	53	11
LCT (with margarine, 858 kcal)‡		
Carbohydrate	301	35
Protein	130	15
Lipid	427	50
MCT (with MCT margarine, 856 kcal)§		
Carbohydrate	300	35
Protein	129	15
Lipid	74 (LCT)	9
	353 (MCT)	41

The composition of the various food items was based on Geigy Scientific Tables (7) and on statements by the manufacturers of jam, margarine (Migros, Zurich, Switzerland), and MCT margarine (Union Deutscher Lebensmittelwerke, Hamburg, FRG). The energy content of carbohydrate was taken to be 4.1 kcal/g starch (or starch equivalent), 4.3 kcal/g protein, 9.1 kcal/g LCT, and 8.3 kcal/g MCT.

\* 75 g white bread, 72 g jam, and 60 g dried meat.

‡ 75 g bread, 72 g jam, 60 g dried meat, and 50 g margarine.

§ 75 g white bread, 72 g jam, 60 g dried meat, and 40 g MCT margarine plus 14 g margarine.

which was assumed to proceed at a constant rate. Energy expenditure and carbohydrate and fat oxidation rates were calculated from the non-protein RQs according to the tables of Lusk (3) over 5-min intervals and averaged over 30-min periods. Substrate balances were computed for periods of 4.5 h and 9 h after the meal.

The thermic effect of the meal was calculated by dividing the increases in energy expenditure above the individual base-line rates by the energy content of the meal. All results are presented as means  $\pm$  SEM. Comparisons between meals were tested using the paired *t* test.

## Results

**Blood parameters.** In the basal postabsorptive state, plasma glucose, insulin, and FFA concentrations were not statistically different from one test to another (Fig. 1). Plasma glucose concentrations reached a peak after 45 min in each of the three tests. The rise in glucose concentration was somewhat attenuated by the presence of fat in the meal, though this effect reached statistical significance only with MCT, 30 min ( $P < 0.05$ ) and 45 min ( $P < 0.01$ ) after the meal. Subsequent differences between average blood glucose levels were not statistically different after the three test meals. When the integrated surface areas above the base line were compared, the blood glucose response was significantly lower during the first 2 h after the MCT meal than after the other two meals ( $P < 0.05$ ). This difference was reflected in the plasma insulin concentrations, which were significantly lower ( $P < 0.05$ ) at 45, 60, 75, 90, and 120 min after the MCT meal, when compared with the low fat meal. No statistical differences were apparent between blood glucose and insulin levels after the two fat-containing meals. Plasma FFA concentrations fell significantly after each test meal, but the lowest values observed after the MCT meal remained higher than after the low fat meal (from 45 to 180 min;  $P < 0.01$ ), presumably due to the

Table I. Physical Characteristics of the Subjects

	Means $\pm$ SD	Range
Weight (kg)	71.6 $\pm$ 8.6	63.6–86.9
Height (cm)	178 $\pm$ 7	166–186
Age (yr)	23 $\pm$ 2	21–26
Body fat (%)	16 $\pm$ 3	10.6–19.8

Table III. Urinary N Excretion and Changes in BUN Levels

Test meal	Urinary N g/9 h	BUN		Change in urea N g/10 h	Protein oxidation rate g protein/h
		Initial mg/dl	Final mg/dl		
Control breakfast	4.7±0.3	12.5±1.1	12.3±1.1	0.0±0.3	3.1±0.1
LCT breakfast	4.5±0.2	13.3±1.3	12.6±1.4	-0.3±0.2	2.8±0.2
MCT breakfast	5.3±0.4	13.5±0.7	12.0±0.6	-0.7±0.1	3.2±0.3

appearance of some medium-chain fatty acids in the circulation. FFA concentrations after the LCT meal reached their lowest level after 75 min, and then began to rise earlier than after the low fat meal, being significantly greater at 120 and 180 min ( $P < 0.05$ ). During the latter part of the test, FFA concentrations tended to be lower with the MCT meal, reaching significance at 300 min (MCT meal vs. LCT meal;  $P < 0.01$ ).

Basal plasma norepinephrine concentrations were the same ( $193 \pm 21$ ,  $204 \pm 35$ , and  $193 \pm 29$  pg/ml) in the low fat, LCT, and MCT tests, respectively, and rose ( $P < 0.01$ ) to a similar extent 2 h after each test meal ( $225 \pm 26$ ,  $232 \pm 32$ , and  $228 \pm 24$  pg/ml,

respectively). However, plasma epinephrine concentrations were not influenced by the test meals.

**Energy expenditure.** The rates of energy expenditure during the three tests are presented in Fig. 2. The mean postabsorptive resting metabolic rates were the same before the test meals were consumed, i.e.,  $1.33 \pm 0.07$  kcal/min (Table IV). In response to the meals, energy expenditure rose promptly, reaching a peak at  $\sim 60$  min, and thereafter declined progressively. After the two fat-containing meals, energy expenditure tended to be higher than after the low fat meal, as would be expected on account of the greater energy content of the fat-supplemented meals. This effect was statistically significant ( $P < 0.01$ ) when the MCT meal was compared with the low fat meal at 75, 285, 375, and 405 min. The increases in the rate of energy expenditure over the base-line rates, integrated over 9 h, were equivalent to  $76 \pm 10$ ,  $96 \pm 11$ , and  $105 \pm 13$  kcal/9 h for the low fat, LCT, and MCT meals, respectively (Table IV). When expressed as a percentage of the energy content of the meals, the thermic effects of the meals averaged  $15.8 \pm 2.1$ ,  $11.2 \pm 1.3$ , and  $12.3 \pm 1.5\%$ , respectively, but they were not statistically different. The thermic effect (20 kcal) that can be attributed to the margarine supplement (374 kcal) corresponds to  $\sim 5\%$  of its energy content, which is in agreement with the relatively low thermic effect of dietary fat

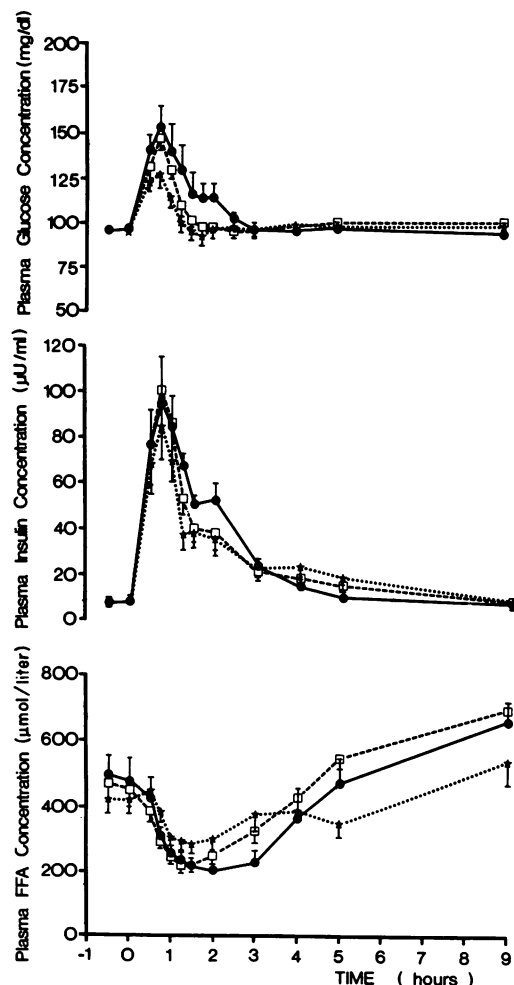


Figure 1. Changes in plasma glucose, insulin, and FFA concentrations in response to a low fat breakfast (●), an LCT-supplemented breakfast (□), and an MCT-supplemented breakfast (▲). (Means±SE;  $n = 7$ .)

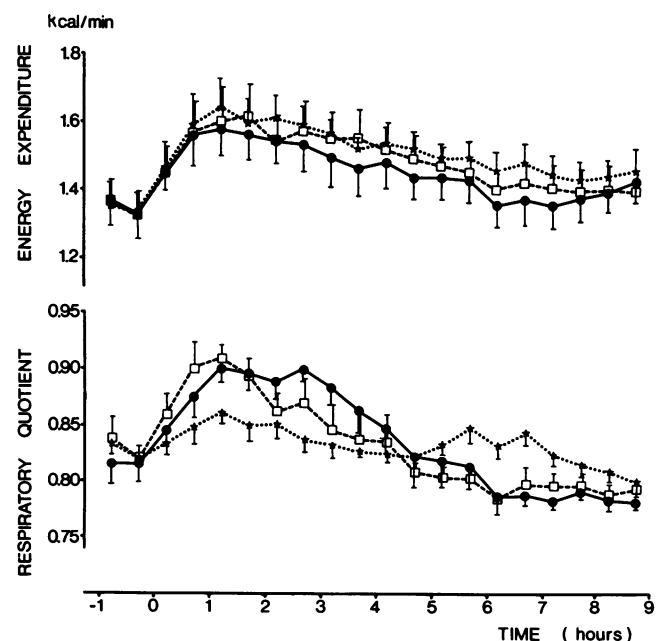


Figure 2. Changes in energy expenditure and RQ in response to a low fat breakfast (●), an LCT-supplemented breakfast (□), and an MCT-supplemented breakfast (▲). (Means±SE;  $n = 7$ .)

Table IV. Resting Energy Expenditure and Thermic Effect of the Three Types of Breakfasts

	Resting energy expenditure	Thermic effect		Energy balance after 9 h
	kcal/min	kcal/9 h, above base line	% energy intake	kcal
Low fat				
meal	1.33±0.07	76±10	15.8±2.1	-310±38
Fat meal	1.32±0.07	96±11	11.2±1.3	49±36
Fat meal				
MCT	1.33±0.06	105±13	12.3±1.5	32±28

(15). In the case of MCT supplementation, this effect amounts to 29 kcal, or 7%, which may be related to the increase in energy dissipation reported to occur in rats when LCT are replaced by MCTs (16).

**Substrate oxidation and balances.** The changes in the RQs induced by the three meals are presented in the lower panel of Fig. 2. In the postabsorptive state the RQs were similar during the half-hour immediately preceding the meals, i.e.,  $0.815 \pm 0.016$ ,  $0.819 \pm 0.013$ , and  $0.820 \pm 0.008$  before the low fat, LCT, and MCT test meals, respectively. The RQ rose rapidly after food intake, reaching peak values after 75 min in each case. At no time could a significant difference between the RQ after the low fat and the LCT meal be detected, which indicates that the effect of dietary fat on substrate oxidation was negligible. However, when compared with the low fat meal, the changes in RQ over time after the MCT meal differed somewhat, being lower after 165 and 195 min ( $P < 0.01$ ), but higher ( $P < 0.01$ ) from 405 to 495 min after the meal.

Carbohydrate, fat, and protein oxidation 4.5 and 9 h after the meal are shown in Fig. 3. It can be seen that the amino acid oxidation that occurred during 9 h (117–132 kcal, or ~16% of total energy expenditure) was approximately equal to the amount of protein provided in the meals and that the fat supplement did not noticeably alter the rate of protein oxidation. At 4.5 h, the total amount of carbohydrate oxidized was identical after the nonfat and LCT meals (~220 kcal), whereas it tended to

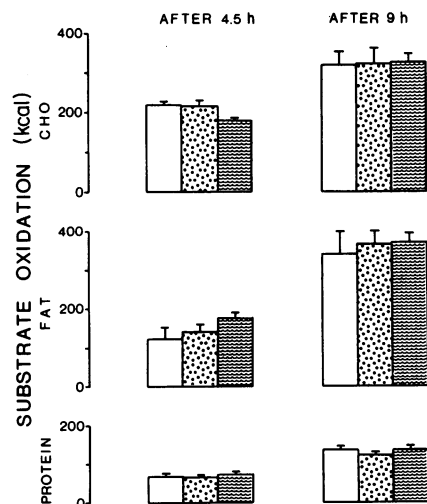


Figure 3. Carbohydrate (CHO), fat, and protein oxidation (kilocalories) 4.5 and 9 h after ingestion of the low fat (white), LCT- (circles), and MCT- (lines) supplemented breakfasts. (Means±SE;  $n = 7$ .)

be smaller after the MCT meal (180 kcal). However, by the end of the test, the amounts of carbohydrate oxidized were the same (322, 323, and 324 kcal, respectively). Total lipid oxidation was also similar after 9 h in each of the three tests (340, 367, and 370 kcal, respectively). The data thus show that the presence of fat in the meal did not promote fat oxidation. As shown in Fig. 4, the carbohydrate balances 9 h after the breakfast (approximately -23 kcal) were not influenced by the presence or absence of fat (LCT or MCT) in this meal. When fat was provided, the fat balance was positive ( $60 \pm 33$  and  $57 \pm 25$  kcal after the LCT and MCT meal, respectively), whereas it was  $-287 \pm 60$  kcal when the breakfast contained very few fatty ingredients. The energy balances were essentially the same as the fat balances.

## Discussion

In the present study, the technique of indirect calorimetry was employed to investigate the influence of dietary fat upon fat oxidation and short-term energy balance in humans. Indirect calorimetry provides a means to determine changes in the protein, fat, and carbohydrate contents of the body (inclusive of the intestinal tract's contents) (17) without requiring the tracing of the fate of ingested nutrients, which would be complicated by the existing multiple compartments and the nutrients' mixing with the flux of endogenous substrates. Using a ventilated hood, continuous monitoring of the subjects' respiratory exchange could be performed without undue stress over a period long enough to ensure extensive, if not complete, absorption and disposal of the ingested meals. During this 9-h period, the addition of fat to a meal of fixed carbohydrate and protein content failed to bring about any increase in fat oxidation.

Meals commonly provide 50–150 g of carbohydrate, but the physiological limits within which blood glucose levels must remain to avoid glucosuria or hypoglycemia permit changes in the body's free glucose pool of ~10 g only. It is not surprising, therefore, that carbohydrate intake elicits marked metabolic and

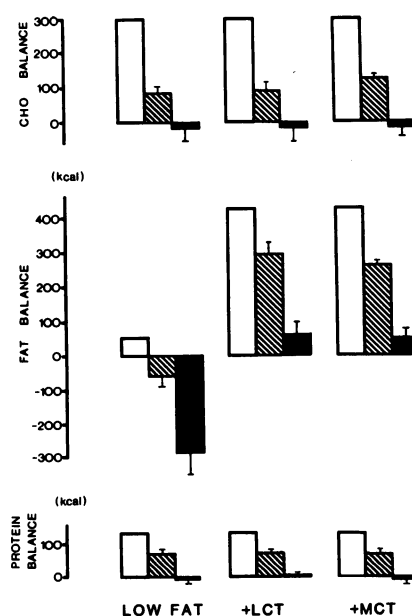


Figure 4. Carbohydrate (CHO), fat, and protein balances immediately after (white), 4.5 h after (hatched), and 9 h after (black) ingestion of the low fat LCT- or MCT-supplemented test meals. (Means±SE;  $n = 7$ .)

hormonal responses. A prompt increase in the rate of glucose oxidation occurs, but the carbohydrate loads ingested usually far exceed the amount of glucose that may be oxidized during the postprandial hours. Most of the ingested glucose absorbed from the gut must therefore be stored, primarily in the form of glycogen (18–20). In the present study, 1.5 h after ingesting 75 g of carbohydrate, the contribution made by glucose to overall energy generation nearly doubled, increasing from 32% to 57%. The subsequent gradual decrease in glucose oxidation as the carbohydrate supplied by the meal was being used up (cf. RQ in Fig. 2) further illustrates how closely carbohydrate oxidation is adjusted to its availability.

The response to protein ingestion has been less amenable to study because its effects on urinary nitrogen excretion are delayed and because the mechanisms involved in the coordinated disposal of all the different amino acids provided by dietary protein are complex. But it is established that protein oxidation is greater during the day than during the night (21), which indicates that the use of amino acids for energy production is related to availability, as in the case of carbohydrate.

The intestinal absorption of fat is delayed, as it requires emulsification with bile salts, followed by partial hydrolysis. Triglycerides are then reformed in the intestinal cells and secreted into the lymph in the form of chylomicrons (22). For example, the levels of primary chylomicron particles in plasma were found to peak 6 h after the ingestion of 250 g of butter fat or corn oil (23), a much greater amount than the 45 g of fat present in our test meals. The fatty acids transported by the chylomicrons are made available to the organism by lipoprotein lipase. This enzyme is most active in the vascular and extracellular compartments of adipose tissue (24), where most of the FFA formed are promptly taken up by the adipocytes, esterified to triglycerides, and stored. The amount of fatty acids that avoids captation by adipose tissue appears to be small, as it is insufficient to compensate for the decrease in FFA release brought about by the insulin secreted in response to the carbohydrate usually consumed along with fat (see Fig. 1).

The absorption and transport of MCT occurs by other mechanisms than those involved for the naturally predominant long-chain fatty acids (25). Instead of being incorporated into chylomicrons, medium-chain fatty acids appear in the portal blood as FFA bound to albumin (26), the form in which all fatty acids are typically made available to tissues for energy generation. Thus, they would be expected to be more readily used for energy generation, as well as less effectively trapped by adipose tissue where their esterification and storage may in fact require prior conversion to fatty acids with longer carbon chains (27). Indeed, the lesser increase in the RQ observed early after the MCT meals (Fig. 2) reveals that ingestion of MCT promoted fat oxidation in the postprandial phase, which resulted in the sparing of some 10 g of carbohydrate (Fig. 5). With this small increase in the amount of glycogen remaining available at the start of the second half of the 9-h period of observation, the RQ stayed higher than after the other two test meals (Fig. 2), which illustrates that the blend of substrates used by the body for energy generation is influenced even by minor differences in its glycogen stores.

We have been interested in characterizing differences in the roles that dietary carbohydrate and dietary fat may exert in the regulation of the body's fuel economy and in the achievement of energy balance and weight maintenance (15, 20). We therefore sought to compare the relative metabolic impacts of fat and of carbohydrate ingestion. Considered in this context, the weakness or absence of a metabolic response to dietary fat is an important,

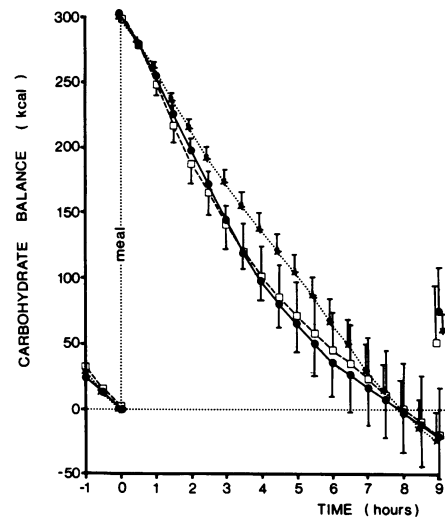


Figure 5. Changes in the carbohydrate balances (kilocalories) as a function of time, before and after a low fat breakfast (●), an LCT-supplemented breakfast (□), and an MCT-supplemented breakfast (▲). The carbohydrate balance at zero time (just before the ingestion of the meal) was taken to be zero. The three free-standing points at 9 h represent the carbohydrate balance had carbohydrate oxidation continued at the same rate as before the meals. (Means  $\pm$  SE;  $n = 7$ .)

rather than an uninteresting, observation. Knowing that carbohydrate intake exerts considerable metabolic leverage, it was necessary to compare meals containing fixed amounts of carbohydrate, while their fat content was varied. This implied a departure from the usual procedure of comparing meals with equivalent caloric contents, because an exchange of fat for carbohydrate calories would not afford the possibility to determine whether observed differences in the metabolic response are due to the decrease in carbohydrate intake or to the increase in fat consumption.

Fig. 3 illustrates the amounts of carbohydrate, fat, and protein oxidized over 4.5 and 9 h after the three types of test meals. The complete failure of common dietary fat (LCT) to alter the composition of the metabolic fuel mix oxidized at any time during the 9-h period after breakfast is quite remarkable and demonstrates how effectively LCT are channeled toward storage. The implications of this observation become most vividly apparent when substrate balances are considered, i.e., the amounts of substrates ingested minus the amounts of substrates oxidized (Fig. 4). At the end of the afternoon, the carbohydrate and protein balances were close to zero on each occasion. Fat oxidation was the same after the low fat meal or the meals supplemented with triglycerides; however, the fat balances differed markedly, depending on whether fat was omitted or added to the breakfast, i.e.,  $-287$  vs.  $+60$  kcal, or  $+57$  kcal ( $P < 0.001$ ), respectively. The subjects' energy balances,  $-310$  vs.  $+49$  kcal ( $P < 0.001$ ), or  $+32$  kcal ( $P < 0.001$ ), were in fact essentially equal to their fat balances. This suggests that fat intake is a particularly important factor in determining short-term energy balance.

The composition of the fat-containing breakfasts was designed to be approximately equal to the composition of the fuel mix oxidized by the subjects in the postabsorptive state preceding the ingestion of the test meals. If dietary carbohydrate and fat were to exert equivalent impacts on fuel metabolism, the subjects' RQ would not be altered by such meals. Therefore, the marked increases in the RQ induced by these meals (Fig. 2) reveal how unequal the impacts of dietary carbohydrate and of dietary fat on metabolism are. These RQ responses demonstrate by them-

selves that the organism tends spontaneously to maintain carbohydrate balance, and that this tendency is not affected by what may happen to the body's fat balance.

In some individuals, fat intake causes satiation, but this phenomenon appears to be highly variable among different subjects. The mechanisms that may be involved in this are not well understood but are probably related to the presence of fat in the intestine, its absorption, and its transport, rather than to a gain in the body's fat stores. Indeed, the amounts of fat consumed in meals are so small by comparison to the massive reserve of fat stored in adipose tissue that a response serving to adjust food and/or fat intake can hardly be imagined to arise as a result of short-term changes in the adipose tissue's fat mass. Furthermore, it appears from our data that if fat should contribute to induce satiety, this would not be mediated by a carbohydrate-sparing effect. While the 9-h period during which these metabolic studies were conducted can be presumed to be long enough to allow extensive, if not nearly complete absorption of the dietary fat consumed (23), the possibility remains that an increased fat oxidation in response to dietary fat intake might become evident at a later time. However, this would be expected only if some of the recently consumed fat were still not absorbed at that time, or if it were held in a transitional pool, and thus not yet "lost" in the enormous adipose tissue triglyceride pool.

The results of the present experiments are relevant to the problem of weight maintenance. It seems reasonable that conscious efforts to avoid excessive food intake should be expected to be most needed, and most worthwhile, when directed at that sector of metabolism where metabolic regulation is least effective in assuring the maintenance of substrate balance. Our investigations show that this is the case for fat, which suggests that deliberate efforts to facilitate weight control may be most likely to be effective in the long run when they serve to limit fat intake. This provides a metabolic rationale in support of current recommendations to counter the trend toward obesity by reducing the diet's fat content, and, thereby, the caloric density of the foods consumed (28).

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