

Effects of Dietary Composition and Exercise Timing on Substrate Utilization and Sympathoadrenal Function in Healthy Young Women

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The effects of dietary composition (high-fat [FAT] or high-carbohydrate [CHO]) and exercise timing (preprandial exercise [Ex-] or postprandial exercise [-Ex]) on postprandial substrate utilization and sympathoadrenal function were studied in seven women aged 20 to 21 years. The experimental protocol included four different sessions (Ex-FAT, FAT-Ex, Ex-CHO, and CHO-Ex). The FAT and CHO diets provided 48% and 5% fat, respectively. On the experimental days, subjects ate a meal containing the same caloric energy at lunchtime, and they exercised for 30 minutes on a bicycle ergometer at an intensity of 60% maximal oxygen consumption ($\dot{V}O_{2\max}$) before and after the meal, followed by rest for 3 hours. The resting respiratory quotient (RQ) was significantly lower ($P < .05$) with the FAT diet or postprandial exercise. The mean RQ during the experimental period was 0.78, 0.75, 0.81, and 0.77 in Ex-FAT, FAT-Ex, Ex-CHO, and CHO-Ex groups, respectively. The total area under the curve of serum norepinephrine (NE) as an index of NE secretion was significantly higher ($P < .05$) with the FAT diet or postprandial exercise (130.2, 175.8, 33.0, and 136.9 $\text{ng} \cdot \text{mL}^{-1} \cdot \text{min}$, respectively). A negative correlation was observed between the RQ and the total area of NE ($r = .49$, $P < .05$). The serum thyroid hormone level was not influenced by dietary composition and exercise timing. These results suggest that postprandial exercise, especially after intake of a FAT diet, increases fat utilization via a slightly larger decrease in the RQ. This might be related to the sympathoadrenal system at rest and during exercise.

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EXERCISE ASSOCIATED with diet therapy is effective for obese individuals due to an increase in energy expenditure and substrate metabolism.^{1,2} Many persons who maintain a normal body weight also have a habit of daily exercise for their health. However, from a practical viewpoint, it is unclear whether it is better to exercise before or after a meal to maximize the thermogenic effect and fat utilization. It has been reported that exercise before a meal enhances the thermogenic response to meals in humans,³⁻⁶ but the effect of exercise after a meal on diet-induced thermogenesis (DIT) is unclear. Segal et al⁷⁻¹⁰ have reported an enhanced postprandial energy expenditure during and after exercise. However, other researchers have maintained that there is no relationship between exercise and DIT.¹¹⁻¹⁴

In addition, the amount of fat in the diet is suggested to be an important factor affecting body fat accumulation.¹⁵ It is well known that DIT is greater with a high-carbohydrate diet compared with a high-fat diet during rest.¹⁶ However, it is unclear whether the difference in the thermic effect between a high-carbohydrate diet and a high-fat diet was enhanced by exercise. Few studies have examined the effects of the type of diet, high-fat or high-carbohydrate, on postprandial energy expenditure during exercise in humans¹⁷ and in animals.^{18,19} We previously reported that postprandial energy expenditure during exercise is higher in rats fed a high-carbohydrate diet versus those fed a high-fat diet, which could be related to the increase in glycogen storage in the former.¹⁹ In the previous study, we did not examine the thermogenic effects of a meal and exercise during a rest period after prolonged exercise.

On the other hand, it is well known that DIT is regulated by

the sympathetic nervous system.²⁰ This form of heat production can be mimicked by norepinephrine (NE) and inhibited by beta blockade, and is associated with increased blood and urinary excretion of catecholamines and an elevated rate of NE turnover.²¹ The catecholamines are profoundly influenced by the action of the other major thermogenic hormones, thyroid hormones.²² The thermogenic effect of catecholamines increases in hyperthyroidism and decreases in hypothyroidism.²³

Physical exercise activates the sympathetic nervous system and increases the secretion of catecholamines from nerve endings or adrenal medulla.²⁴ While exercise plays an important role in preventing the excess accumulation of body fat, few studies have examined the effects of the interactions between dietary composition and exercise timing on sympathoadrenal function.

The purpose of this study was to examine the effect of dietary composition and exercise timing on sympathoadrenal function with respect to postprandial energy expenditure and substrate utilization in healthy young women.

SUBJECTS AND METHODS

Subjects

Seven young Japanese women (aged 20 to 21 years) who did not customarily exercise daily were recruited from Sanyo Women's College (Hiroshima, Japan) to participate in the study. All procedures were approved in advance by the Human Use Committee of Sanyo Women's College and were followed in accordance with the Helsinki Declaration of 1975, as revised in 1983. After a detailed explanation of the study, each subject provided informed written consent. Subjects were ascertained to be free of disease by a medical examination at Hiroshima General Hospital before the study. Physical characteristics of the subjects are shown in Table 1. The percent body fat was measured with a bioelectrical impedance analyzer (model TBF-102; Tanita, Tokyo, Japan). Maximal oxygen consumption ($\dot{V}O_{2\max}$) was determined by the method reported by Astrand and Rodahl,²⁵ using a bicycle ergometer (Monark, Varberg, Sweden). $\dot{V}O_{2\max}$ in the subjects was average or high compared with the standard value for 20-year-old females.

Experimental Design

During the period of the study, each subject maintained a normal life-style and ate ad libitum except for the day before the experimental period, in which each subject ate the same supper (50 kJ \cdot kg⁻¹ body

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Submitted February 23, 1999; accepted June 30, 1999.

Supported in part by a grant from the Uehara Memorial Foundation.

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0026-0495/99/4812-0022\$10.00/0

Table 1. Subject Characteristics

Subject No.	Age (yr)	Height (m)	Weight (kg)	Body Fat (%)	$\dot{V}O_{2\max}$ (mL · kg ⁻¹ · min ⁻¹)
1	21.0	1.55	46.3	26.5	40.1
2	20.0	1.51	41.7	22.6	44.6
3	21.0	1.57	51.8	25.5	45.5
4	20.0	1.60	62.3	35.3	39.4
5	21.0	1.63	61.1	27.4	44.3
6	20.0	1.71	67.2	26.0	38.6
7	21.0	1.61	47.3	21.4	43.1
Mean ± SE	20.6 ± 0.2	1.60 ± 0.02	54.0 ± 3.6	26.4 ± 1.7	42.2 ± 1.1

weight) at 7:00 PM. The subjects fasted overnight and entered the experimental room at 9:00 AM, where they rested until the start of experiments at 11:30 AM. The experimental protocol included four different sessions. All experiments were performed in the preovulatory phase on days 8 to 12 after the onset of menstruation.²⁶ The experimental sessions were divided into two types, preprandial exercise (Ex-) and postprandial exercise (-Ex). They were then divided into two subtypes, high-fat diet (FAT) and high-carbohydrate diet (CHO). The four types of experimental sessions were labeled Ex-FAT, FAT-Ex, Ex-CHO, and CHO-Ex (Fig 1). There were at least 2 days between sessions, and the subjects performed each session on 4 days in approximately 8 weeks. The four sessions of the study were performed in randomized order. The FAT diet provided 37%, 48%, and 15% energy as carbohydrate, fat, and protein, respectively, whereas the CHO diet provided 80%, 5%, and 15%, respectively. The energy values for the experimental diets were 8,440 and 6,350 J · g⁻¹ in the FAT and CHO diets, respectively. The experimental diet consisted of rice (Japonica), milk, low-fat milk, yogurt, cheese, and butter. The fatty acid composition of the two experimental diets was nearly the same.

On the days of the experiment, the subjects ate a meal containing the same amount of energy (57 kJ · kg⁻¹ body weight) from 12:45 to 1:00 PM (Ex-FAT and Ex-CHO) or noon to 12:15 PM (FAT-Ex and CHO-Ex). The subjects exercised for 30 minutes on a bicycle ergometer (Monark) at an intensity of 60% $\dot{V}O_{2\max}$ from noon to 12:30 PM (Ex-FAT and Ex-CHO) or 12:30 to 1:00 PM (FAT-Ex and CHO-Ex). The subjects then rested for 3 hours. During rest and exercise, oxygen uptake and the nonprotein respiratory quotient (RQ) were measured (from 11:30 AM to 4:00 PM). Blood samples were collected from the cephalic vein at the level of the forearm to obtain serum and plasma at noon and 1:00, 2:00, 3:00, and 4:00 PM. All procedures were performed in the experiment room under the same conditions (temperature 22 ± 1°C and humidity 60%).

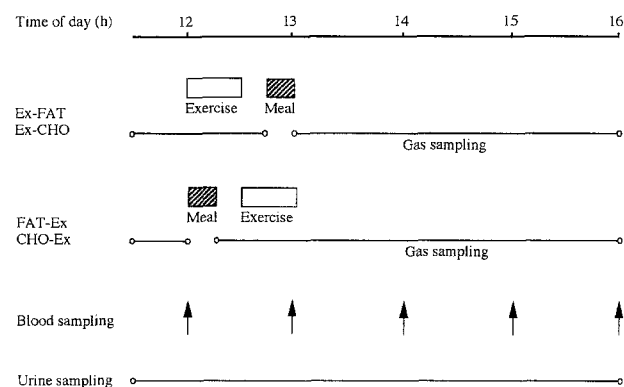


Fig 1. Experimental design.

Measurements

For measurement of oxygen uptake and the RQ, the subjects wore face masks (Takei, Tokyo, Japan) continuously throughout 270 minutes of each experiment, except for mealtime (15 minutes). All expired gas was collected into a Douglas bag (Takei), and the bag was changed every 15 minutes during rest and every 5 minutes during exercise. Oxygen and carbon dioxide in the expired collected gas were immediately measured by a gas analyzer (models RAS-30 and RAS-31; AIC, Tokyo, Japan). The RQ was calculated from oxygen consumption, carbon dioxide production, and urinary nitrogen loss at standard temperature and pressure-dry as an index of carbohydrate and fat utilization.²⁷ Energy expenditure was calculated with equations described previously²⁷: metabolic rate (kJ · min⁻¹) = 4.184 · [(4.686 + 1.096 · (RQ - 0.707)) · $\dot{V}O_{2np}$ + 4.60 · $\dot{V}O_{2p}$], where np is nonprotein and p is protein.

Serum epinephrine (E) and NE were assayed by high-performance liquid chromatography with electrochemical detection as modified by Refshauge et al.²⁸ A quantity of 2 mL serum was added to 250 µL 13-mmol · L⁻¹ Na₂S₂O₅, 50 mg dihydroxybenzylamine (internal standard), 1 mL 1-mol · L⁻¹ Tris-hydrochloride buffer (pH 8.6) containing 1% EDTA, and 30 mg acid-wash alumina. This mixture was stirred for 15 minutes. After decanting, the alumina was washed three times with 5 mL distilled water. After centrifugation, the supernatant was removed. The catecholamines (E, NE, and dihydroxybenzylamine) fixed on the alumina were eluted with 100 µL 0.22-mol · L⁻¹ acetic acid, 0.15 mmol sodium metabisulfite, and 0.025% EDTA in an Eppendorf tube and shaken and filtered through a Millipore Millex HV4 filter (Yonezawa, Japan). A 10-µL sample of the extract was injected into a reverse-phase analytic column (CA-50DS, 150 × 4.6 mm; Eicom, Kyoto, Japan) of a Shimadzu high-performance liquid chromatographic system (Kyoto, Japan). The mobile phase consisted of 100 mmol · L⁻¹ citric acid, 100 mmol · L⁻¹ sodium acetate, 1 mmol · L⁻¹ EDTA, 1 mmol · L⁻¹ sodium octylsulfonate, and 15% methanol. The flow rate of the mobile phase was fixed at 1 mL · min⁻¹. An electrochemical detector (model ECD-100; Eicom) was used (potential, 0.6 V).

Serum thyroxine (T₄) and triiodothyronine (T₃) levels were measured with the SPAC T₃ and T₄ radioimmunoassay kit (Daiichi Radioisotope Laboratory, Tokyo, Japan), respectively.

The total area under the curve for NE, E, T₃, and T₄ was calculated by time integration using a personal computer (Macintosh LC 575; Apple Japan, Tokyo, Japan).

Statistical Analysis

Statistical analysis was performed using a personal computer (Macintosh LC 575) with a statistical package (StatView 4.02; Abacus Concepts, Berkeley, CA). The effects of dietary composition and exercise timing were analyzed by repeated-measures ANOVA followed by paired comparisons using *t* tests.

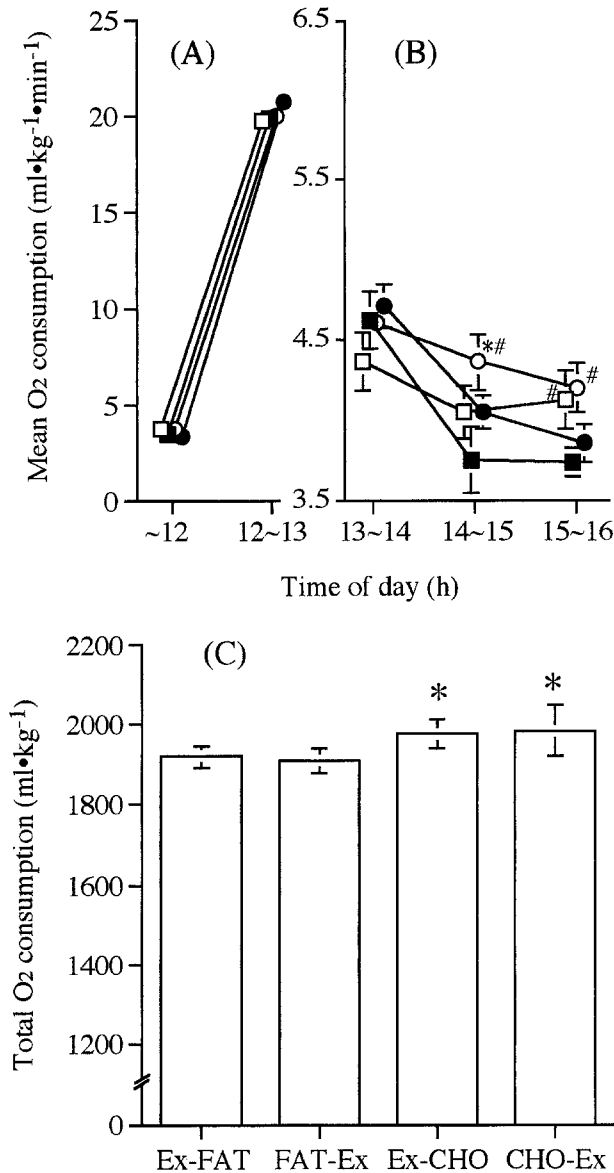


Fig 2. Mean oxygen consumption at rest and during exercise for Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups from 12:00 noon to 1:00 PM (A) and from 1:00 to 4:00 PM (B). Total oxygen consumption during the experimental period (C). Values are the mean \pm SEM for 7 subjects. * $P < .05$ v FAT diet, # $P < .05$ v postprandial exercise (repeated-measures ANOVA followed by paired comparisons with the *t* test).

RESULTS

Oxygen Consumption and RQ During Rest and Exercise

Oxygen consumption during rest and exercise was measured for 4 hours to assess the thermogenic effects of the diets and exercise (Fig 2). The mean values for total oxygen consumption are also shown. Oxygen consumption from 12:00 to 1:00 PM (meal and exercise time) did not differ among the four sessions. For the CHO diet, oxygen consumption from 2:00 to 4:00 PM was significantly higher during Ex-CHO versus CHO-Ex ($P < .05$). Regardless of exercise timing, oxygen consumption

from 12:00 to 4:00 PM was significantly higher for the CHO diet versus the FAT diet ($P < .05$). On the other hand, the RQ from 1:00 to 4:00 PM was significantly lower ($P < .05$) with the FAT diet or postprandial exercise (Fig 3). The mean values from 12:00 to 1:00 PM did not differ among the four sessions (mean, 0.82 ± 0.01). The mean RQs for the experimental period (11:30 AM to 4:00 PM) were 0.78, 0.75, 0.81, and 0.77 in the Ex-FAT, FAT-Ex, Ex-CHO, and CHO-Ex groups, respectively. Postprandial exercise decreased the mean RQ for both diets. Especially for the CHO diet, the RQ was significantly lower ($P < .05$) with postprandial exercise versus preprandial exercise (Fig 3).

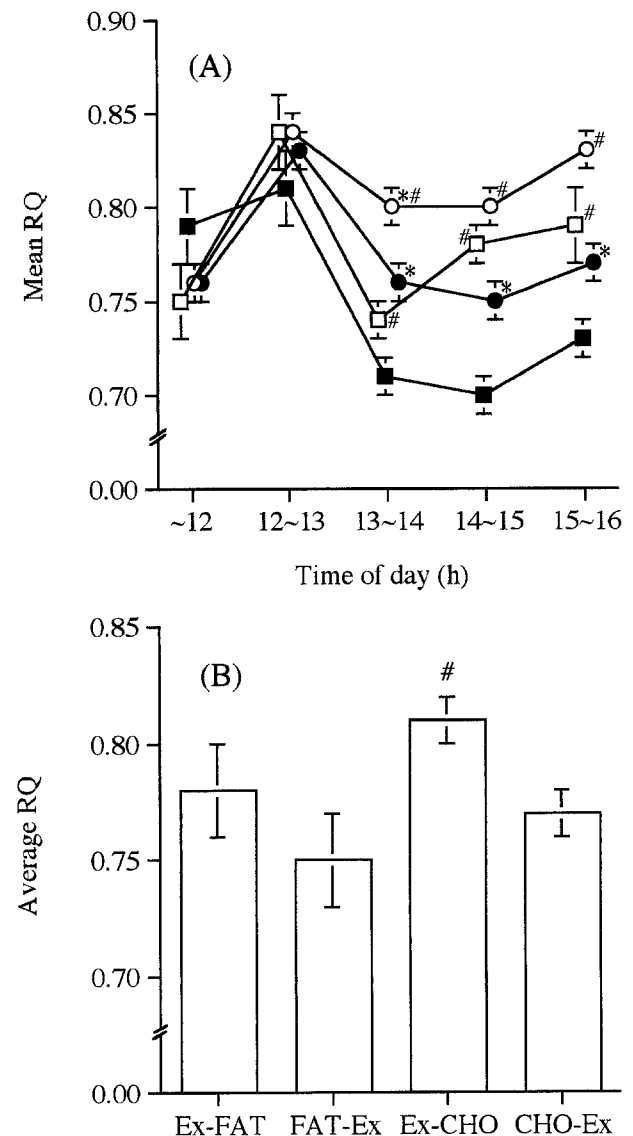


Fig 3. Mean RQ per hour from 12:00 noon to 4:00 PM (A) and average RQ of experimental period (B) in Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups. Values are the mean \pm SEM for 7 subjects. * $P < .05$ v FAT diet, # $P < .05$ v postprandial exercise (repeated-measures ANOVA followed by paired comparisons with the *t* test).

Serum NE, E, T_3 , and T_4

Serum NE and E increased temporarily after meal ingestion and exercise (Figs 4 and 5). The increases in NE and E were larger for postprandial exercise. Compared with preprandial exercise, postprandial exercise caused an increase in the total area of NE, which became statistically significant ($P < .05$) after intake of the CHO diet. For preprandial exercise, NE concentrations were significantly higher ($P < .05$) with the FAT diet versus the CHO diet (Fig 4). The total area of E was higher for FAT-Ex versus Ex-CHO, but the differences were not significant (Fig 5). Exercise timing did not significantly affect serum E levels. Figures 6 and 7 show that serum T_3 and T_4

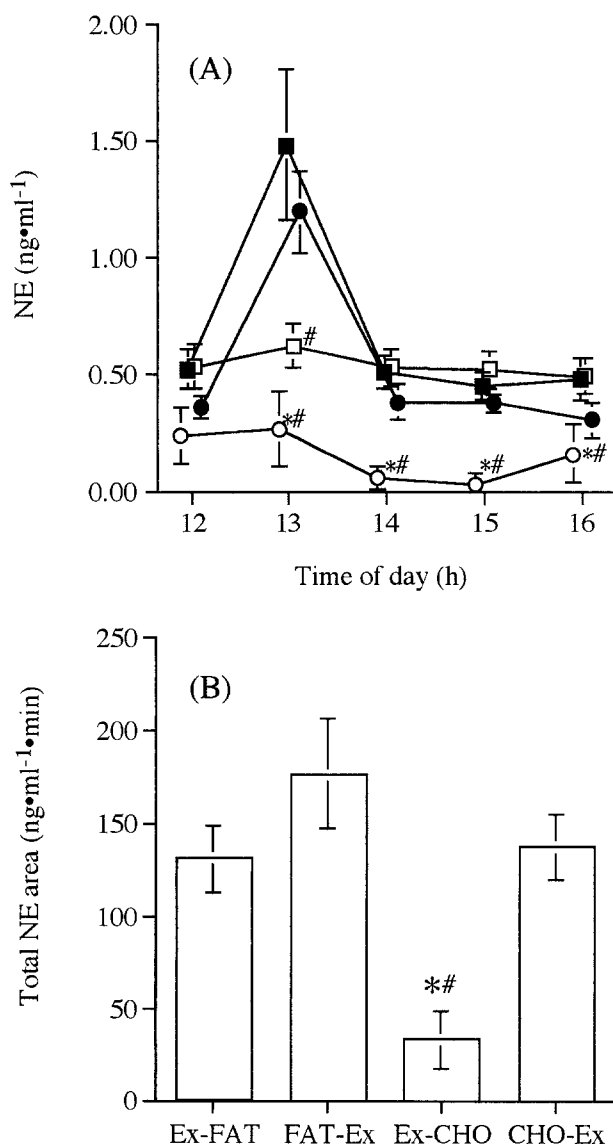


Fig 4. Serum NE concentration (A) and total area of NE (B) in Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups. Total area was calculated from (A). Values are the mean \pm SEM for 7 subjects. * $P < .05$ v FAT diet, # $P < .05$ v postprandial exercise (repeated-measures ANOVA followed by paired comparisons with the t test).

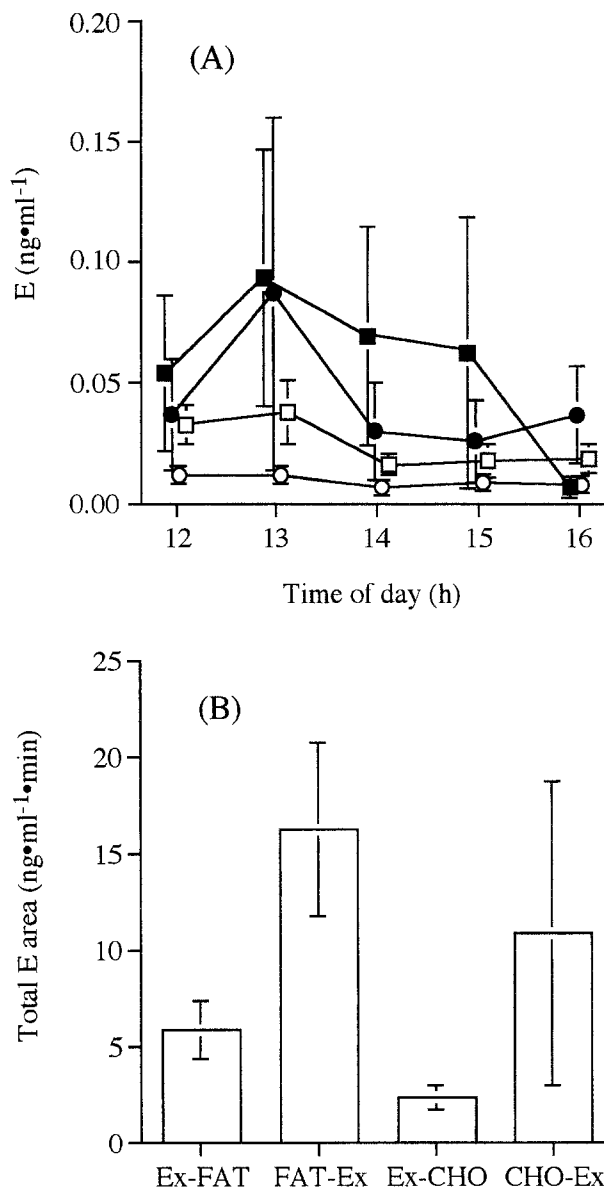


Fig 5. Serum E concentration (A) and total area of E (B) in Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups. Total area was calculated from (A). Values are the mean \pm SEM for 7 subjects.

concentrations did not change after the meal intake and exercise. The total area of T_3 and T_4 was not significantly different among the four sessions.

DISCUSSION

In the present study, oxygen consumption during the 4-hour rest and exercise period was significantly higher for the CHO diet compared with the FAT diet. Especially at certain points between 2:00 and 4:00 PM, oxygen consumption was significantly higher with preprandial exercise. The RQ results show that total carbohydrate utilization was higher for Ex-CHO versus FAT-Ex. Since increased carbohydrate oxidation causes enhanced liver and muscle glycogen synthesis,¹⁹ the present

findings suggest that the rates of substrate cycling were increased by Ex-CHO, and consequently, extra energy was required.^{29,30} Oxygen consumption for the experimental period is the result of at least four components: the basal metabolic rate, the increase in the metabolic rate during exercise, DIT, and excess postexercise oxygen consumption. We did not examine the conditions of fasting or not exercising in this experiment, as our study is based on the premise of exercise and meal intake within 1 hour of each other.

The mean values for oxygen consumption during exercise were 662, 668, 662, and 706 mL · kg⁻¹ · 30 min⁻¹ for Ex-FAT, FAT-Ex, Ex-CHO, and CHO-Ex, respectively. We previously reported that postprandial oxygen consumption during exercise (3 hours) is higher in rats fed a CHO diet versus those fed a FAT diet,¹⁹ and the findings of the present study not only correspond

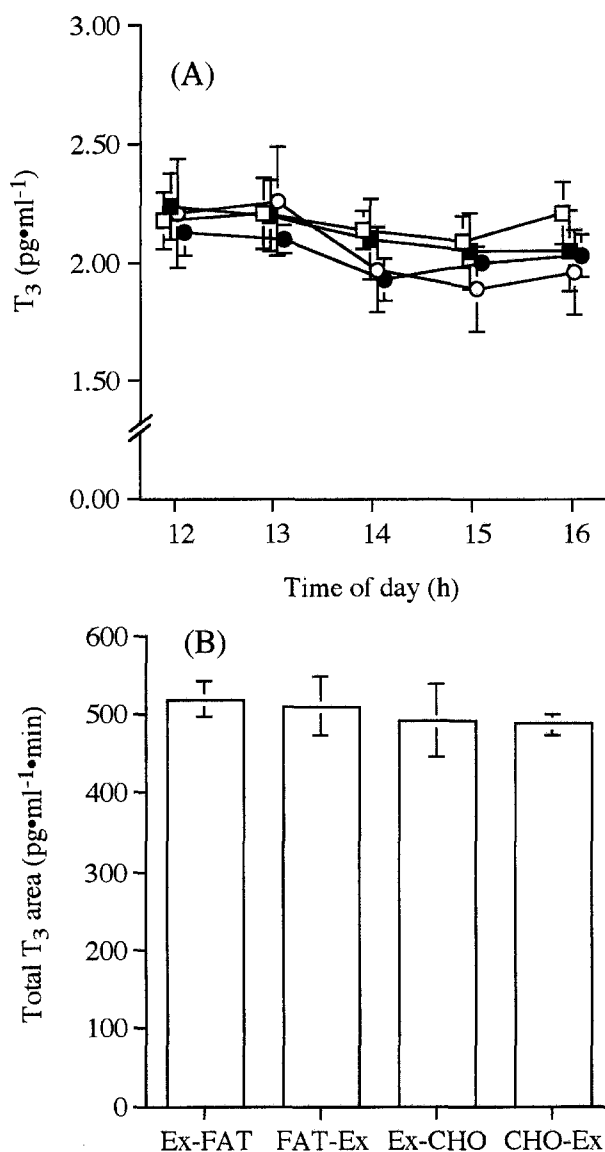


Fig 6. Serum T₃ concentration (A) and total area of T₃ (B) in Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups. Total area was calculated from (A). Values are the mean ± SEM for 7 subjects.

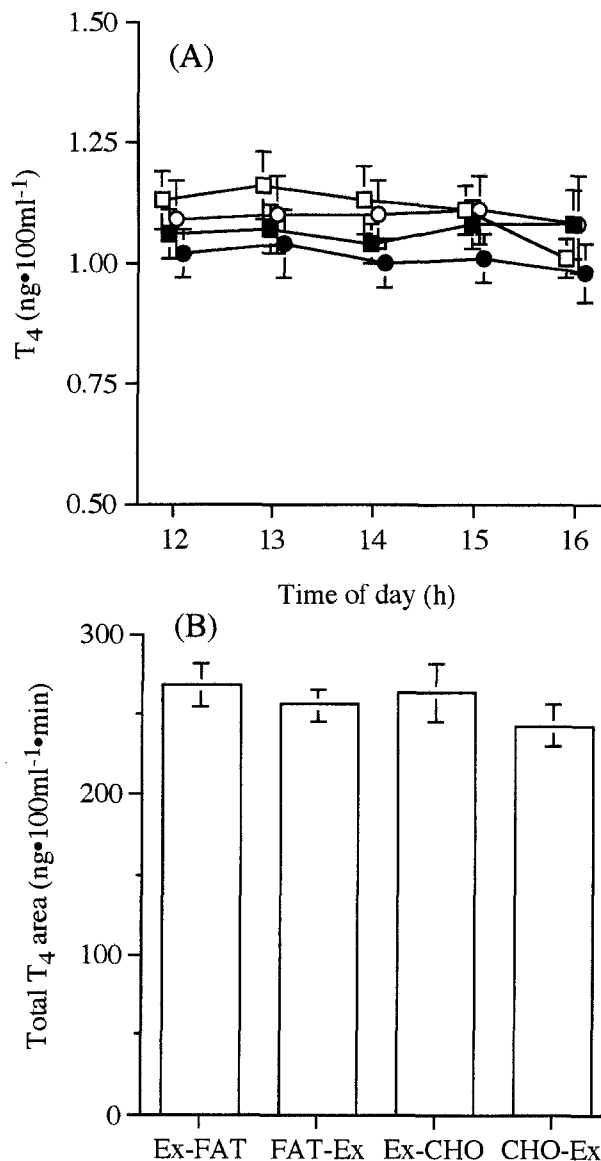


Fig 7. Serum T₄ concentration (A) and total area of T₄ (B) in Ex-FAT (□), FAT-Ex (■), Ex-CHO (○), and CHO-Ex (●) groups. Total area was calculated from (A). Values are the mean ± SEM for 7 subjects.

with the results in rats but also show consistency between humans and rats for energy expenditure during exercise. Among the four cases, the difference in oxygen consumption during exercise was slight, and oxygen consumption from noon to 1:00 PM was not significantly different. The present findings in healthy humans suggest that the contribution of diet and exercise to energy expenditure is more important in the rest period after a meal and exercise than during exercise, as exercise cannot be performed over a long period for relatively sedentary humans.

The mean RQs were lower for FAT-Ex versus Ex-CHO. Fat oxidation was higher with the FAT diet compared with the CHO diet, especially with postprandial exercise. Since it has been reported that analyses of fat accumulation and fat oxidation are affected by catecholamines,³¹ serum NE and E concentrations

were measured in the present study. We clearly show that the total area of NE estimated from these concentrations at each point was higher for FAT-Ex versus Ex-CHO. We observed a negative correlation between the RQ and the total area under the curve for NE ($r = .49$, $P < .05$). Poehlman et al³² subjected older men and women to 8 weeks of endurance training to examine its effects on basal fatty acid availability and total-body fat oxidation, as measured from [¹⁴C]palmitate infusion and indirect calorimetry, and NE kinetics from infusions of tritiated NE. They suggested that increases in fat oxidation were related to the increase in the rate of appearance of NE. The results of our experiment agree with their findings, at least in part.

On the other hand, in our previous study (designed similarly to this experiment), the serum free fatty acid concentration was significantly higher in subjects on the FAT diet versus the CHO diet, irrespective of exercise timing.³³ Unfortunately, we did not measure serum free fatty acid levels in this study. However, NE-induced lipolysis in adipose tissue may increase due to the FAT diet compared with the CHO diet, increasing fat oxidation.

The actions of thyroid hormones and catecholamines are intimately interrelated.³⁴ Thyroid hormones increase the number and affinity of β -adrenergic receptors in the heart and possibly some other tissues, and the effects of thyroid hormones on the heart resemble those of β -adrenergic stimulation.³⁴ In the present study, serum T₃ and T₄ concentrations were not affected by the acute stimulation of diet intake and exercise. The major

role of thyroid hormone in the regulation of overall heat production lies in the control of endothermic thermogenesis, one of the two components of obligatory thermogenesis.³⁵ It exerts this control by reacting with both nuclear and mitochondrial T₃ receptors in most body tissues to produce changes in the properties and overall mass of mitochondria and in the activity of the sodium pump. The physiological role of this action of thyroid hormone is apparently to set the obligatory heat production at a suitable level to balance the normal heat loss from a mammal at thermoneutrality.³⁵ From this point of view, thyroid hormones may not be affected by the acute stimulation of diet intake or exercise.

In this study, the FAT and CHO diets (57 kJ · kg⁻¹ body weight) contained 48% and 5% energy as fat and 37% and 80% energy as carbohydrate. In fact, the composition of these experimental diets may be the most extreme preexercise or postexercise meal possible. A medium-fat diet (about 25% energy as fat) should be examined in another experiment.

In summary, compared with preprandial exercise, postprandial exercise produced higher fat utilization when the FAT diet was consumed, and this might be related to the rate of sympathoadrenal response at rest and during exercise.

ACKNOWLEDGMENT

We wish to convey our appreciation to the subjects who participated faithfully in the experiment.

REFERENCES

1. LeBlanc J: Exercise training and energy expenditure, in Bray G, LeBlanc J, Inoue S, Suzuki M (eds): Diet and Obesity. Basel, Switzerland, Karger Basel, 1988, pp 181-190
2. Sweeney ME, Hill JO, Heller PA, et al: Severe vs moderate energy restriction with and without exercise in the treatment of obesity: Efficiency of weight loss. *Am J Clin Nutr* 57:127-134, 1993
3. Bielinski R, Schutz Y, Jequier E: Energy metabolism during the postexercise recovery in man. *Am J Clin Nutr* 42:69-82, 1985
4. Machlum S, Grandmaitagne M, Newsholme EA, et al: Magnitude and duration of excess postexercise oxygen consumption in healthy young subjects. *Metabolism* 35:425-429, 1986
5. Young JC, Treadway JL, Balon TW, et al: Prior exercise potentiates the thermic effect of a carbohydrate load. *Metabolism* 35:1048-1053, 1986
6. Poehlman ET, Horton ES: The impact of food intake and exercise on energy expenditure. *Nutr Rev* 47:129-137, 1989
7. Segal KR, Gutin B: Thermic effects of food and exercise in lean and obese women. *Metabolism* 32:581-589, 1983
8. Segal KR, Presta E, Gutin B: Thermic effects of food during graded exercise in normal weight and obese men. *Am J Clin Nutr* 40:995-1000, 1984
9. Segal KR, Gutin B, Nyman AM, et al: Thermic effects of food at rest, during exercise, and after exercise in lean and obese men of similar body weight. *J Clin Invest* 76:1107-1112, 1985
10. Segal KR, Chun A, Coronel P, et al: Effects of exercise mode and intensity on postprandial thermogenesis in lean and obese men. *J Appl Physiol* 72:1754-1763, 1992
11. Dailloso HM, James WPT: Whole-body calorimetry studies in adult men. *Br J Nutr* 52:65-72, 1984
12. Hickson JF, Hartung GF Jr, Pate TD, et al: Effect of short-term energy intake level and exercise on oxygen consumption in men. *Eur J Appl Physiol* 55:198-201, 1986
13. Pacy PJ, Barton N, Webster JD, et al: The energy cost of aerobic exercise in fed and fasted normal subjects. *Am J Clin Nutr* 42:764-768, 1985
14. Schutz Y, Bessard T, Jequier E: Exercise and postprandial thermogenesis in obese women before and after weight loss. *Am J Clin Nutr* 45:1424-1432, 1987
15. Flatt JP: The difference in storage capacities for carbohydrate and for fat, and its implications in the regulation of body weight. *Ann NY Acad Sci* 449:104-123, 1987
16. Jequier E: Influence of nutrient administration on energy expenditure in man. *Clin Nutr* 5:181-186, 1986
17. Abbott WGH, Howard BV, Ruotolo G, et al: Energy expenditure in humans: Effects of dietary fat and carbohydrate. *Am J Physiol* 258:E347-E351, 1990
18. Gleeson M, Waring JJ: Influence of diet on the storage, mobilization and utilization of energy reserves in trained and untrained rats. *Comp Biochem Physiol* 85:411-415, 1986
19. Saitoh S, Matsuo T, Suzuki M: The effects of a high carbohydrate diet on postprandial energy expenditure during exercise in rats. *Eur J Appl Physiol* 66:445-450, 1993
20. Landsberg L, Saville E, Young JB: Sympathoadrenal system and regulation of thermogenesis. *Am J Physiol* 247:E181-E189, 1984
21. Himms-Hagen J, Phil D: Thermogenesis in brown adipose tissue as an energy buffer. *N Engl J Med* 311:1549-1555, 1983
22. Gibson A: The influence of endocrine hormones on the autonomic nervous system. *J Auton Pharmacol* 1:331-358, 1981
23. Fregly MJ, Field FP, Katovich MJ, et al: Catecholamine-thyroid hormone interaction in cold-acclimated rats. *Fed Proc* 38:2162-2169, 1979
24. Mazzeo RS, Grantham PA: Norepinephrine turnover in various tissues at rest and during exercise: Evidence for a training effect. *Metabolism* 38:479-483, 1989

25. Astrand PO, Rodahl K: Textbook of Work Physiology (ed 3). New York, NY, McGraw-Hill, 1986
26. Ferraro R, Lillioja S, Fontvieille AM, et al: Lower sedentary metabolic rate in women compared with men. *J Clin Invest* 90:780-784, 1992
27. Jequier E, Acheson K, Schutz Y: Assessment of energy expenditure and fat utilization in man. *Annu Rev Nutr* 7:187-208, 1987
28. Refshauge C, Kissinger PT, Dreiling R, et al: New high performance liquid chromatographic analysis of brain catecholamines. *Life Sci* 14:311-322, 1973
29. Bahr R, Ingnes I, Vaage O, et al: Effect of duration of exercise on excess postexercise O₂ consumption. *J Appl Physiol* 62:485-490, 1987
30. Bahr R, Sejersted OM: Effect of feeding and fasting on excess postexercise oxygen consumption. *J Appl Physiol* 71:2088-2093, 1991
31. Granner DK: Hormones of the adrenal medulla, in Murray RK, Granner DK, Mayes PA, Rodwell VW (eds): *Harper's Biochemistry*. Norwalk, CT, Appleton & Lange, 1993, pp 536-541
32. Poehlman ET, Toth MJ, Fonong T: Exercise, substrate utilization and energy requirements in the elderly. *Int J Obes* 19:S93-S96, 1995 (suppl 4)
33. Matsuo T, Sumida H, Jimbo H, et al: Effects of dietary composition and exercise timing on energy expenditure and substrate utilization in healthy young women. *J Clin Biochem Nutr* 20:161-172, 1996
34. Ganong WF: *Review of Medical Physiology*. Norwalk, CT, Appleton & Lange, 1993, pp 287-301
35. Himms-Hagen J: Thyroid hormones and thermogenesis, in Girardier L, Stock MJ (eds): *Mammalian Thermogenesis*. London, UK, Chapman & Hall, 1983, pp 141-177