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ORIGINAL CONTRIBUTION

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Nutrient oxidation and metabolic rate as affected by meals containing different proportions of carbohydrate and fat, in healthy young women

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Abstract The amount and composition of food eaten influence body weight regulation, which requires that, in the long term, energy intake matches energy expenditure and that the oxidation rate to be equal to intake for each and separate nutrients. The aim of this study was to examine the influence of two liquid formulas with different macronutrient composition, a high carbohydrate (HC) meal as compared to a high fat (HF) meal, on substrate oxidation and on thermic effect of food (TEF). Eighteen lean and healthy women which were fed a HC diet during the 3 preceding d were studied for a further 4 h after meal intake. The test meals provided fixed energy intake and whose calculated FQ were 0.77 for HF meal, and 0.96 for HC meal. The mean NPRQs were higher ($P<0.01$) in the HC group than in the HF group, even with values greater than 1.00 indicating net lipid synthesis (NL), and which correlated with metabolic rate (MR) value ($P<0.05$), glucose ($P<0.05$), and heart rate (HR) values. Carbohydrate (CHO) oxidation was higher with the HC than with HF meal ($P<0.01$) and correlated with the MR ($P<0.05$). Protein oxidation rate rose above baseline ($P<0.01$); this increase was accompanied by with a negative CHO balance. It is concluded that the change in fuel selection and the increase of TEF is mainly due to CHO intake and metabolism, respectively, and that surplus of dietary CHO of preceding days together with a large load of CHO can exceed the glycogen storage capacity and trigger NL.

Key words Nutrient oxidation – thermic effect of food – sympathetic nervous system – respiratory quotient – diet composition – energy balance – net lipid synthesis.

Introduction

There is scientific evidence that a high dietary fat intake is an important factor that may lead some people to obesity (1). However, whether dietary fat induced obesity is due to a decrease in metabolic rate, an increase in caloric intake, or a combination of both factors is still unclear. Weight maintenance requires that, in the long term, energy intake matches energy expenditure (2). Apart from energy balance, this also requires the oxidation rate to be equal to intake for each and separate nutrients (3). Thus, the ingestion of fat and carbohydrate may not be equivalent with regard to the regulation of body weight (4). Indeed, in the short term, adjustment of oxidation to intake must be more accurate for carbohydrates than for fats, because the body's glycogen stores (in terms of energy content) are 100 times smaller than its fat reserves (5, 6). Gains or losses of carbohydrate or fat can, thus, be expected to have different impacts on metabolism, on the regulation of food intake, and thereby on energy balance and body weight. Fat balance is poorly regulated, and fat deposition would depend on positive or negative energy balances (7). On the other hand, there is evidence of the involvement of sympathetic nervous system (SNS) activ-

ity in determining metabolic rate (8, 9). Thus, a higher SNS tone may either promote or reflect elevated levels of spontaneous physical activity or other thermogenic mechanisms influencing both energy balance and body composition (10).

Diet composition clearly has an important influence on body weight regulation. In response to acute changes in diet composition, it has been shown that humans subjects increase carbohydrate oxidation and total energy expenditure in response to excess fat (11).

Furthermore, short-term alterations in energy intake induce a number of metabolic and hormonal changes in humans. The most extensively documented consequence of surplus energy intake is the effect of excess carbohydrate on whole body fuel selection (12). Stimulation of carbohydrate oxidation after high carbohydrate (HC) meals is not surprising (13, 14), since increase in glucose and insulin concentrations suppress oxidation of fat and increase oxidation of carbohydrate (15). How carbohydrate replaces fat in tissue fuel mixture in the postabsortive state is not clear, since fat generally represents the major fuel consumed in the postabsortive state (16). Therefore, another area of uncertainty is whether surplus carbohydrate intake stimulates *de novo* lipogenesis in humans. Since the body glycogen stores are limited, their saturation by ingestion of meals rich in carbohydrate could lead to a high degree of satiety, to increased net lipogenesis, or both; although some of the surplus carbohydrate may be dissipated by adaptive thermogenic mechanisms (17-19).

In this study, we examined the influence of two liquid formulas with different macronutrient composition (HC meal compared with HF meal) on substrate oxidation, on nutrient induced thermogenesis, particularly on its relation with the sympathetic nervous system activity assessed by the heart rate (20), in subjects with initially saturated body glycogen stores by dietary means.

Subjects and methods

Subjects

Eighteen women aged 20 to 27 years old were studied. The mean body weight and height were 59.8 ± 1.6 kg and 163.9 ± 1.2 cm, the mean body mass index and body fat percentage 21.6 ± 0.4 and 24.1 ± 0.8 % respectively, and the mean fat-free mass in kg was 44.9 ± 1.3. The subjects were all healthy (evaluated by a physician) and took no medication for at least 1 month. Before entering the study, they were interviewed by a dietitian to assess their eating habits and to exclude subjects who potentially had an eating disorder.

Design

Height was measured to the nearest 5 mm with a wall-mounted anthropometer and weight was measured to the nearest 0.1 kg with a calibrated beam balance. Body mass index (Quetelet index) was calculated as weight (in kg) divided by the square of the height (in m). Body fat mass was calculate from triceps skinfold thickness (20), who was assessed twice with a caliper (Holtain Ltd. Crymech. United Kingdom) over the left triceps muscle, midway between the acromion and olecranon processes and the mean value was recorded (21, 22). All anthropometric determinations were performed at the beginning in the morning in the fasted state. Upon arriving about 8 am to the Metabolic Room by car or bus, after 12 h overnight fast and urinating, subjects rested supine for 30 minutes. A measurement of basal EE was started at 8:30 am (30 minutes after their arrived), and after the liquid meal was quickly ingested. Postprandial calorimetry measurements were performed for a further 4 h, every 30 minutes (12, 23). Heart rate was recorded (Partner, Cardiosport) at the same time as the calorimetric measurements were performed, when the subject was in lying and standing conditions. Capillary blood glucose was measured with a blood glucose test strip (Glucometer Elite) every 30 minutes during the first 2 h and each hour later, respectively. The urinary nitrogen excretion was measured by the Kjeldahl method in the postabsortive state and during whole postprandial study (24).

The participants received detailed dietary instructions in order to ensure that their food intake for the 3 d preceding both test meals (high carbohydrate and high fat diets) would result in a relatively high carbohydrate diet. They therefore were asked to consume a virtually fat-free meal the evening before the study and were given instructions to achieve this. Also, for the 3 d preceding each test, the subjects maintained their usual activity but were told to refrain from strenuous physical exercise (25).

Test meal

The test meals were two different liquid formulas providing fixed energy intake for all subjects. The composition of the high fat (HF) and high carbohydrate (HC) meals is summarized in Table 1. Diet composition was calculated from manufacturer specifications. The food quotient (FQ) was calculated as the following equation (26,27):

$$\mathbf{FQ} = (\ \%\ \mathrm{CHO} \times 1.0 + \%\ \mathrm{Fat} \times 0.71 + \%\ \mathrm{Prot} \times 0.835) / 100$$

where carbohydrate (CHO), fat and protein (prot) are in kJ.

Gas exchange measurements

Respiratory exchange measurements were performed using a computerized open-circuit indirect calorimeter with a rigid transparent ventilated hood (Deltatrac,

Table 1 Energy content and fuel substrates distribution of the high fat (HF) and high carbohydrate (HC) liquid formulas, in kJ and in percentage of total energy

	HC meal composition in kJ	HC meal composition in %	HF meal composition in kJ	HF meal composition in %
Protein	368	18	364	17.5
Total carbohydrates		80	314	15
Sucrose	1087	52		
Lactose	577	28		
Fat	50	2	1404	67.5
Total	2082	100	2082	100
FQ		0.96		0.77

Datex-Engstrom, Finland). Oxygen consumption and carbon dioxide production were integrated over periods of 15-20 minutes. The urinary nitrogen values were used to calculate the nonprotein respiratory quotients (NPRQ) before and during the test. Carbohydrate and fat oxidation were calculated from the nonprotein oxygen consumption ($NPVO_2$), their relative oxidative proportions, as indicated by the NPRQ, and the amount of oxygen consumed per gram of substrate oxidized (15, 28, 29, 30). The calculations were performed as follows:

NPRQ = $NPVCO_2$ / $NPVO_2$
CHO oxidation (g/min) =
$NPVO_2$ x (NPRQ - 0.707)/ 0.293 x 0.746

Fat oxidation (g/min) =
$NPVO_2$ x (1 -NPRQ)/ 0.293 X 0.746
Protein oxidation (g/min) = N x 6.25 x 0.966

Metabolic rate (MR) was computed using the following equation:

MR (kJ/min) = 16.4 VO_2 + 4,5 VCO_2 - 3.4 N

where $NPVO_2$ is measured in liters per minute, 1.000 is the NPRQ for total carbohydrate (CHO) oxidation, 0.293 is the difference between 1.000 and 0.707; the RQ of 0.707 is the NPRQ for total fat oxidation; 0.746 is the number of liters of oxygen consumed per gram glucose oxidized (in the fasted state 0.829 concerning liver glycogenolysis origin of glucose) (18, 24, 31), 2.019 is the number of liters of oxygen consumed per gram triacylglycerol oxidized, N is the urinary nitrogen value in g/min (fasted and postprandial values), 6.25 is the conversion factor of nitrogen to protein, 0.966 is the number of liters of oxygen consumed per gram protein oxidized.

When the amount of *de novo* synthesized fatty acids exceeds those concomitantly oxidized, it can be reflected in NPRQ greater than 1 (28). In this case, the value obtained for carbohydrate oxidation is indicated by the amount of carbohydrate oxidized in addition to that utilized for lipid synthesis, and the following equations can be used to estimate the rate of net lipid synthesis ($L_{G,F}$) and glucose oxidation (G) (24):

$$\mathbf{L}_{G,F} = 1.67\ (VCO_2 - VO_2) + 1.92\ N$$
$$\mathbf{G} = 1.34\ (VCO_2 - 4.88\ N)$$

Thermic effect of food

The thermic effect of food (TEF) was calculated as the increase in energy expenditure above baseline for 4 h after the test meal and was expressed as a percentage of ingested energy (32, 33):

TEF = (postprandial EE - prepandrial EE) x time (min) x 100/ 2.100

The energy balances over the 4 h period were determined as energy intake minus energy expenditure, and substrate balances as substrate intake minus oxidation of each nutrient (5,7).

Statistical analysis

All data are presented as means ± SEM. Data on nutrient oxidation, RMR, blood glucose, and heart rate were analyzed by analysis of variance or Kruskall-Wallis test. Where appropriate, paired *t* test and nonparametric Mann-Whitney *U* test were used to establish significant differences between means. Associations between variables were tested by Pearson's correlation analysis. All statistical analysis were calculated using the Stat View 4.01 Non-FPU, and a level of $P < 0.05$ was considered to be statistically significant.

Results

Metabolic rate

The postabsortive or basal metabolic rate (BMR) was 3.99 ± 0.08 in kJ/min, which showed a statistically significant association with height (r=0.610, P<0.01), weight (r=0.651, P<0.01) and fat free mass (r=0.700, P<0.01). After the test meal, the resting metabolic rate (RMR) increased in both groups with no significant differences between the groups at final of study (Fig. 1). The RMR increased above fasting values during the first 30 minutes after intake in HC group and then started a slow fall to baseline values. This initial peak was less pronounced on HF group, which reached its maximum plateau at 2 h and was followed by a small decline toward the end of study, but showing statistical differences throughout the complete postprandial period. The thermic effect of both meals expressed as a percentage of the energy content of the load was lower in the HF group when compared with that in the HC group 30 minutes after intake, but the overall TEF was higher in the HF group (Table 2).

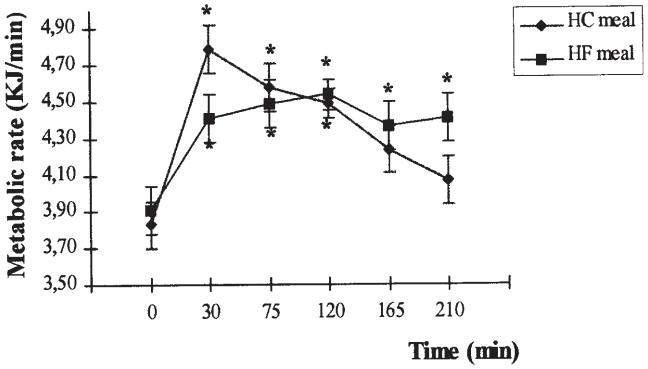

Fig. 1 Metabolic rate before (0 minutes) and after ingestion of different meals: high carbohydrate meal (HC meal) and high fat meal (HF meal). Results are the mean ± SEM; n=9. Statistical significance is indicated in the figure for differences above baseline values. ANOVA, * P<0.05.

Table 2 Effects of meals on Thermic Effect of Food (TEF), Non-Protein Respiratory Quotient (NPQR), Net Lipogenesis (NL), Blood Glucose and Heart Rate across the experimental time

	Time	TEF	NPQR	NL	Glucose (mg/dL)	Heart rate (beats/min)
Before carbohydrate meal (overnight fast)	8.30 a.m		0.959±0.029		90±1	62.5±1.9
Carbohydrate based meal	9.15–9.30 a.m	1.37±0.15	1.108±0.031*	0.031±0.01*	119±6 **	67.1±1.9
	10.00–10.15 a.m	2.70±0.27	1.084±0.030**	0.021±0.01	106±12	67.2±2.7
	10.50–11.10 a.m	3.80±0.42	1.051±0.026 *	0.015±0.01	99±4	64.4±2.0
	11.40–12.00 a.m	3.10±0.59	1.039±0.028	0.016±0.01	95±5	61.5±1.3
	12.30–13.00 p.m	4.03±1.34	0.990±0.031	0.005±0.003	86±1	60.3±1.3
Before fat meal (overnight fast)	8.30 a.m		0.852±0.026		84±4	65.3±3.9
Fat based meal	9.15–9.30 a.m	0.74±0.14†	0.888±0.019		90±6	66.2±2.9
	10.00–10.15 a.m	2.10±0.27	0.911±0.015		89±3	68.9±3.6
	10.50–11.10 a.m	3.63±0.58	0.906±0.017		84±6	60.9±7.8
	11.40–12.00 a.m	3.91±0.65	0.888±0.103		85±2	65.3±3.7
	12.30–13.00 p.m	5.02±0.99‡	0.903±0.017		87±2	66.6±3.9

NOTE: Values represent the mean ± SEM
***** Indicates significant differences as compared with baseline values (P<0.05)
****** Indicates significant difference as compared with baseline values (P<0.01)
† Indicates significant difference as compared with HC meal (P=0.0118)
‡ Indicates significant difference from carbohydrate based meal measured at the same postprandial time (P<0.01)

Respiratory quotient and substrate oxidation

The postabsortive respiratory quotient (NPRQ) was rather higher than usually found in both groups, in agreement with the high carbohydrate diets on the preceding days. Therefore, they were not significant differences between the groups (0.96 ± 0.03 for the HC group and 0.89 ± 0.02 for the HF group). The postprandial NPRQ were significantly higher on the HC group than on the HF group, which rose significantly (P<0.01) with the HC meal at the start of the experiment, even with values greater than 1 indicating the occurrence of net lipogenesis existence, and declined slowly about the third hour of study (Table 2). Moreover, the calculated values of net lipid synthesis from glucose were correlated with metabolic rate values (r=0.904, P<0.05). The HF meal produced no significant differences along the study (ANOVA, P=0.2033), despite the initial decrease in the NPRQ, 30 minutes after the intake, indicating an increase in fat oxidation in that moment.

Postprandial carbohydrate oxidation was higher with the HC than with HF liquid formula (P<0.01), except at the end of study (4 hours later) when there were no statistical differences. On the other hand, the carbohydrate oxidation was significantly associated with the MR (P<0.01) in HF group. Also, there was a significant increase in carbohydrate oxidation from the first to the fifth calorimetric determination (P<0.01) and a reciprocal decrease in fat oxidation with negative values in relation with a process of net lipid synthesis, in the HC group (Fig. 2A).

Fat oxidation was significantly lower after consumption of the HC than after the HF meal (P<0.01). The HF meal induced an initial increase in fat oxidation (P<0.01), but there were no subsequently statistical differences throughout the postprandial period (Fig. 2B). The protein rate oxidation increased significantly above the basal value in HF group (P<0.01). Therefore, this increase was supported with a negative carbohydrate balance (calculated as intake minus oxidation) in this group (Table 3). In contrast, after HC load the protein oxidation remained without significant changes above the fasted measurement (Fig. 2C).

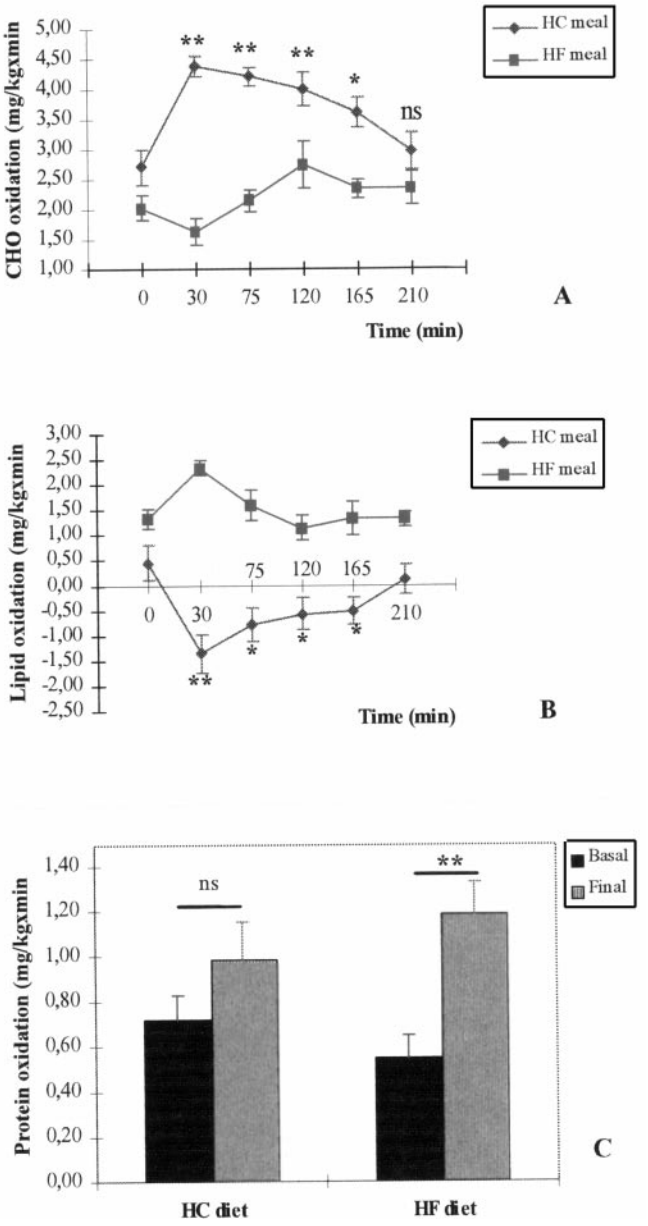

Fig. 2 Changes in carbohydrate (CHO) (A), lipid (B), and protein (C) oxidation induced by fat and CHO meals. Time 0 is the baseline value, and 30, 75, 120, 165, 210, 240 the postprandial measurements after intake. Basal is the postabsortive protein oxidation value and final, postprandial protein oxidation value. Results are mean ± SEM, n=9. NS: not significant; * P<0.05; ** P<0.01.

Blood glucose and heart rate

The blood glucose concentration increased above premeal values during the first 30 minutes after HC meal (P<0.01), followed by a gradual decline to basal levels toward the end of the study. In addition, this variable showed statistical associations with the lipogenesis rate (r=0.930, P<0.05) in this group. There were no changes in blood glucose after the HF meal; these values remained almost identical throughout the postprandial period (Table 2).

Heart rate (HR) asessments showed no statistical differences between the groups (Table 2). However, there were interesting statistical positive associations with the RMR (r=0.871, P<0.01), carbohydrate oxidation (r=0.839, P<0.01), net lipogenesis (r= 0.886, P<0.01), and negative with the fat oxidation (r= -0.846, P<0.01) in the HC group. On the other hand, there were no statistical associations between heart rate and RMR (r=0.727, P=0.1101), even carbohydrate (r=0.503, P=0.3379) or lipid oxidation (r=-0.079, P=0.8905), in the HF group.

Table 3 Total energy and substrate balances (kj) during the study with carbohydrate and fat based meals

	Net Energy Balance	Carbohydrate Balance	Fat Balance	Protein Balance
Carbohydrate based meal	1088.5 ± 40.2	845.6 ± 40.1	375.8 ± 146.7	162.6 ± 41.8
Fat based meal	1086.8 ± 24.7	-188.5 ± 33.0 **	657.1 ± 99.5	104.9 ± 36.8

NOTE: Values represent mean SEM.
** Indicates significant difference from carbohydrate based meal (P<0.01)

Discussion

NPRQ and substrate utilization

The current study investigated the metabolic responses to isoenergetic meals containing different proportions of carbohydrate and fat, after manipulation of glycogen stores three days preceding the test meal. These previous dietetic conditions led to higher fasting NPRQ than usually reported, between 0.80 and 0.84 (34). Thus, extra CHO was able to replace fat as the main substrate in the postabsortive state as has been found by others investigators (16, 18, 35). Since the influence of the last meal had essentially ended in the postabsortive state, the fasting NPRQ value probably gives an indication of the state of the body glycogen stores. Although the effect of the treatment on liver glycogen stores were not quantified, it has been reported than 3 d of a subsequent high carbohydrate diet induced concentrations two fold higher than baseline (7, 19, 36).

The most extensively documented consequence of surplus energy intake has been reported to be the effect of excess carbohydrate in whole body fuel selection. Several authors have reported that the addition of surplus CHO energy on the mixed diet results in nearly complete replacement of fat by carbohydrate in the whole body oxidation (11, 37). Likewise, in our study, with a meal containing a large load of simple dietary carbohydrate (lactose and sucrose), a rise of its own oxidation is observed. Stimulation of CHO oxidation is not surprising, since increased glucose concentrations suppress oxidation of fat and increases of CHO. The mean CHO balance was 845.6 ± 40.1 in the HC group, and this represents an important increase in glycogen stores if one considers that a positive CHO balance results almost exclusively in glycogen deposition.

The postprandial NPRQs greater than 1.00 observed in the HC group suggest a net lipogenesis and confirm that surplus of dietary CHO during the preceding days of study together with a large load of CHO, exceed the glycogen storage capacity and trigger *de novo* lipogenesis processes, which can become a major metabolic pathway for the disposal of excess glucose carbons. Other studies have shown that *de novo* lipogenesis does not contribute to increasing the body fat stores even when very large amounts of carbohydrate are consumed. Our data suggest that when glycogen stores became saturated, so that the only way of disposing of additional excess of carbohydrate is by fat synthesis in addition to maximal use of glucose for energy generation. The validity of indirect calorimetry for net lipogenesis determination has been addressed elsewhere by an algebraic approach with minimal assumptions about stoichiometries, by several authors (18, 24, 28). However, it is evident that lipogenesis can occur at NPRQs less than 1, when lipid oxidation is in excess of lipid synthesis (38). In this situation, the total amount of lipid synthesized cannot be measured as such but is “seen” or calculated as carbohydrate oxidation, i.e., the calculations are independent of the intermediate steps of glucose metabolism. Thus, overall substrate utilization can be calculated from respiratory exchange data.

The HC meal induces a fast increase in its own oxidation, which remained throughout nearly the complete postprandial period in high values. The change in fuel selection is controlled by carbohydrate intake, and when carbohydrate oxidation rises in response to intake there is a profound contarregulatory suppression of fat oxidation. This study extends observations on the oxidative hierarchy of macronutrient regulation and have in common with another studies the notion that availability of glucose, rather than free fatty acids (39), determines the relative contribution of the components of substrate oxidation (15, 40). This can be supported by the observation of the correlation between carbohydrate oxidation and blood glucose, and between net lipogenesis and glucose on the HC group. Other studies have examined the mechanism of the increase in CHO oxidation in response to overfeeding in more detail. In this way, Schwarz et al. (41) have examined the effect of 25% and 50% increases and decreases in CHO intake and reported a graded dose response in terms of carbohydrate oxidation mediated by increased hepatic glucose production, which stimulated moderate hyperinsulinemia. This decreased lipolysis and fatty acid availability, and the net effect was to increase glycogen stores and enhance the delivery of extracellular glucose, thus, favoring increased carbohydrate oxidation and a reciprocal decrease in fat oxidation. Furthermore, Clore et al. (42) showed similar metabolic responses with diets containing a high proportion of energy as CHO and which subjects started the study after manipulation of glycogen stores the preceding days. They inferred that under

these conditions gluconeogenesis is suppressed and that glucose derived from glycogen is released in excess of metabolic needs, accounting for the increase in whole body CHO oxidation.

In contrast with the observation concerning the HC meal, the excess in energy intake led almost exclusively to an accumulation of body fat with the HF meal, despite the initial increase in lipid oxidation at 30 minutes after meal intake. In regulating substrate oxidation, the effect of glycogen content should be considered. According to Flatt's model, fat oxidation can be raised on HF diets by maintaining glycogen concentrations in a lower range. In our study, a positive fat balance and a negative carbohydrate balance which must have resulted in reduction of the glycogen stores have been found. On the other hand, the postprandial protein oxidation rate increased significantly above baseline in this group, which maybe as a consequence of a process of gluconeogenesis (43).

It has long been assumed that there is autoregulatory control of protein oxidation about as efficient as that of carbohydrate (44). Whereas, there is no evidence of fat-driven autoregulation, rather, fat oxidation simply reflects the difference between the rates of carbohydrate and protein oxidation, and probably the higher fat oxidation rate in this group than on HC group, was related to the lower carbohydrate content of the meal. This is consistent with the concept that dietary CHO promotes its own oxidation whereas fat does not (12).

Other authors (45) reported no differences in substrate oxidation over a 9 h postprandial period following the addition of 50 g fat to a breakfast containing 10 g fat. Equally, Schutz et al. (6) reported no differences in fat oxidation during a 24 h period in seven young men when an additional 106 g fat was added to a mixed diet. These findings support the view that even over short periods of time surplus energy in the form of fat fails to promote fat oxidation and leads to a positive fat balance. In the present study, we compared meals which were isoenergetic and which were high in energy content to ensure a positive energy balance, which were almost identical after two meals.

Thermic effect of food

After meal intake, there is an increase in energy expenditure which has been referred to as dietary or nutrient induced thermogenesis. In the HC group, this increase in metabolic rate remained high for 120 minutes of the postprandial period and can be accounted for by the cost of both glucose storage as glycogen and lipid synthesis processes, which showed a strong statistical correlation with RMR. Similar correlations between net lipogenesis and both RMR and glucose have been reported by other authors (35) in a test meal containing a large load of CHO. However, it is well established that the measured increase in resting metabolic rate is in excess of this theoretically calculated value. This difference has been referred to as the regulatory component of thermogenesis modulated by the sympathetic nervous system (SNS) activity.

On the other hand, HR has been considered to reflect the activity of SNS (20). Thus, as reported by other authors (46, 47) the HR method provides an acceptable estimation of energy expenditure. The HR during the first 30 minutes after meal intake indicated higher values on the HC group, related to greater TEF, carbohydrate oxidation rate, and capillary blood glucose levels. Thereafter, and throughout the postprandial period, HR was higher in those subjects who received the HF liquid formula, according to the increase in carbohydrate oxidation rate and thermic effect of food in this group. In a previous report, Dulloo et al. (48) suggest that medium chain triacylglycerols (MCT), used in their study as the main fat in HF meal, could increase the energy expenditure by rising SNS activity, without changes in other metabolic responses like NPRQ or urinary nitrogen excretion. Therefore, its effect differs from those of long chain triacylglycerols: the gastric emptying is not slow and they are more rapidly hydrolyzed and absorbed (49).

Our results suggest that the postprandial increase of TEF is mainly due to carbohydrate metabolism, not only in the HC group after intake of a large load of carbohydrate but also in the HF group. This statement was supported by the statistical associations between the MR and carbohydrate oxidation rate in both dietetic groups. Moreover, other correlations could associate this increase in TEF with the SNS activation, as the statistical relationship between HR and RMR, carbohydrate oxidation rate and calculated net lipogenesis, in HC group, in agreement as described by other authors (43). This effect has been attributed in several studies (17, 43, 50) to a central action of insulin on hypothalamic areas, and other investigators (14, 51, 52) reported that is some aspect of cellular metabolism, and not insulin per se, that is responsible for activation of the sympathetic nervous system.

Energy balances

The effect of the excess of dietary fat content in energy and nutrient balances and their possible relationships, in both lean and obese subjects, have gained interest in human nutrition studies. Some of these reports (6, 7, 45) have shown close correlations between the overall energy balance and fat balance but not carbohydrate balance, which support the view that even over short periods of time surplus energy in the form of fat fails to promote fat oxidation and leads to a positive energy balance which closely correlates with the fat balance. However, it should be noted that these studies compared rates of postprandial substrate oxidation in meals with different energy contents and where fat was added to a mixed control diet. In the present study, isoenergetic meals were compared, which were high in energy content to ensure a positive energy balance. Energy balance was almost identical after

two meals, whereas substrate balances varied according to meal composition, showing the interrelationship between carbohydrate and fat metabolism at rest following isoenergetic meals.

Taking all data, we can conclude that the change in fuel selection and the increase of TEF is mainly due to CHO intake and metabolism, respectively, and that surplus of dietary CHO in the preceding days together with a large load of CHO can exceed the glycogen storage capacity and trigger the net lipid synthesis.

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