Metabolic effects of a mixed and a high-carbohydrate low-fat diet in man, measured over 24 h in a respiration chamber

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1. The relation between dietary carbohydrate:lipid ratio and the fuel mixture oxidized during 24 h was investigated in eleven healthy volunteers (six females, and five males) in a respiration chamber. Values of the fuel mixture oxidized were estimated by continuous indirect calorimetry and urinary nitrogen measurements.

2. The subjects were first given a mixed diet for 7 d and spent the last 24 h of the 7 d period in a respiration chamber for continuous gas-exchange measurement. The fuels oxidized during 2·5 h of moderate exercise were also measured in the respiration chamber. After an interval of 2 weeks from the end of the mixed-diet period, the same subjects were given an isoenergetic high-carbohydrate low-fat diet for 7 d, and the same experimental regimen was repeated.

3. Dietary composition markedly influenced the fuel mixture oxidized during 24 h and this effect was still present 12 h after the last meal in the postabsorptive state. However, the diets had no influence on the substrates oxidized above resting levels during exercise. With both diets, the 24 h energy balance was slightly negative and the energy deficit was covered by lipid oxidation.

4. With the high-carbohydrate low-fat diet, the energy expenditure during sleep was found to be higher than that with the mixed diet.

5. It is concluded that: (*a*) the composition of the diet did not influence the fuel mixture utilized for moderate exercise, (*b*) the energy deficit calculated for a 24 h period was compensated by lipid oxidation irrespective of the carbohydrate content of the diet, (*c*) energy expenditure during sleep was found to be higher with the high-carbohydrate low-fat diet than with the mixed diet.

Adaptation of energy expenditure to various energy intakes has been often studied in man (Passmore *et al.* 1955; Durnin & Norgan, 1969; Danforth *et al.* 1978; Garrow, 1978). Dauncey (1980) has recently shown that over-eating (or under-eating) for only 1 d induces changes in energy expenditure, and that the resting metabolic rate measured 14 h after the last meal is affected by the previous day's energy intake. However, the relation between dietary composition and the fuel mixture in 24 h has received less attention. The aim of the present study was to investigate the fuel mixture oxidized over 24 h when modifying dietary carbohydrate:lipid.

A comparison of the nutrients ingested and the fuel mixture oxidized in 24 h gives the 24 h nutrient balance. Alternatively the relation between nutrient intake and the fuels oxidized can be studied by comparing the mean respiratory quotient (RQ) measured over 24 h with the mean food quotient (FQ), i.e. the ratio, volume of CO_2 produced:volume of O_2 consumed for the combustion of the energy intake (Flatt, 1978). The FQ of a usual balanced diet is approximately 0·85, which corresponds to an energy partition of approximately 45, 40 and 15% for carbohydrates (CHO), fat and protein respectively. When the mean FQ of the 24 h energy intake is similar to the subject's mean 24 h RQ, the fuel mixture oxidized corresponds to the CHO-fat composition of the diet. When the 24 h RQ is smaller than the 24 h FQ, the amount of lipid oxidized is larger than that ingested, i.e. there is a net endogenous lipid oxidation. Since this is the aim when treating obesity, it is of interest to study conditions which induce a 24 h RQ which is lower than the 24 h FQ.

In the present study two isoenergetic diets were given to healthy young adults for 1 week, one with a high FQ (high-CHO low-fat diet; HCLFD), and the other with a medium FQ

Table 1. *Composition of the diets (g/d), food quotient during (a) the 7 d experimental period and (b) 24 h measurement period*

(Mean values with their standard errors)

Diet	Protein		Lipid		Carbohydrate		Energy intake (kJ/d)		Food quotient	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
					(*a*) 7 d period*					
Mixed (MD)	89	4	92	4	227	10	8730	358	0·845	0·002
High-carbohydrate low-fat (HCLFD)	89	3	12	0·4	383	15	8337	308	0·944	0·001
					(*b*) 24 h period					
Mixed (MD)	88	2	85	3	228	5	8653	176	0·849	0·003
High-carbohydrate low-fat (HCLFD)	88	3	9·5	1	406	1	8678	515	0·948	0·002

* Average of 7 d.

(balanced mixed diet; MD) in order to establish whether nutrient balance could be obtained under both diets, and to study whether the metabolic adaptation to the diet was still present 12 h after the last meal.

A further aim of the present study was to estimate the fuel mixture oxidized with both diets during a 2·5 h period of moderate exercise. Since prolonged exercise is known to stimulate fat oxidation (Armstrong *et al.* 1961; Ahlborg *et al.* 1974), we investigated whether subjects receiving a low-fat diet may have to oxidize more endogenous lipid during exercise than subjects receiving a larger amount of exogenous lipid.

MATERIALS AND METHODS

Subjects

Eleven healthy medical students (six females and five males) volunteered for the investigation. Their mean age was 23·6 ± 1·2 years and body-weight 62·6 ± 2·9 kg. All subjects were within ± 10% of their ideal body-weight according to the Metropolitan Life Insurance Tables (1959).

Diets

Two diets, MD and HCLFD were used to obtain the desired FQ of 0·84 and 0·94 respectively. Each type of meal was designed to obtain the required FQ. Food which had previously been frozen provided a fixed amount of energy (4173 ± 133 kJ/d) for lunches and dinners, and additional energy intake was chosen *ad lib.* by the subjects for breakfast and snacks. The nutrient compositions of the frozen meals were obtained from the manufacturer and food tables (Souci *et al.* 1979) were used to calculate the composition of breakfasts and snacks. Atwater's coefficients were used to obtain the metabolizable energy content of all the meals (Atwater & Bryant, 1899).

Experimental procedure

Each subject was first given diet MD for 7 d (Table 1). Most of them had their lunch at the Institute, whereas breakfast, snacks and the evening meal were always taken at home. Subjects spent the last 24 h of the diet period in a respiration chamber for continuous

gas-exchange measurements. At 2 h after entering (at 08.00 hours) the respiration chamber, subjects were asked to perform a light muscular exercise i.e. pedalling at 30 W (184 kg-m/min) for 2·5 h on a bicycle ergometer (Quinton Instruments). For the remainder of the day, subjects were free to do as they wished e.g. working at the desk, sitting or lying on the bed, watching television, but no intense physical activity (e.g. gymnastics) was allowed. The subjects received their meals when in the respiration chamber (Table 1) through an air-tight communication compartment. On the morning following the 24 h spent in the chamber, subjects had their resting metabolic rate (RMR) measured for 1·5 h. After an interval of 2 weeks from the end of the period on diet MD, the same subjects were given diet HCLFD for 7 d (Table 1). The experimental procedure was exactly the same as that described for the period on diet MD.

Measurements

RMR open-circuit indirect calorimetry with a ventilated hood. The head of the subject is inserted into a transparent ventilated hood, the air-tight cloth of which is secured around the subject's neck. Outside air is drawn continuously through the hood at a constant rate (range 25–35 l/min according to the subject) by a ventilator inserted into the outlet tube. The flow rate of air leaving the hood is measured using a pneumotachograph and a differential manometer (Digital Pneumotachograph, Model 47303 A, Hewlett Packard). A fraction of the outflowing air is continuously sampled and its O_2 and CO_2 concentrations are measured using a thermomagnetic O_2 analyser (Magnos 2T, Hartmann and Braun) and an infra-red CO_2 analyser (Uras 2T, Hartmann and Braun, Frankfurt, Germany).

With open circuit indirect calorimetry, it is important for calculating $\dot{V}_{O_2}$ to take into account the fact that the flow rate of air at the inlet is different to that at the outlet, due to the fact that the RQ is usually less than 1·0. The following equation was used to obtain $\dot{V}_{O_2}$:

$$\dot{V}_{O_2} = \frac{1}{1-F_{in,\,O_2}} [\dot{V}_{out}(\Delta F_{O_2} - \Delta F_{CO_2} \times F_{in,\,CO_2})], \quad (1)$$

where $F_{in,\,O_2}$ is the fraction of O_2 at the inlet, $\dot{V}_{out}$ is the flow of outgoing air, $\Delta F_{O_2} = F_{in,\,O_2} - F_{out,\,O_2}$, $F_{out,\,O_2}$ is the fraction of O_2 at the outlet, $\Delta F_{CO_2} = F_{out,\,CO_2} - F_{in,\,CO_2}$, $F_{out,\,CO_2}$ is the fraction of CO_2 at the outlet, $F_{in,\,CO_2}$ is the fraction of CO_2 at the inlet.

The CO_2 production rate was calculated with the following equation:

$$\dot{V}_{CO_2} = \dot{V}_{out} \times \Delta F_{CO_2}. \quad (2)$$

Values of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ were averaged for 5 min periods.

24 h Energy expenditure (24 EE): respiration chamber. We have constructed a respiration chamber (5 m long, 2·5 m wide and 2·5 m high) which is part of an open circuit ventilated indirect calorimeter. The O_2 and CO_2 concentrations are continuously measured in the outflowing air using the same analysers as those described for the hood system, and the flow rate of air leaving the chamber is measured by a pneumotachograph and a differential manometer (Digital Pneumotachograph, Model 47303 A, Hewlett Packard). Provided that the expiratory gases mix rapidly with the air in the chamber, the concentrations of $O_2 + CO_2$ at the outlet are equal to their mean concentrations in the chamber. To fulfil this condition, mixing of expiratory gases with air is accelerated using a blower inside the chamber, and the response time to a step change is approximately 2 min. This fast response makes it unnecessary to take this delay into account in the calculations of substrate oxidation rates and energy expenditure.

For the calculation of $\dot{V}_{O_2} + \dot{V}_{CO_2}$ with the chamber, it is necessary to make allowance for two terms, i.e. the difference between the flow of $O_2 + CO_2$ in and out, and the rate at

which the O_2+CO_2 concentrations change in the chamber. The equation of McLean & Watts (1976) was used:

$$\dot{V}_{O_2} = \frac{1}{1-F_{in,O_2}}\left[\dot{V}_{out}(\Delta F_{O_2} - \Delta F_{CO_2} \times F_{in,O_2}) + V\left(\frac{dF_{out,O_2}}{dt} - \frac{dF_{out,CO_2} \times F_{in,O_2}}{dt}\right)\right]. \quad (3)$$

The symbols are the same as in equation (1); V is the volume of air in the respiration chamber (STPD).

$\dot{V}_{CO_2}$ was calculated using the following equation:

$$\dot{V}_{CO_2} = \dot{V}_{out}\,\Delta F_{CO_2} + V\frac{dF_{out,CO_2}}{dt}. \quad (4)$$

For both the hood and the chamber, calculations of energy expenditure and the metabolic mixture oxidized were performed using the equations of Consolazio *et al.* (1963). These calculations necessitate the measured values of $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$ and urinary N.

The accuracy of the measurements of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ was controlled by burning butane inside the chamber. In fourteen calibration tests of one hour each, the measured values of $\dot{V}_{O_2}$ were $100{\cdot}44 \pm 0{\cdot}34\%$ of the real value obtained by weighing the amount of butane which had been burnt and by stoichiometric calculation of the volume of O_2 utilized; the measured values of $\dot{V}_{CO_2}$ were $99{\cdot}64 \pm 0{\cdot}50\%$ of the real $\dot{V}_{CO_2}$. Calibration of the hood system gave similar results.

Physical activity. Spontaneous physical activity was monitored using a radar system based on the Doppler effect (Schutz, Ravussin *et al.* 1981) and physical activity was expressed as a percentage of the time during which the subject was moving. All measurements were averaged for 30 min periods.

Urine samples. Urine was collected during the 24 h EE and RMR measurement periods and analysed for urinary N using the Kjeldhal method (Hawk, 1947). The subjects voided before entering the chamber and urine was collected before the exercise, 1 or 2 h after the exercise, and then at approximately 6 h intervals. Urinary glucose was analysed during the 24 h EE measurement using a qualitative hexokinase method (Gluketur-test^R; Boehringer).

Blood samples. Blood samples were obtained in the postabsorptive state before and after the 7 d diet periods. To obtain blood samples in the respiration chamber, a specially-designed arrangement was used; the investigator entered the room through an air-tight communication compartment breathing through a mask connected to the outside air to avoid altering the composition of the air in the chamber. Blood samples were analysed for the following: blood glucose using the hexokinase method (Slein, 1965), plasma immunoreactive insulin (IRI) (Herbert *et al.* 1965), plasma free fatty acids (FFA) using the Dole & Meinertz (1960) extraction and determination of Ho (1970) as modified by Heindel *et al.* (1974).

Statistical analysis. Results are given as mean values with their standard errors. Values were analysed using Student's t test for paired values, each subject being his own control.

RESULTS

Energy balance

The mean energy intake and expenditure of the subjects during the 24 h period in the respiration chamber are presented in Table 2. The energy content of the two diets was almost identical; 24 EE was larger than intake for both diets, resulting in a negative energy balance. RMR values were not significantly different for the two diets, 272 ± 10 and 266 ± 9 kJ/h for diets MD and HCLFD respectively.

Table 2. *Energy intake (kJ/24 h), energy expenditure (kJ/24 h) during the 24 h measurement period, food quotient, mean respiratory quotient (RQ) measured during 24 h and during resting metabolic rate (RMR)*

(Mean values with their standard errors)

	Energy intake		Expenditure		Difference of the means		Food-quotient		RQ during the 24 h of measurement		RQ (RMR)	
Diet	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Mixed (MD)	8653	176	10074	392	−1421*	443	0·849	0·003	0·795†§	0·007	0·800‡§	0·008
High-carbohydrate low-fat (HCLFD)	8678	515	10591	502	−1913*	524	0·948	0·002	0·879†‖	0·005	0·856‡‖	0·007

* Difference of the means between energy intake and expenditure; MD *v.* HCLFD, was not statistically significant.
† Mean RQ during the experimental day; MD *v.* HCLFD ($P < 0·001$).
‡ Mean RQ (RMR); MD *v.* HCLFD ($P < 0·001$).
§ Comparison between mean RQ during the 24 h measurement period and mean RQ (RMR) for the MD, was not statistically significant.
‖ Comparison between mean RQ during the 24 h measurement period and mean RQ (RMR) for diet HCLFD, ($P < 0·05$).

Table 3. *Substrates ingested and oxidized (g/24 h) during the 24 h measurement period for the mixed diet (MD) and the high-carbohydrate low-fat diet (HCLFD) and substrates oxidized during resting metabolic rate (RMR) (mg/min) for diets MD and HCLFD*

(Mean values with their standard errors)

Diet		Ingested		Oxidized		Difference between the means	1 h RMR (mg/min)	
		Mean	SE	Mean	SE		Mean	SE
MD	Protein	88	2	77*	6	+11	50§	9
	Lipid	85	3	160†	11	−75	60‖	7
	Carbohydrate	228	5	165‡	15	+63	69¶	8
HCLFD	Protein	88	3	71*	7	+17	45§	8
	Lipid	9·5	1	90†	8	−80·5	49‖	4
	Carbohydrate	406	1	359‡	15	+47	117¶	11

* Protein oxidized during 24 h; MD *v.* HCLFD, not significant.
† Lipid oxidized during 24 h; MD *v.* HCLFD, $P < 0{\cdot}001$.
‡ CHO oxidized during 24 h; MD *v.* HCLFD, $P < 0{\cdot}001$.
§ Protein oxidation rate during RMR; MD *v.* HCLFD, not significant.
‖ Lipid oxidation rate during RMR; MD *v.* HCLFD, $P < 0{\cdot}005$.
¶ CHO oxidation rate during RMR; MD *v.* HCLFD, $P < 0{\cdot}001$.

FQ and RQ

The mean values for FQ, RQ during the 24 h EE, and RQ during RMR measurements (obtained after a 12 h postabsorptive period) are also presented in Table 2. Mean RQ values for 24 h were always significantly lower than FQ values ($P < 0{\cdot}05$). RQ measured during 24 h or during RMR were both significantly higher with diet HCLFD than with diet MD.

Substrate balance

In Table 3, substrate oxidation rates during the 24 h in the respiration chamber are compared with the substrates ingested. Protein oxidation rates were not affected by the type of diet eaten. On the other hand, lipid and CHO oxidation rates were significantly different ($P < 0{\cdot}001$), depending on the kind of diet consumed. When diet MD was fed, subjects oxidized on average 160 ± 11 g lipid and 165 ± 15 g CHO during the 24 h in the chamber, whereas when diet HCLFD was fed subjects showed an almost twofold increase in CHO oxidation rate 359 ± 15 g with a concomitant decrease in lipid utilization 90 ± 8 g. Whichever the diet consumed, lipid utilization was always larger than lipid intake, whereas the amount of CHO oxidized was smaller than CHO intake.

The composition of the diet also influenced the substrates utilized during RMR, i.e. more CHO was oxidized with diet HCLFD than with diet MD; the reverse was true for the lipid oxidation.

Fuel utilization during exercise

The energy expended and the substrates oxidized during muscular exercise are presented in Table 4. In this instance, the substrates oxidized represent the net increase above the mean oxidation rates measured during the 2 h period preceding the exercise in the chamber. The proportion of lipid and CHO oxidized during the exercise was not influenced by the composition of the diet consumed, and was almost identical in the two experimental conditions. The proportion of protein in the fuel oxidized during the exercise was very small and represented approximately 1 g for each diet.

Table 4. *Total energy expenditure (kJ/2·5 h) and total lipid+carbohydrate oxidized (g/2·5 h) during the 2·5 h exercise for the mixed diet (MD) and the high-carbohydrate low-fat diet (HCLFD). Energy expenditure (kJ/2·5 h) and lipid+carbohydrate oxidized (g/2·5 h) above resting values during the 2·5 h exercise. Resting values were obtained by averaging the values obtained during the 2 h period preceding the exercise in the chamber*

(Mean values with their standard errors)

	Mixed diet		High-carbohydrate low-fat diet		Statistical significance
	Mean	SE	Mean	SE	
Energy expenditure (total)	2599	77	2606	77	NS
Energy expenditure (above resting)	1887	78	1785	61	NS
Lipid oxidation (total)	39	3	30	2	$P < 0.02$
Lipid oxidation (above resting)	25	2	23	2	NS
Carbohydrate oxidation (total)	66	7	87	5	$P < 0.005$
Carbohydrate oxidation (above resting)	56	7	55	5	NS

NS, not significant.

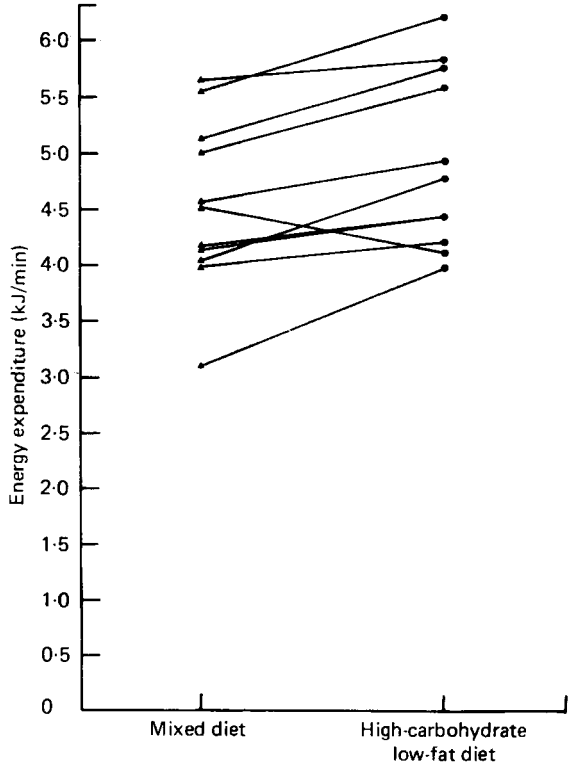

Fig. 1. Comparison of energy expenditure (kJ/min) during sleep only. Mixed diet (▲); high-carbohydrate low-fat diet (●). Statistical significance: $P < 0.005$.

Table 5. *Blood glucose (mmol/l) free fatty acids (FFA) (μmol/l) and immunoreactive insulin (IRI) (μU/ml) before and after the 7 d measurement period, before, during and after the exercise period*

(Mean values with their standard errors)

		Blood glucose (mmol/l)		FFA (μmol/l)		IRI (μU/ml)	
	Diet	Mean	SE	Mean	SE	Mean	SE
Postabsorptive state before the experiment (08.00 hours)	MD	4·7	0·1	559	92	16·7	1·7
	HCLFD	4·9	0·1	627	101	18·9	2·2
Postabsorptive state after experimental week (08.00 hours)	MD	4·7	0·1	684	109	15·1*	1·5
	HCLFD	4·7	0·1	530	75	19·8*	1·8
Before the exercise (10.00 hours)	MD	4·4	0·2	568	72	15·8	2·5
	HCLFD	4·0	0·2	418	168	18·0	1·9
After 1·5 h exercising (11.30 hours)	MD	4·2	0·1	1002	139	9·1	1·1
	HCLFD	4·3	0·1	928	103	10·8	1·4
End of exercise (2·5 h) (12.30 hours)	MD	4·3	0·1	1625	169	10·4	1·9
	HCLFD	4·4	0·1	1538	164	12·6	1·6
Postabsorptive state after the 24 h of measurement and RMR (09.00 hours)	MD	4·8	0·1	460	60	12·3†	1·0
	HCLFD	4·8	0·1	369	41	15·3†	1·3

MD, mixed diet; HCLFD, high-carbohydrate low-fat diet.
* IRI, after the experimental week; MD *v.* HCLFD ($P < 0·05$).
† IRI, after the 24 h of measurement; MD *v.* HCLFD ($P < 0·02$).

Energy expenditure during sleep

Total energy expenditure during the period of sleep is presented in Fig. 1. Only those periods where the mean spontaneous physical activity (measured by the radar system) was between 0 and 1% for each 30 min period were considered sleeping periods; the average sleeping period was 6 h 18 ± 23 min with diet MD and 6 h 19 ± 18 min with diet HCLFD. In addition, the times at which the subjects went to sleep were 00.22 hours (± 21 min) on the MD diet and 00.18 hours (± 21 min) on the HCLFD. Energy expenditure was significantly lower with diet MD than with diet HCLFD by an average of 148 ± 40 kJ for the sleeping period ($P < 0·005$).

Blood indices

There was no difference between values for postabsorptive blood glucose on day 1 and after 1 week on diets MD and HCLFD; nor was there any difference in values for postabsorptive blood glucose between the two diets after 7 d (Table 5). During exercise, blood glucose decreased slightly below fasting levels for both diets.

Postabsorptive plasma FFA levels were similar after 1 week for both diets (Table 5). During exercise, plasma FFA levels rose similarly with the two diets.

Postabsorptive insulin levels were significantly higher with diet HCLFD than with diet MD after 1 week on the diet ($P < 0·05$) and after the 24 h measurement ($P < 0·02$), but it remained within the physiological range. During exercise there was a similar decrease in insulin levels with both diets. None of the subjects had glucose in their urine with either of the dietary treatments.

DISCUSSION

The purpose of this investigation was to study the change in the fuels oxidized at rest and during exercise after 7 d on a high-CHO low-fat diet. The calculation of substrate oxidation rates from respiratory exchange measurements and urinary N only give estimates and the method has limitations. For example, the assessment of protein oxidation from urinary N excretion is not a precise procedure and must only be considered as an index of the protein oxidation. Reactions of deamination of glutamine yielding NH_4^+, or deamination of other amino acids in the process of gluconeogenesis do not correspond to protein oxidation. However, uncertainty about protein oxidation does not significantly affect the calculation of metabolic rate or that of carbohydrate and lipid oxidation. It is well known that changes in ventilation (hyper- or hypoventilation) result in changes of RQ which are not related to the fuels oxidized. However, integration of the values of $\dot{V}_{CO_2}$ and $\dot{V}_{O_2}$ during periods of 30 min for 24 EE measurements and allowance for a 15 min period of adaptation to the hood for RMR measurement eliminate these respiratory influences.

The composition of the fuel mixture oxidized was clearly influenced by that of the diet (Table 3) even though plasma levels of glucose and FFA were similar. Metabolic adaptation to diet HCLFD was not only present during the 24 h measurement period, but it persisted in the morning postabsorptive state, 12 h after the last meal.

During the 24 h test period, the subjects who could eat snacks *ad lib.* were nevertheless in negative energy balance. This might be due to the somewhat artificial conditions of living in a closed chamber. It is interesting to note that with both diets MD and HCLFD: (1) the energy deficit was compensated by a similar amount of endogenous fat oxidation, (2) the energy provided for the exercise was approximately 50% CHO and 50% fat oxidation, (3) a positive CHO balance was observed despite the energy deficit.

The results suggested that the composition of the diet had no influence on the extra energy oxidized to compensate a 24 h energy deficit: with both diets MD and HCLFD, the deficit was compensated by using endogenous fat. Diet HCLFD, which is known to maintain elevated CHO stores in liver (Hultman & Nilsson, 1971) and muscles (Bergström *et al.* 1967) did not favour CHO utilization for the 2·5 h exercise period at moderate intensity. The tendency to keep a positive CHO balance with both diets under conditions of negative energy balance may be of a transient nature. It is not possible repeatedly to store large amounts of CHO since the storage capacity of glycogen is limited (Hildes *et al.* 1949). However Passmore & Swindells (1963), Hermansen *et al.* (1967), Saltin & Hermansen (1967) and more recently Acheson *et al.* (1979) have supported the view that the capacity to store CHO in man might be larger than usually described (Cahill, 1970). In the present study, RQ of more than 1, indicative of net *de novo* lipogenesis, were not observed. Thus, despite a large CHO intake, there was no net gain of fat by lipogenesis. Furnass (1960), Passmore & Swindells (1963) and Acheson *et al.* (1979) also found limited net conversion of CHO into fat after large CHO intakes.

Numerous studies have dealt with the fuels oxidized during exercise in relation to ingested diets (Christensen & Hansen, 1939; Bergström *et al.* 1967; Pruett, 1970*a*). Pruett (1970*a*), Ahlborg *et al.* (1974) and Astrand & Rodhal (1970) showed that in low-intensity long-duration exercise, approximately half the energy is derived from fat and half from CHO. In the present study, we found a similar fuel distribution, and this was not affected by the adaptation to either diet HCLFD or diet MD. Ingestion of HCLFD did not promote CHO oxidation for exercise, and consequently lipids were not spared with diet HCLFD. Moreover, the changes in plasma FFA and IRI levels during exercise were similar with both diets as also reported by Pruett (1970*a*, *b*). These results confirm the fact that prolonged exercise at low intensity is performed using body stores of both fat and CHO, provided that CHO stores are sufficient. Only when CHO stores have been previously depleted

(Ravussin *et al.* 1979) does fat oxidation become more important than CHO metabolism. The present results confirm that it is beneficial to include moderate physical activity in a weight maintenance programme in order to favour fat oxidation as suggested by Flatt (1978) & Nelson (1978). However, depletion of fat stores over a few hours of exercise does not necessarily imply weight loss over several days.

With similar 24 h energy intake, there was a tendency to expend more energy with diet HCLFD than with diet MD, but the difference was not significant over 24 h (Table 2). However, when computing the energy expenditure during the sleeping time, the subjects given diet HCLFD expended significantly more energy than when receiving diet MD. It is not possible to explain the lower energy expenditure due to diet MD either by an acclimatization to the respiration chamber, since diet MD was fed first, or by a difference between the total time asleep or by a difference between the times at night when the subjects went to sleep. The reason for the higher energy expenditure during sleep with diet HCLFD is unclear. Recently, the effects of CHO on sympathetic activity have been reviewed by DeHaven *et al.* (1980). They reported that underfeeding in humans or rats is accompanied by a decreased sympathetic activity which is primarily due to low dietary CHO rather than to a reduction in total energy intake. Furthermore, Landsberg & Young (1978) described a rise in norepinephrine turnover in rats given a high-sucrose diet. We have recently observed increased diurnal urinary catecholamine excretion in subjects receiving a high-CHO diet (Schutz, Acheson *et al.* 1981) but there was no change in urinary catecholamine during the night. Thus, whether an increase in sympathetic activity might explain the elevated energy expenditure observed with diet HCLFD remains to be established.

In conclusion, dietary composition markedly influenced the substrates oxidized during 24 h, and this effect was still present 12 h after the last meal in the postabsorptive state. The fuel mixture used for prolonged exercise was not modified by the previously ingested diet, 50% of the energy being derived from fat and 50% from CHO with both diets MD and HCLFD. Finally, energy expenditure during sleep was found to be higher with diet HCLFD than with diet MD.

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