Body weight, body composition, and energy metabolism in lean and obese Zucker rats fed soybean oil or butter¹,²

Valérie Rolland, Suzanne Roseau, Gilles Fromentin, Stylianos Nicolaidis, Daniel Tomé, and Patrick C Even

ABSTRACT
Background: Dietary fat composition is thought to affect body weight regulation independent of the amount of fat ingested.
Objective: We analyzed the feeding behavior, body weight gain, body composition, and energy metabolism in lean and obese rats fed a diet in which fat was in the form of either butter or soybean oil.

Design: Ten lean (Fa/?) and 10 obese (fa/fa) adult Zucker rats were divided into 4 groups according to a 2 × 2 experimental design. They were fed a normally balanced diet over 11 wk in which 30% of energy was either soybean oil or butter. Food intake, body weight gain, and body composition were measured. Indirect calorimetry was used to study energy metabolism at rest and in relation to feeding and activity.

Results: Food intake increased similarly in lean and obese rats after butter feeding. Body weight gain increased in obese rats and decreased in lean rats after butter feeding. Body weight gain in obese rats was due mainly to an increase in the weight of lean tissues besides muscle, whereas adiposity and distribution of fat between the various pads did not change. Resting metabolic rates and postprandial lipid oxidation increased in butter-fed obese rats. Lipid oxidation during exercise was not significantly different between obese and lean rats. Fat oxidation increased in butter-fed lean rats during treadmill running at moderate intensity.


KEY WORDS Dietary fats, saturated fatty acids, unsaturated fatty acids, medium-chain triacylglycerol, long-chain triacylglycerol, obesity, organ mass, respiratory quotient, substrate oxidation, exercise, indirect calorimetry, rats

INTRODUCTION
The role of increased fat intake in the prevalence of overweight and obesity has been well shown. Some individuals are more inclined than others to eat high-fat diets, and it appears that the hedonic preference for fat and the higher intake of such preferred food items are involved in diet-induced overweight and obesity (1–3). The effect of fat on body weight, body composition, and energy expenditure may also depend on the composition of the fat, ie, the saturated and unsaturated fat content, but results in this area remain mixed. A diet high in saturated fat, compared with one high in unsaturated fat, was reported to either increase, decrease, or have no effect on body weight (4–8) and body fat storage (5–9). Diets high in polyunsaturated fats were reported to increase total energy expenditure in mice (10); resting metabolic rate, thermic effect of food (TEF; 11), and fat oxidation in humans (12); and norepinephrine stimulation of lipolysis in white adipose tissue of rats (13). The composition of the dietary fat might also act by altering cellular membrane lipid composition. A few polyunsaturated fatty acids were reported to increase the sensitivity of the adipocyte β-adrenergic receptors (14) and thus have the potential to build more labile fat stores. As a result, the differences in the behavioral responses and metabolic consequences of dietary fat intake between lean and obese subjects remain to be understood. Among the various animal models of obesity, the obese Zucker rat is a classic model of genetic obesity (15) and was shown to be very sensitive to a high-fat diet (16). In the present study, we tested the hypothesis that for 2 commonly used forms of fat, ie, butter (animal fat rich in saturated fatty acids) and soybean oil (vegetable fat rich in polyunsaturated fatty acids), the response of lean and obese Zucker rats might differ. For this purpose, we measured food intake, body weight gain, body composition, and energy metabolism in lean and obese Zucker rats fed diets enriched with either butter or soybean oil.

MATERIALS AND METHODS
Materials
This study was performed with 10 lean (Fa/?) and 10 obese (fa/f) male Zucker rats provided by D Gripois from the Laboratoire de NeuroEndocrinologie (Université Paris Sud, France).

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Received March 16, 2000.
Accepted for publication March 6, 2001.
All rats were adapted to a feeding regimen of 65% of energy as carbohydrate, 25% as protein, and 10% as lipids (Extralabo, Provins, France). All rats dwelled in conditions of 22°C in an artificial 12-h light and 12-h dark cycle (lights on at 0600) for ≥15 d. Rats were aged 6 wk at the onset of the experiment. Obese and lean rats weighed an average of 12.4 g, respectively. The experiment was conducted according to the French committee recommendations for animal care.

### Food intake and body weight

After adaptation, obese and lean Zucker rats were matched for initial body weight and then divided into 4 groups according to a 2 × 2 experimental design. They were fed a balanced diet for 11 wk that provided 30% of energy as either soybean oil or butter, 50% as carbohydrate, and 20% as protein ([Table 1](#tab1)). The composition of butter and soybean oil are shown in [Table 1](#tab1).

### Metabolic responses to feeding

Energy expenditure components were measured by use of open-circuit indirect calorimetry on unrestrained rats housed in a 9 L cylindrical, air-proof cage (floor, 408 cm2; height, 17.5 cm) ([17](#footnote17)). Spontaneous activity was recorded with 3 Piezo electric force transducers (FR91967; Kistler SA, Les Ulis, France) with a sensitivity of 1 g force, which were fixed under the floor of the cage. Food intake was recorded from a 0–100-g gauge (sensitivity 0.1 g) that weighed the food cup included in the cage. All the parameters were recorded at 10-s intervals. The metabolic response to feeding was measured in rats housed in the metabolic chamber from 0900 with water but no food available until a 4-g test meal of the maintenance diet was given at 1830. This long period of recording before the meal allowed the rats to become accustomed to the metabolic chamber. The TEF was measured by continuously recording respiratory exchanges and spontaneous activity. The metabolic cost of activity was computed from the increases in metabolic rate specifically associated with bursts of spontaneous activity and was then extracted according to a process of filtering previously described ([17](#footnote17), [18](#footnote18)). Background metabolism was calculated by subtracting the energetic cost of activity from total metabolism. The TEF was computed as the meal-induced increase in background metabolism. Total and background metabolism were computed by using classic stoichiometric formulas assuming that protein oxidation accounted for a constant 10% of total oxidation ([17](#footnote17)).

### Metabolic response to spontaneous activity

The rates of glucose and lipid oxidation specifically devoted to fueling the working muscles were computed by measuring the changes in oxygen consumption and carbon dioxide production specifically associated with physical activity, from immediately postmeal to 12 h postmeal. To do so, we located the exact onset of all the bursts of spontaneous activity that occurred in the rats of

### TABLE 1

<table>
<thead>
<tr>
<th>Composition (g)</th>
<th>Soybean oil</th>
<th>Butter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat (soybean oil or butter)</td>
<td>14.13</td>
<td>17.09</td>
</tr>
<tr>
<td>Carbohydrate (starch)</td>
<td>56.52</td>
<td>54.37</td>
</tr>
<tr>
<td>Protein (casein)</td>
<td>21.2</td>
<td>20.39</td>
</tr>
<tr>
<td>Cellulose</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Minerals</td>
<td>5.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Vitamins</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Methionine</td>
<td>0.15</td>
<td>0.15</td>
</tr>
<tr>
<td>Energy (kJ/g)</td>
<td>17.72</td>
<td>17.05</td>
</tr>
</tbody>
</table>

1 Values are per 100 g diet.

2 Minerals provided per 100 g diet: calcium phosphate, 19.44 g; potassium phosphate, 12 g; calcium carbonate, 9 g; magnesium sulfate, 4.44 g; sodium chloride, 3.51 g; magnesium oxide, 1 g; ferric sulfate, 0.43 g; zinc sulfate, 0.26 g; manganese sulfate, 0.26 g; cupric sulfate, 0.051 g; sodium fluoride, 0.041 g; potassium chromate, 0.0022 g; potassium iodide, 0.0022 g; ammonium molybdate, 0.0011 g; cobalt carbonate, 0.0011 g; and sodium selenite, 0.0011 g.

3 Vitamins provided per 100 g diet: vitamin A (retinyl acetate), 0.5 mg; cholecalciferol, 6.25 mg; all-rac-α-tocopherol acetate, 500 mg; vitamin K<sub>3</sub> (menadione), 0.1 mg; thiamine, 0.1 mg; riboflavin, 0.1 mg; nicotinic acid, 0.45 mg; Ca-pantothenate, 0.3 mg; pyridoxine-HCl, 0.1 mg; niacin, 0.5 mg; biotin, 0.02 mg; folic acid, 0.2 mg; vitamin B-12 (cyanocobalamin), 0.00135 mg; vitamin C, 10 mg; amino acid benzoic acid, 5 mg; and choline chloride, 75 mg.
Metabolic response to treadmill running

The metabolic response to treadmill running was measured in rats running on a sealed, motorized treadmill that was built in the workshop of the laboratory. Respiratory exchanges during exercise were continuously measured from 3 h before until 1 h after exercise by connecting the treadmill to the same open-circuit metabolic device used for measuring the metabolic response to feeding and spontaneous activity of rats housed in the metabolic chamber (see above). Rates of glucose and lipid oxidation were computed assuming that protein oxidation was constant at 1 mg/min, even during treadmill running (10–15% of preexercise resting metabolism). Housing the rats in the treadmill 3 h before the running test allowed them to become accustomed to the environment. In particular, the rats learned to avoid the electrical grid at the rear of the line when the belt was motionless. Rats were housed in the treadmill at 1100. Exercise began at 1400 and lasted 90 min. Measurements ended at 1700. In lean rats, the speed of the treadmill was set at 10 m/min. In obese rats, the speed was increased progressively from 10 to 20 m/min in lean rats and from 7 to 14 m/min in obese rats. For the sake of statistical comparisons, exercise on the treadmill was subdivided into 4 of the most representative time periods: 1) the preexercise period (30 min before exercise while the rat rested), 2) the exercise period (the last 30 min of the steady-speed exercise), 3) the acceleration period (the last 15 min of acceleration), and 4) the recovery period (a 30-min period following the first 30 min after exercise ended).

Statistical analysis

Data are presented as means ± SEMs. Changes in body weight and food intake over time were analyzed with analysis of variance (ANOVA) for repeated measurements. Tests conducted on body composition and variables of energy metabolism in relation to treadmill running were analyzed by using ANOVA. When significant differences were detected, post hoc comparisons of means were performed by using Scheffe’s procedure. Statistical significance was set at P < 0.05. SAS software (version 6.11; SAS, Cary, NC) was used for the analyses.

Because the computer program used to handle the data files and extract the changes related to spontaneous activity provided means ± SDs but did not extract individual values, rates of basal glucose and lipid oxidation in relation to spontaneous activity were not compared with ANOVA but with Student’s t test. Bonferroni’s correction was applied to correct for the fact that repeated t tests were performed. Statistical significance was set at P < 0.05.

RESULTS

Food intake, body weight, and body composition

Food intake was greater in butter-fed rats than in soybean oil–fed rats (Figure 1). However, despite an apparently larger effect in obese Zucker rats, there was no interaction between phenotype and diet, suggesting that the effect did not differ significantly between obese and lean rats. A phenotype effect appeared transiently between 1 and 4 h after the food cups were refilled, indicating that food intake began earlier in lean than in obese rats, but that the difference disappeared thereafter.

Initial body weight was slightly greater in obese than in lean rats (227.0 ± 36.0 compared with 195.5 ± 35.0 g; P = 0.07) (Figure 2). Thereafter, body weight increased more rapidly in obese rats. By day 5, body weight was significantly different between phenotypes. Thereafter, the difference increased throughout the study. No general effect of diet was observed, but a significant diet × phenotype interaction appeared because body weight was greater in butter-fed than in soybean oil–fed obese rats and lower in butter-fed than in soybean oil–fed lean rats.

Carcass analysis showed that in obese rats, fat mass and residual body mass weights were higher and carcass weights lower in obese rats than in lean rats after both diets (Table 3). A significant interaction between diet and phenotype further indicated that butter feeding increased fat mass and residual body mass weights more in obese than in lean rats. Among organs with a high metabolic rate, the liver, heart, and to some extent, the kidneys, but not brain, were heavier in obese rats. In addition, butter feeding increased specifically the weight of the liver in obese and lean rats. When fat mass, residual body mass, and carcass...
The distribution of fat between the various fat pads was significantly different between lean and obese rats; obese rats had a larger proportion of fat in the subcutaneous deposits and less in the mesenteric and epididymal deposits. In addition, the dietary regimen did not affect the distribution of fat between the various pads in either obese or lean rats.

**Metabolic response to feeding**

Analysis of the metabolic parameters and of their changes in relation to feeding showed that premeal respiratory quotient (RQ) and premeal metabolism were higher in obese than in lean rats (Table 4). In addition, a significant interaction between diet and phenotype was observed in the premeal metabolism, indicating that butter feeding increased metabolism in butter-fed obese but not lean rats. The thermogenic effect of the meal accounted for 10–12% of the energy intake supplied by the test meal in all groups. In contrast, the test meal increased the RQ more in lean than in obese rats. However, because the premeal RQ was much lower in lean than obese rats, the average RQ measured during TEF remained lower in lean than obese rats (Table 4).

**Metabolic response to spontaneous activity**

In all rats, basal glucose oxidation decreased as the time after consumption of the meal increased (Table 5). In lean rats, basal lipid oxidation was low early after consumption of the meal, then increased progressively as the time after the meal increased. In soybean oil–fed obese rats, basal lipid oxidation was low at all time intervals. In contrast, butter-fed obese rats always had a high rate of resting basal lipid oxidation, even early after the end of the meal.

### Table 3

<table>
<thead>
<tr>
<th>Tissues and organs</th>
<th>Obese rats</th>
<th>Lean rats</th>
<th>P (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Butter (n = 5)</td>
<td>Soybean oil (n = 5)</td>
<td>Butter (n = 5)</td>
</tr>
<tr>
<td>Body weight</td>
<td>580.83 ± 32.51</td>
<td>516.01 ± 7.79</td>
<td>355.45 ± 17.37</td>
</tr>
<tr>
<td>Carcass</td>
<td>176.81 ± 9.29</td>
<td>178.21 ± 4.51</td>
<td>202.93 ± 8.00</td>
</tr>
<tr>
<td>Fat mass</td>
<td>141.76 ± 13.41</td>
<td>111.54 ± 5.13</td>
<td>18.44 ± 1.58</td>
</tr>
<tr>
<td>RMB</td>
<td>262.25 ± 11.58</td>
<td>226.26 ± 4.96</td>
<td>134.07 ± 8.14</td>
</tr>
<tr>
<td>Liver</td>
<td>18.99 ± 1.46</td>
<td>15.73 ± 0.61</td>
<td>9.297 ± 0.843</td>
</tr>
<tr>
<td>Heart</td>
<td>1.211 ± 0.084</td>
<td>1.140 ± 0.032</td>
<td>1.015 ± 0.032</td>
</tr>
<tr>
<td>Kidney</td>
<td>2.761 ± 0.382</td>
<td>2.656 ± 0.104</td>
<td>2.250 ± 0.084</td>
</tr>
<tr>
<td>Brain</td>
<td>1.87 ± 0.05</td>
<td>1.95 ± 0.00</td>
<td>1.95 ± 0.00</td>
</tr>
<tr>
<td>(% of total body wt)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass</td>
<td>24.13 ± 1.123</td>
<td>21.603 ± 0.927</td>
<td>5.16 ± 0.25</td>
</tr>
<tr>
<td>Carcass</td>
<td>30.476 ± 0.34</td>
<td>34.54 ± 0.696</td>
<td>57.22 ± 0.62</td>
</tr>
<tr>
<td>RMB</td>
<td>45.39 ± 1.13</td>
<td>43.86 ± 0.83</td>
<td>37.62 ± 0.45</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesenteric</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epididymal</td>
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<td></td>
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<tr>
<td>Retroperitoneal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal fat</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. x ± SEM. RBM, residual body mass (body weight − carcass − fat).
2. Measured at time of carcass analysis between days 64 and 88 and thus do not exactly coincide with body weights reported in Figure 1.
3. Calculated as the sum of epididymal, mesenteric, and retroperitoneal fat pads.
Lipid oxidation

Changes in basal glucose and lipid oxidation rates in relation to the time elapsed since the delivery of the test meal

TABLE 5
Average RQ during TEF 0.96 ± Zucker rats
Meal-induced changes in respiratory quotient (RQ) and energy metabolism after 4 g of the butter or soybean oil maintenance diet in lean and obese

Average increase in TEF 0.054 ± butter-fed lean rats, indicating a high ratio of lipid to glucose oxidation. This tendency was confirmed by the fact that preexercise metabolism between the groups, indicating that during this period of higher intensity exercise, substrate mobilization was similar in all groups.

Metabolic response to treadmill running

Resting metabolism measured immediately before exercise was higher in obese than in lean rats, but was not affected by the feeding regimen (Table 6). Preexercise RQ was not significantly different between the groups, despite the low RQ (0.82) in butter-fed lean rats, indicating a high ratio of lipid to glucose oxidation. This tendency was confirmed by the fact that preexercise glucose oxidation was higher and lipid oxidation was lower in obese rats than in lean rats. In addition, a significant interaction between diet and phenotype indicated that glucose oxidation decreased and lipid oxidation increased as a result of the feeding regimen in the lean rats only (Table 6).

During the steady state period, exercise increased metabolism in the rats by an average of 2.77 W (118%). The difference in metabolism between lean and obese rats was lower than at rest but remained significant. Glucose oxidation was greater in obese than in lean rats and a significant interaction between diet and phenotype indicated that the butter-fed lean rats oxidized significantly less glucose. No significant differences in lipid oxidation were observed, indicating that obese rats used the same amount of lipids as did lean rats during exercise.

During the acceleration period, metabolism increased by an average of 4.64 W (199%) relative to preexercise metabolism. Metabolism remained significantly higher in obese rats. No further significant differences were observed in glucose oxidation, lipid oxidation, and RQ between the groups, indicating that during this period of higher intensity exercise, substrate mobilization was similar in all groups.

In obese rats fed the soybean oil diet only, energy expenditure returned to the preexercise level within 60 min after completion of the run (Figure 4). In butter-fed obese rats, there remained a slight but significant excess in postexercise oxygen consumption. In lean rats, a large excess in postexercise oxygen consumption was observed in both soybean oil–fed and butter-fed rats. The maintenance of a larger excess in postexercise oxygen consumption in the lean rats abolished the difference in metabolism between lean and obese rats during the postexercise period (Table 6), but glucose oxidation remained significantly lower in lean rats. In all groups, the reduction in RQ, ie, the increase in metabolism returned to the preexercise level within 60 min after completion of the run (Figure 4). In butter-fed obese rats, there remained a slight but significant excess in postexercise oxygen consumption. In lean rats, a large excess in postexercise oxygen consumption was observed in both soybean oil–fed and butter-fed rats. The maintenance of a larger excess in postexercise oxygen consumption in the lean rats abolished the difference in metabolism between lean and obese rats during the postexercise period (Table 6), but glucose oxidation remained significantly lower in lean rats. In all groups, the reduction in RQ, ie, the increase in metabolism between lean and obese rats was lower than at rest but remained significant. Glucose oxidation was greater in obese than in lean rats and a significant interaction between diet and phenotype indicated that the butter-fed lean rats oxidized significantly less glucose. No significant differences in lipid oxidation were observed, indicating that obese rats used the same amount of lipids as did lean rats during exercise.

No significant differences were observed between rats or between diets regarding the respective roles of glucose and lipids during periods of spontaneous activity (Figure 3). In all rats and over all periods considered, glucose and lipid oxidation were equally stimulated. Only in butter-fed lean rats did lipid oxidation fuel the major part of the energetic cost of activity in the early postprandial period. However, it is difficult to draw any firm conclusion from these data because only 2 periods of spontaneous activity could be obtained from the 5 rats during this time interval.

TABLE 4
Meal-induced changes in respiratory quotient (RQ) and energy metabolism after 4 g of the butter or soybean oil maintenance diet in lean and obese Zucker rats

<table>
<thead>
<tr>
<th></th>
<th>Obese rats</th>
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</tr>
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<tbody>
<tr>
<td></td>
<td>Butter (n = 5)</td>
<td>Soybean oil (n = 5)</td>
<td>Butter (n = 5)</td>
</tr>
<tr>
<td>Premeal RQ</td>
<td>0.91 ± 0.02</td>
<td>0.91 ± 0.02</td>
<td>0.83 ± 0.02</td>
</tr>
<tr>
<td>Premeal metabolism (W)</td>
<td>2.54 ± 0.18</td>
<td>2.12 ± 0.13</td>
<td>1.88 ± 0.06</td>
</tr>
<tr>
<td>TEF (kJ)</td>
<td>6.96 ± 0.80</td>
<td>7.24 ± 0.71</td>
<td>8.37 ± 0.24</td>
</tr>
<tr>
<td>(%)</td>
<td>10.21 ± 1.17</td>
<td>10.21 ± 1.00</td>
<td>12.28 ± 0.36</td>
</tr>
<tr>
<td>Average increase in RQ during TEF</td>
<td>0.054 ± 0.011</td>
<td>0.076 ± 0.012</td>
<td>0.104 ± 0.009</td>
</tr>
<tr>
<td>Average RQ during TEF</td>
<td>0.96 ± 0.01</td>
<td>0.99 ± 0.02</td>
<td>0.93 ± 0.01</td>
</tr>
</tbody>
</table>

*SEM. TEF, thermic effect of food.

1 ± SEM. TEF, thermic effect of food.

2 Student’s t test followed by Bonferroni’s correction.
3 n = 4.
4 n = 2.
5 Significantly different from 1–3 h (Student’s t test followed by Bonferroni’s correction): 1 P ≤ 0.001, 2 P < 0.005, 3 P < 0.05.
6 Significantly different from 3–6 h (Student’s t test followed by Bonferroni’s correction): 1 P ≤ 0.001, 2 P < 0.005.
the ratio of lipid to glucose oxidation induced during exercise, was maintained after the completion of the exercise.

**DISCUSSION**

The present study showed that food intake was greater in both lean and obese Zucker rats during butter feeding than during soybean oil feeding. However, body composition was affected mainly in obese rats: residual body mass and fat mass were significantly increased by butter feeding whereas absolute carcass weight was not affected. This phenomenon led to a significant decrease in the weight of the carcass relative to body weight. In contrast, liver weight increased in both lean and obese rats. Measurements of basal metabolism, feeding, and activity-related energy expenditure showed that in obese rats, butter feeding increased basal metabolism. In contrast, butter feeding did not affect metabolism, but tended to increase lipid oxidation in lean rats, particularly during exercise. These phenomena were probably responsible for the fact

**FIGURE 3.** Mean (±SEM) resting and activity-stimulated glucose and lipid oxidation rates during the bursts of activity that occurred spontaneously between 1 and 3 h, 3 and 6 h, and 6 and 12 h after the test meal (n = 5 per group). Glucose and lipid oxidation were computed from oxygen consumption and carbon dioxide production assuming that protein oxidation fueled a constant 10% of resting metabolism. Results in the various groups are given from left to right, and, within each group, the changes over time are given from top to bottom.
that in both phenotypes, the increase in food intake induced by butter feeding did not have much effect on final body weight.

**Food intake, body weight, and body composition**

In the present study, food intake increased in all rats during butter feeding. This observation confirms the suggestion that both obese and lean rats are sensitive to the palatability of the food (20, 21). However, because food intake did not increase as much in lean as in obese rats, lean rats may not be as sensitive to palatability (3). Body composition changed mainly in obese rats, in which residual body mass and fat mass significantly increased; this finding explained most of the slightly greater body weights in the butter-fed obese rats by the end of the study. The absence of significant changes in body weight after long-term manipulation of dietary fat, for up to several months, has been observed repeatedly (5–7, 22). Either a very long-term feeding (7) or a very high-fat content in the diet, ie, >55% of total energy intake (4, 8, 23), seems to be required to induce differences in body weight gain in rats fed different dietary fats, even in spontaneously obese rats (24). Such observations correspond with the concept that individuals strongly defend a set point for their body weight during dietary manipulations, even in obese rats, although their body weight set point is higher than that of lean rats (25).

Butter feeding induced no significant increase in the proportion of fat (ie, in adiposity), and there were no significant changes in the distribution of fat between the various depots in both lean and obese rats. This result confirms the lack of effect of dietary fat on adipocyte differentiation previously observed in lean Wistar rats (7) and confirms that dietary fats may not affect adipocyte differentiation, growth, or hormonal binding in vivo as it does in vitro (8, 9, 26, 27).

**Basal metabolism**

RQ values and metabolic rates were measured before the test meal after an overnight fast, ie, in a state close to basal metabolism (28). During this period, the RQ was higher in obese than in lean rats, indicating that obese Zucker rats rely less on lipid oxidation than do lean rats, a mechanism previously suggested to play a role in the development of their obesity (29, 30). In addition, as already observed (31–33), the metabolic rate was also higher in obese than in lean rats. Butter feeding increased the metabolism of obese rats, which is consistent with an increase in residual body mass. Whether this increase in body mass was entirely due to the increase in residual body mass weight or involves an additional increase in the specific metabolic intensity of the organs remains to be elucidated by specific studies. However, because no increase in basal metabolism was observed in butter-fed lean Zucker rats in which body composition remained unchanged, it is probable that the increase in residual body mass weight was the main factor responsible for the increase in basal metabolism observed in the butter-fed obese rats. This increase in basal energy expenditure may have enabled the butter-fed obese rats to limit their weight gain. In addition, the type of dietary fat did not affect basal RQ in lean or obese rats. This indicates a lack of effect of dietary fat on lipid mobilization and utilization after an overnight fast.

**Thermic effect of feeding**

The measurement of TEF in obese subjects has provided conflicting results (34). In the present study, TEF did not differ in obese and lean rats and was not affected by the quality of the dietary fat. This result agrees with previous results in obese Zucker rats fed a standard maintenance diet (32) and suggests that a reduced TEF is probably not involved in the development of obesity in Zucker rats. Rather, the low rate of basal lipid oxidation indicated by the high premeal RQ observed in obese rats may be the main mechanism responsible for the accumulation of fat in these rats. However, the meal-induced increase in RQ was significantly smaller in obese than in lean rats, thus reducing the difference in RQ observed in the postabsorptive state
during the postprandial period. In addition, the specific measurement of the postprandial rates of glucose and lipid oxidation showed that the butter-fed obese rats oxidized more lipids than did the other rats, particularly during the 3 h that immediately followed feeding. Therefore, it seems that in these rats the fatty acids supplied by butter are better oxidized than are those supplied by soybean oil, a phenomenon that may partly compensate for the usual defective fat oxidation in obese Zucker rats. This phenomenon, together with the increase in resting metabolic rate, prevented an excessive body weight gain in the butter-fed obese rats. Butter contains a larger proportion of short- and medium-chain fatty acids than does soybean oil (≈8%). Because short- and medium-chain fatty acids are more soluble than are long-chain fatty acids, they are brought to the liver instead of being absorbed through the lymphatic system and then delivered directly to the fat pads. As a result, they are theoretically prone to induce a higher postprandial fat oxidation than are long-chain fatty acids (35). The observation that liver weight increased in both lean and obese rats may indeed reflect the fact that the liver stored more lipids in butter-fed than in soybean oil–fed rats. The handling by the liver of part of the fatty acids supplied by the butter-containing meals may potentially favor their oxidation. Accordingly, Kaunitz (36) found that the weight of normal and obese subjects diminished when long-chain fatty acids were replaced with medium-chain fatty acids in their diets.

**FIGURE 4.** Mean (±SEM) energy expenditures and respiratory quotients during the preexercise (pre-ex) period (30 min before the start of exercise), the exercise (Ex) period (last 30 min of the 1-h exercise at steady speed), the acceleration (Acc) period (last 15 min of the 30-min acceleration), and the recovery period (30 min after the first 30 min after termination of exercise). Values within the same group with different superscript letters are significantly different, *P* < 0.05.
Energy expenditure during activity

The measurement of glucose and lipid oxidation before and during bursts of spontaneous activity showed that whatever the delay between the onset of the meal and the occurrence of activity, fatty acids supplied ~50% of the energy required to fuel muscular efforts in all groups. This indicates that mobilization of lipids during usual periods of activity is not defective in obese rats and thus may not be a factor in the genesis of obesity.

During the first hour of treadmill exercise, all rats had an RQ < 0.86, which was consistent with a relatively high-fat diet and exercise of moderate intensity. Analysis of glucose oxidation and lipid oxidation during steady exercise showed that both glucose oxidation and lipid oxidation increased in obese rats as a result of a higher metabolism during exercise. During peak exercise, as a result of the diminution of the difference in metabolism between lean and obese rats, there were no further differences in glucose oxidation and lipid oxidation between lean and obese rats. Together, these results confirm the conclusion, in regard to spontaneous activity, that exercise-induced lipid oxidation is not impaired in obese rats. During steady exercise, the only significant difference observed was that glucose oxidation was lower in lean rats fed the butter diet than in the other rats. In parallel, lipid oxidation tended to increase. This observation shows that the working muscles of these rats rely less on carbohydrates and more on lipids than do the working muscles of the rats in the other groups, which was already suggested during the periods of spontaneous activity. A more efficient transport of fatty acids from adipocytes and liver to muscles may be responsible for this phenomenon (37, 38).

Again, this phenomenon could be related to a larger content of short-chain, more labile, and more soluble fatty acids in the adipose tissue of the lean rats fed the butter diet or to additional release of fatty acids from the liver. On the other hand, butter feeding may also have favored the deposition of fat stores within and around muscular fibers, a phenomenon observed in trained subjects (39, 40) and known to improve the utilization of lipids by working muscles during exercise.

Conclusion

The present results show the importance of the quality of dietary fats in the control of fat metabolism, at rest and during exercise. Soybean oil–fed obese Zucker rats showed an inability to use fat in the resting state. This deficiency was lowest in obese rats fed the butter diet. Although the use of fats such as butter may promote adverse cardiovascular effects in humans, the results from the present study suggest that in some forms of obesity, fats derived from butter might be more readily oxidized than fats derived from soybean oil. Furthermore, exercise of moderate intensity seems to be sufficient to promote fat oxidation and should be preferred to exercises of greater intensity during which the proportion of oxidation of lipids to that of carbohydrate may be less favorable. A delay should also be preserved between the end of the exercise and feeding to favor the continuation of an increased rate of lipid oxidation promoted during exercise. In lean rats, butter feeding increased lipid oxidation by working muscles, therefore suggesting that butter feeding provided more readily available fatty acids to be used during exercise. The precise mechanisms and possible consequences of this observation on performance and endurance, as well as on energy balance, appear to be worth further investigation.

REFERENCES