MINIREVIEW

Dietary Fat Type and Level Influence Adiposity Development in Obese but Not Lean Zucker Rats (44265)

M. Y. LOH, W. P. FLATT, R. J. MARTIN, AND D. B. HAUSMAN[1]

Department of Foods and Nutrition, University of Georgia, Athens, Georgia 30602

Abstract. **The development of obesity is influenced by a variety of factors including genetics and dietary fat type and level. To examine the interaction between these factors, male lean and obese Zucker rats (5 weeks initial age) were fed either a low-fat (15% calories) or one of two high-fat diets (65% calories; predominant fat source of either soybean oil or palm olein) for 8 weeks. Body weight, food intake, indirect calorimetry, and body composition determinations were performed. As expected, food intake, body weight, feed efficiency, oxygen consumption, heat production and carcass lipid were all significantly higher in obese compared to lean rats. Dietary fat level and/or type influenced body weight gain, oxygen consumption, heat production, energy balance, and carcass weight and lipid content in the obese but not in the lean Zucker rats. Oxygen consumption and carcass weight were increased approximately 25% and 10% respectively in obese rats fed either of the two high-fat diets as compared to those fed the low-fat diet. The type of fat fed in the high-fat diets also influenced body weight gain, heat production, energy balance, and carcass lipid content of the obese rats. Body weight gain and carcass lipid content were increased (16%–17%; $P < 0.005$) in obese rats fed the high-fat palm olein diet as compared to those fed the low-fat diet. These parameters were not increased in obese rats fed the high-fat soybean oil diet. In contrast, indirect calorimetry measurements indicated a moderate increase in heat production (Kcal/effective body mass/day; 14.5%) and decrease in energy balance (44.8%) in the obese rats fed the high-fat soybean oil diet as compared to those fed the low-fat diet. Energy expenditure and lipid accumulation were negligibly influenced by dietary fat level or type in the lean Zucker rats. The differential response of the lean and obese Zucker rats to this short-term dietary manipulation demonstrate that genetic background can influence an individual's response to dietary fat type and level. The genetically obese Zucker rat appears to be a good model for further studies of high-fat diet–induced obesity.** [P.S.E.B.M. 1998, Vol 218]

Offering animals diets high in fat is known to result in increased body weight and the subsequent development of obesity (1). Hyperphagia (2, 3) and decreased energy expenditure (*via* changes in diet-induced thermogenesis) (4) are believed to be contributing factors to the development of high-fat diet–induced obesity. Whether the type of fat fed in a high-fat diet has an independent influence on these physiological responses and the subsequent development of obesity has been difficult to ascertain. Previous studies of the obesity-inducing potential of different types of dietary fat have produced inconsistent results. An intake of a diet high in saturated as compared to unsaturated fats has been reported to either increase (5), decrease (6) or have no effect (7–12) on final body weight or weight

[1] To whom requests for reprints should be addressed at Department of Foods and Nutrition, The University of Georgia, Dawson Hall, Rm. 269, Athens, GA 30602. E-mail: dhausman@fcs.uga.edu

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gain. Likewise, higher intakes of saturated as compared to unsaturated fatty acids have alternatively been associated with either an increase (12, 13) or decrease (7, 14) or no alteration (8–11) in body fat accumulation. These discrepancies may be due to a variety of factors including animal age, the duration of the diet, and individual differences in response to the dietary manipulation.

The subtle differences in metabolic response induced by the various types of dietary fat could perhaps be potentiated if imposed on a genetic background of altered energy balance. The genetically obese Zucker rat is a model of early onset obesity (15). Obesity in the fatty strain of Zucker rats is inherited due to an autosomal recessive trait at the single gene mutation *fa* (for fatty), a missense mutation of the leptin receptor (16, 17). Recent evidence suggests that the defects in energy intake and expenditure in the obese Zucker rat are secondary to defective leptin-receptor signaling and chronically increased neuropeptide Y (NPY) activity (18). Thus, positive energy balance and excessive fat deposition in these animals are the results of genetically-induced alterations in both energy intake and energy expenditure.

This study was designed to test the hypothesis that different levels and types of dietary fat and genetic background can interact to influence energy balance and the development of obesity. Thus, we determined the influence of level and type of dietary fat on body weight, food intake, energy expenditure, and body composition in lean and obese Zucker rats.

Materials and Methods

Animals and Diets. Forty male obese (*fa/fa;* OB) and lean (*Fa/?*; LN) Zucker rats obtained from the UGA colony were maintained on a nonpurified diet (Purina Rodent Chow, Ralston Purina, St. Louis, MO) from weaning until phenotype verification at 5 weeks of age. At that time they were ranked by body weight and randomly assigned by phenotype to one of three dietary groups of six to seven rats each. Diets containing either 65% calories as fat (high-fat soybean oil or high-rat palm olein) or 15% calories as fat (low-fat) were fed *ad libitum* for 8 weeks (Tables I and II). Fat was added to the diet at the expense of carbohydrate. The higher fiber content of the high-fat diets was necessary to maintain an acceptable consistency (19). All diets contained 20% calories as protein and adequate vitamins and minerals. Diets were prepared weekly and stored at 4°C. The diet was analyzed by the Poultry Nutrition Lab of the University of Georgia for caloric gross energy, percentage of moisture, percentage of total lipid and fatty acid composition. Body weight and food intake were monitored weekly. All protocols for animal use were approved by the University of Georgia Institutional Animal Care and Use Committee.

Energy Expenditure. Total daily energy expenditure was measured in six rats from each group for 2 days during Week 7 of the study. Twenty-four-hour energy expenditure

Table I. Composition of the Experimental Diets

	Low Fat	HFSO[d]	HFPO[d]
	g/100g diet		
Casein	19.5	23.5	23.5
Cornstarch	53.6	15.0	15.0
Sucrose	10.0	2.7	2.7
Fat:			
Soybean oil	6.85	35.8	5.8
Other	—	—	30.0
Fiber[a]	5.0	17.9	17.9
Minerals[b]	3.5	3.5	3.5
Vitamins[b]	1.0	1.0	1.0
L-Cysteine	0.3	0.3	0.3
Choline chloride	0.25	0.25	0.25
Caloric gross energy (Kcal/g)	4.421	6.252	6.095
% dry matter[c]	92.8	95.8	96.3

[a] Celufil, non-nutritive bulk (United States Biochemical Co.; Cleveland, OH).
[b] AIN-93 mineral and vitamin mixtures (32).
[c] Diet compositional gross energy analysis (Poultry Nutrition Laboratory, Dept. Poultry Science, University of Georgia).
[d] Abbreviations: HFSO, high fat soybean oil; HFPO, high fat palm olein.

Table II. Fatty Acid Composition of Oils and Fats Used in Experimental Diets (as % of Total Fatty Acid)

	Low Fat[a]	HFSO[a,b,d]	HFPO[a,c,d]
C 12:0	0.02	0.08	0.45
C 14:0	0.11	0.13	1.11
C 16:0	10.41	11.09	31.80
C 16:1	0.14	0.11	0.50
C 17:0	0.13	0.14	0.00
C 18:0	3.37	3.97	3.75
C 18:1	31.46	23.94	39.96
C 18:2	46.13	52.01	20.25
C 18:3	5.84	7.41	1.47
C 20:0	0.79	0.54	0.27
C 20:2	0.06	0.04	0.05
Other	1.54	0.54	0.31
Total	100.0	100.0	100.0

[a] Values based on compositional analysis of diet samples (Poultry Nutrition Laboratory, Dept. Poultry Science, University of Georgia).
[b] ICN Biochemicals (Costa Mesa, CA).
[c] Fuji Vegetable Oil, Inc. (Savannah, GA).
[d] Abbreviations: HFSO, high fat soybean oil; HFPO, high fat palm olein.

was measured using a computer controlled indirect calorimeter with ten open circuit respiration chambers (Oxymax; Columbus Instrument, Columbus, OH). An infrared analyzer was used to measure the carbon dioxide concentration, and an Oxymax oxygen sensor battery was used to measure oxygen concentration. A mass flow controller measured the air flow. Average oxygen consumption, average carbon dioxide production, respiratory quotient, and average heat production after adjustment for metabolic body size ($BW^{0.75}$) were then determined. Energy balance measurement was based on subtraction of heat production (energy expenditure) from digestible energy intake, assuming

negligible energy losses as combustible gas and urine. Other parameters such as chamber temperature, water lick counts, and feeding activity counts were also recorded.

The determination of digestible dry matter was conducted during Week 7. Feces from each group of rats were collected over 24-hr periods for 2 days and dried overnight at 105°C to determine fecal dry matter. Apparent digestible dry matter was calculated as the difference between food intake dry matter and fecal dry matter.

After 8 weeks on the experimental diets, the animals were overdosed with sodium pentobarbital after fasting overnight. The inguinal, retroperitoneal and epididymal fat pads were removed and weighed. The remainder of the carcass (minus the GI tract) was stored at −20°C for subsequent determination of carcass composition.

Body Composition. Body composition analysis was performed on stored carcasses according to the method of Harris and Martin (20). Briefly, frozen carcasses were autoclaved in individual sealed beakers for 1 hr at 121°C. When cool, each carcass was ground in a blender with water. The slurry was then homogenized and samples of the homogenate taken for water, ash, and fat analyses. Water content was determined by observing the difference in weight of triplicate aliquots of the homogenate before and after drying to a constant weight (85°C for 48 hr). Ash was measured by subsequent ashing of the same samples in a furnace at 600°C for 12 hr. Lipid was determined gravimetrically after extraction of the homogenate for chloroform-methanol and evaporation of the extract to constant weight under a fume hood. Protein was estimated by subtracting the weight of lipids and ash from that of the dry matter.

Data Analysis and Statistics. The effects of diet treatment and phenotype on growth performance, energy expenditure, and body composition were assessed by two-factor analysis of variance procedures (SuperANOVA program for the PC). Comparison between the means was accomplished using the Least Squares Means procedure (21). Differences were accepted as significant with a P-value < 0.05.

Results

Body weight of Zucker rats throughout the 8-week study period is shown in Figure 1. Phenotype differences in initial body weight were observed, and body weight was significantly greater (1.5-fold) between the two phenotypes (lean versus obese) throughout the entire study period. Growth performance during the study period is summarized in Table III. After 8 weeks on the experimental diets, final body weight of obese rats fed the high-fat palm olein diets was approximately 12% greater than that of obese rats fed the low-fat diet. This increase was statistically significant (P < 0.005). Final body weight of obese rats fed the high-fat soybean oil diet was intermediate between that of obese rats fed the low-fat diet and those fed the high-fat palm olein diet. Similarly, obese rats fed the high-fat palm olein diet had a greater body weight gain during the 8-week dietary period than obese rats fed the low-fat or high-fat soybean oil diets (16% and 7%, respectively). In contrast, no significant differences in body weight gain or final body weight were observed between lean rats fed the three different dietary treatments.

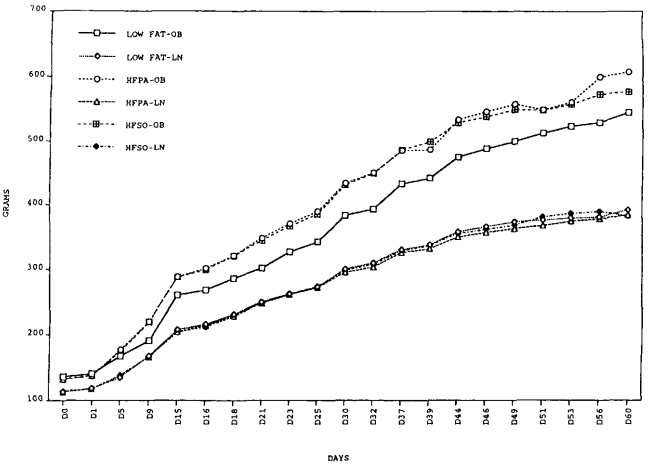

Figure 1. Body weight of lean and obese Zucker rats fed a low-fat diet or one of two high-fat diets for 8 weeks. Data points represent least square means for six to seven rats per diet group. Standard error of the means (SEM) ranged from 7–9 during Weeks 1–2 to 12–16 during Weeks 7–8. Abbreviations: LN, lean; OB, obese; HFSO, high-fat soybean oil; HFPO, high-fat palm olein.

Total food intake throughout the study period was 1.5-fold greater in obese versus lean rats (P < 0.0001) (Table III). Total food intake (Kcal) was also 19%–25% greater in obese rats fed the high-fat diets than in obese rats fed the low-fat diet (P < 0.05). When corrected for estimated digestible energy, intake throughout the 8-week study was similar for all obese rats. Nevertheless, feed efficiency was significantly greater in obese rats fed the high-fat diets than in obese rats fed the low-fat diet. Analysis of digestible energy intake on a weekly basis indicated a 20%–23% reduction in intake by the obese rats fed the high-fat soybean oil and low-fat diets during the final 2 weeks of the study (Fig. 2). Such a reduction in digestible energy intake was not observed in obese rats fed the high-fat palm olein diet. There were no diet-associated differences in total food intake, cumulative or weekly estimated digestible energy intake, or feed efficiency among the lean rats.

Energy expenditure parameters are shown in Table IV. Average O_2 consumption and CO_2 production were significantly higher in obese as compared to lean rats. In addition, obese rats fed the high-fat diets had 23%–27% greater average O_2 consumption than obese rats fed the low-fat diet (P < 0.05). A significant difference in respiratory quotient was observed between rats fed the high-fat diets as compared to those fed the low-fat diet (P < 0.05). As expected, respiratory quotient was highest with low-fat feeding. Significantly higher respiratory quotients were also observed in obese as compared to lean rats fed either the low-fat or high-fat palm olein diets. Respiratory quotient was not influenced by phenotype in rats consuming the

Table III. Growth Performance of Zucker Rats During 8-Week Period on a Low-Fat or One of Two High-Fat Diets

		Low-Fat[a]	HFSO[a,b]	HFPO[a,b]	SEM	*P*-value		
						TRT	PHE	TRT * PHE
Initial body weight	(LN)[b]	113[c]	113[c]	112[c]	7	NS[b]	0.0009	NS
(grams)	(OB)[b]	136[d]	132[d]	132[d]	7			
Final body weight	(LN)	393[c]	383[c]	385[c]	14–15	NS	0.0001	0.07
(grams)	(OB)	543[d]	576[d,e]	606[e]	14–15			
Body weight gain	(LN)	280[c]	270[c]	272[c]	14–15	NS	0.0001	0.05
(grams)	(OB)	408[d]	444[d]	474[e]	14–15			
Total food intake	(LN)	4630[c]	5265[c]	4971[c]	266–288	0.002	0.0001	0.09
(Kcal)	(OB)	6322[d]	7529[e]	7922[e]	266–288			
Estimated DE[b]	(LN)	4287[c]	4176[c]	3879[c]	212–229	NS	0.0001	NS
(Kcal)	(OB)	5880[d]	5857[d]	6229[d]	212–229			
Feed efficiency	(LN)	0.066[c,d]	0.066[c]	0.071[c,d]	0.003–0.004	NS	0.05	NS
(g gained/Kcal DE)	(OB)	0.069[c]	0.076[d,e]	0.077[d,e]	0.003–0.004			

[a] Values are least Squares Means ± SEM for six to seven rats per diet group. Estimated digestible energy intake for the 8-week dietary period is based on digestible dry matter determinations of (92.59, 93.007%), (79.31, 77.79%), (78.02, 78.62%) for low-fat, soybean oil, and palm olein (Lean, Obese) groups respectively.
[b] Abbreviations: LN, lean; OB, obese; HFSO, high-fat soybean oil; HFPO, high-fat palm olein; DE, digestible energy; NS, nonsignificant.
[c,d,e] Values within a parameter with different superscripts are significantly different ($P < 0.05$).

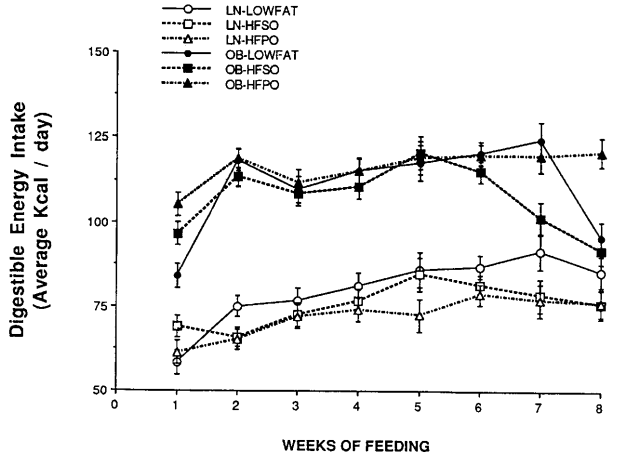

Figure 2. Digestible energy intake of lean (open figures) and obese (filled figures) Zucker rats fed a low-fat diet (○,●) or one of two high-fat (□,■; HFSO or △,▲; HFPO) diets for 8 weeks. The estimated digestible energy intakes were based on digestible dry matter determinations of (92.59, 93.01%), (79.31, 77.79%), (78.02, 78.62%) for low-fat, soybean oil, and palm olein (lean, obese) groups respectively. Data points represent least square means ± SEM for six to seven rats per diet group. Abbreviations: HFSO, high-fat soybean oil; HFPO, high-fat palm olein.

high-fat soybean oil diet. Heat production expressed on a whole-animal basis (Kcal/rat/day) was significantly greater in obese as compared to lean rats ($P < 0.0001$). Heat production (Kcal/rat/day) was not influenced by diet in the lean rats but tended to be greater in obese rats fed the high-fat soybean oil diet as compared to those fed the low-fat diet ($P = 0.07$). Heat production likewise tended ($P = 0.07$) to be greater in obese as compared to lean rats when expressed on the basis of metabolic body size (Kcal/$BW^{0.75}$/day), the usual manner of expressing energy expenditure. However, correction for metabolic size as $BW^{0.75}$ may not be the most appropriate means of expressing energy expenditure data in lean as compared to obese animals due to their drastically different body compositions. It has been suggested that expression per unit effective body mass is a more appropriate means of comparing energy parameters in lean versus obese rats (22). When expressed in this manner, a phenotype-associated difference was again observed, with heat production (Kcal/EBM/day) being greater overall in obese as compared to lean rats ($P < 0.0001$). Heat production per unit effective body mass also tended ($P = 0.08$) to be greater in rats fed the high fat soybean oil diets as compared to those fed the low-fat or high-fat palm olein diets. Within the obese rats, a 14% greater rate of heat production (Kcal/EBM/day) was observed in rats fed the high-fat soybean oil diet as compared to those fed the low-fat diet ($P < 0.02$). Digestible energy (DE) intake during the 2-day calorimetry study was higher overall in obese as compared to lean rats ($P < 0.0001$). However, a phenotype × diet interaction was observed with lean rats fed the high-fat palm olein diet consuming 33% less than their obese counterparts. During the calorimetry trial, digestible energy intake was similar between lean and obese rats fed the low-fat and high-fat soybean oil diets. Energy balance during the calorimetry trial, calculated by subtracting heat production (Kcal/day) from digestible energy intake (Kcal/day) was reduced 45%–53% in obese rats fed the high-fat soybean oil diets as compared to those fed the low-fat diet or the high-fat palm olein diet. Energy balance was not influenced by dietary fat level or type in the lean rats.

Energy balance is the result of prevailing energy intake and energy conditions. If energy intake and total energy expenditure are not equal for a given period, a change in body energy content will occur (23). As previously noted, both factors in the energy balance equation, intake and expenditure, were affected by the level and/or type of fat fed to the obese Zucker rats. Final carcass composition was likewise influenced by phenotype and by the level and type

Table IV. Energy Expenditure Parameters of Zucker Rats Fed a Low-Fat or One of Two High-Fat Diets for 8 Weeks

		Low-Fat[a]	HFSO[a,b]	HFPO[a,b]	SEM	*P*-value		
						TRT	PHE	TRT * PHE
O_2 consumption	(LN)[b]	6.88[c]	7.40[c,d]	6.66[c]	0.43	0.01	0.0001	0.05
(Liter/day)	(OB)[b]	8.54[d]	10.83[e]	10.51[e]	0.43			
CO_2 production	(LN)	6.47[c]	5.87[c]	5.13[c]	0.51	NS[b]	0.0001	NS
(Liter/day)	(OB)	8.70[d]	9.29[d]	9.07[d]	0.51			
CO_2/O_2	(LN)	0.93[c]	0.79[d,f]	0.76[d]	0.23	0.0001	0.0001	NS
(RQ)[b]	(OB)	1.01[e]	0.86[f]	0.86[f]	0.23			
Heat production	(LN)	34.2[c]	35.4[c]	31.7[c]	2.2	0.07	0.0001	0.07
(Kcal/rat/day)	(OB)	43.3[c]	52.7[d]	51.3[c,d]	2.2			
Heat production	(LN)	68.8[c]	71.9[c,d]	65.4[c]	3.7	NS	0.07	NS
(Kcal/$BW^{0.75}$/day)	(OB)	68.3[c]	80.2[d]	75.5[c,d]	3.7			
Heat production	(LN)	68.8[c]	71.9[c]	65.4[c]	4.0	0.08	0.0001	NS
(Kcal/EBM[a]/day)	(OB)	83.9[d]	98.2[c]	90.9[c,d]	4.0			
DE[b]/day	(LN)	76.3[c,e]	68.2[c,d]	64.7[d]	3.5	0.05	0.0001	0.005
(Kcal/day)	(OB)	82.8[e]	74.6[c,e]	96.8[f]	3.5			
Energy balance	(LN)	42.1[c,e]	32.7[c]	32.9[c]	4.1	0.005	NS	0.05
(DE-heat production)	(OB)	39.5[c,e]	21.8[d]	46.6[e]	4.1			

[a] Values are Least Square Means ± SEM for six rats per diet group. Indirect calorimetry was performed on individual rats for two consecutive 24-hr periods during the final week of the 8-week feeding trial. Effective Body Mass (EBM) is based on the computations of Refinetti (22). Energy balance is based on subtraction of heat production (Kcal/day) from digestible energy.
[b] Abbreviations: LN, lean; OB, obese; HFSO, high-fat soybean oil; HFPO, high-fat palm olein; RQ, respiratory quotient; DE, digestible energy; NS, nonsignificant.
[c,d,e] Values within a parameter with different superscripts are significantly different ($P < 0.05$).

of dietary fat (Table V). As expected, significant phenotype differences in carcass composition were observed between the lean and obese rats. Empty carcass weight as well as carcass dry matter, lipid, and protein contents were all significantly higher, and carcass ash content was significantly lower in obese as compared to lean rats. Carcass weight was influenced by diet in the obese but not in the lean rats. Obese rats fed either of the two high-fat diets for 8 weeks had significantly greater carcass weights than those fed the low-fat diet. Carcass dry matter and lipid content also tended ($P = 0.09$) to be influenced by dietary fat type and level in the obese but not in the lean rats. Within the obese phenotype, rats consuming the high-fat palm olein diet had significantly greater carcass dry matter and lipid content than obese rats fed the low-fat diet. Carcass lipid content was increased 17% in obese rats fed the high-fat palm olein diet as compared to obese rats fed the low-fat diet ($P < 0.05$), but was not different between obese rats consuming the low-fat diet and the high-fat soybean oil diet. Carcass ash and protein content were not influenced by the level or type of dietary fat fed to either the lean or obese Zucker rats.

Discussion

There is considerable evidence from both human and animal studies that genetics and high-fat diets are etiologic factors in obesity. Results of this study provide evidence that genetic background can interact with the level and type of fat in the diet to influence obesity development. In this

Table V. Carcass Composition of Zucker Rats Fed a Low-Fat or One of Two High-Fat Diets for 8 Weeks

		Low-Fat[a]	HFSO[a,b]	HFPO[a,b]	SEM	*P*-value		
						TRT	PHE	TRT * PHE
Carcass weight (g)	(LN)[b]	326.7[c]	295.7[c]	321.5[c]	11–12	NS[b]	0.0001	0.05
	(OB)[b]	407.5[d]	440.0[e]	455.0[e]	11–12			
Dry matter (g)	(LN)	86.4[c]	86.1[c]	89.3[c]	12–13	0.05	0.0001	0.09
	(OB)	206.6[d]	231.1[d]	267.6[e]	12–13			
Lipid (g)	(LN)	28.1[c]	26.6[c]	27.4[c]	7–8	0.09	0.0001	0.09
	(OB)	166.3[d]	167.4[d]	194.8[e]	7–8			
Ash (g)	(LN)	3.47[c,d]	3.58[c,d]	4.05[d]	0.2	NS	0.0005	NS
	(OB)	3.14[c,e]	2.72[e]	2.94[e]	0.2			
Protein (g)	(LN)	54.9[c]	55.9[c]	58.4[c,d]	7–8	NS	0.05	NS
	(OB)	58.8[c,d]	70.4[c,d]	78.2[d]	7–9			

[a] Values are least Square Means ± SEM for six to seven rats per diet group.
[b] Abbreviations: LN, lean; OB, obese; HFSO, high-fat soybean oil; HFPO, high-fat palm olein; NS, nonsignificant.
[c,d,e] Values within a parameter with different superscripts are significantly different ($P < 0.05$).

study, genetically obese rats fed high levels of palm olein (a saturated fat) manifested a positive energy balance, and a significantly greater fat deposition and body weight gain that obese rats fed either a low-fat diet or a diet containing high levels of soybean oil (a polyunsaturated fat). In contrast, no discernible effects of dietary treatments were observed in lean rats.

Our observations are consistent with the finding of Lemonnier *et al.* (24), that body weight is increased by a combination of high-fat diet and the obese gene in *fa/fa* rats. However, Lemonnier *et al.* (24) observed that body weight and body fat stores also increased, although to a lesser extent, in lean rats fed a high-fat diet. The diets fed to Zucker rats in our study were begun earlier in life (at 5 weeks of age) while the diets fed in the previous study were introduced later in life (at 5 months). In contrast to our findings of no influence of dietary fat type or level on weight gain, body composition, or energy parameters in lean rats, Vasselli and Maggio (25) observed that body weight gain was greater with consumption of a high-fat diet as compared to chow or a high-protein diet in both lean and obese rats when the diets were fed from 5 weeks to 28 weeks of age. It is possible that we would have likewise been able to see an influence of dietary fat level and/or type on weight gain or body lipid content had the lean rats been exposed to the diets for a longer time. The differential response of the lean and obese rats to this short-term dietary manipulation emphasizes the greater susceptibility of the genetically obese animal to diet-induced obesity. Thus, the interaction of dietary fat and genetics on the development of obesity may be influenced not only by level and type of high-fat feeding but also by animal age and the duration of feeding.

Diet-associated differences in both food intake and energy expenditure may contribute to the varied response of the obese rats to alterations in dietary fat level and/or type. The heightened obesity-inducing potential of the high-fat palm olein diet in the present study may have been due to a prolongation of the typical period of hyperphagia in the obese animal. Notably, hyperphagia was observed throughout the entire 8-week period of our feeding study, from the initial age of about 5 weeks until 13 weeks of age, in those obese rats fed the high-fat palm olein diet. In contrast, obese rats fed the high-fat soybean oil and low-fat diets exhibited a tendency toward a normalization of their food intake toward the end of the feeding trial. During the final week of the study, there was no significant difference in digestible energy intake between obese and lean rats consuming the low-fat diet. These observations are in partial agreement with those of Vasselli and Maggio (25) regarding the developmental pattern of hyperphagia in genetically obese Zucker rats. These investigators had observed hyperphagia in chow-fed genetically obese rats as early as the third week of life until about 15 weeks of age, where hyperphagia reached a peak or breakpoint. In addition, they noted that the breakpoint in hyperphagia occurred earlier, at approximately 10 weeks of age, in obese rats fed a high-fat diet. We also observed early hyperphagia in the obese rat; however, in our study a breakpoint in hyperphagia occurred after 5 weeks of feeding the high-fat soybean oil diet (at 10 weeks of age) or after 7 weeks (at 12 weeks of age) on the low-fat diets. In contrast, food intake in the obese rats fed the high-fat palm olein diet remained at a chronically high level throughout the 8-week feeding trial. It is interesting that obese rats fed diets high in unsaturated fat, predominantly soybean oil (present study) or corn oil (25), reduced their energy intake in response to the high-fat diets whereas those fed the diet high in saturated fat, predominantly palm olein, did not. This would suggest that the type of dietary fat can influence stimulatory/inhibitory processes involved in food regulation in the obese Zucker rat (18, 26) with a net result of sustained hyperphagia. This possibility needs to be explored in future, specifically designed studies. With regard to the present study, it is apparent that the increased adiposity attained in the obese rats fed the high-fat palm olein diets may be partly due to a higher intake of digestible energy. At the same time, food intake was similar among the lean rats; therefore, we failed to observe a dietary treatment difference in altering fat deposition in the lean rats.

Results of this study indicate that dietary fat as well as genetics can influence energy expenditure parameters and thereby contribute to adipose tissue lipid storage in obese Zucker rats. The pattern of substrate oxidation is a relevant issue to energy balance with the relative proportion of fat or carbohydrates oxidized being indicated by the respiratory quotient (RQ) (23). An RQ approaching 0.7 is indicative of a predominance of fat oxidation whereas an RQ approaching 1.0 is indicative of a predominance of carbohydrate oxidation. Both groups of high-fat (high-fat soybean oil and high-fat palm olein) fed obese rats had an RQ higher than 0.7, which would suggest a lower level of fat oxidation. This would further suggest a higher level of dietary fat energy available for partitioning to storage as fat. These observations are consistent with the recent suggestion that dietary fat may be shunted away from oxidation toward storage in obese Zucker rats (27). This suggestion was based on the observation of reduced oxidation of ^{14}C-oleic acid (dietary addition) by skeletal muscle and increased incorporation of ^{14}C-oleic acid into adipose tissue triglycerides of obese as compared to lean rats. However, that study utilized only a single fatty acid, oleic acid, for the direct measurement of nutrient flux in the lean and obese rats. It remains to be seen whether the partitioning of metabolic fuels could be differentially influenced by different types of dietary fats such as those used in the present study.

Although the influence of dietary fat type on energy partitioning *per se* was not determined in the present study, total body energy expenditure (heat production) was found to be consistently highest in those rats consuming the high-fat soybean oil diets. This trend was observed in both the lean rats and to a greater extent in their obese counterparts. Previous studies have reported that diets high in essential fatty acids increase thermogenesis in brown adipose tissue

(28, 29) and fatty acid oxidation in the liver (30, 31) in comparison to diets high in saturated fat. In any event, the higher heat production and thus moderately lower energy balance may further explain why the obese rats fed the high-fat soybean oil diets did not accumulate as much fat as the obese rats fed the high-fat palm olein diet.

Previous studies with Wistar rats suggested that the amount of fat in the diet appears to have a greater impact on body composition and obesity development than the type of fat (4). However, results of this study clearly demonstrate that the genetic background can influence an individual's response to dietary fat feeding. Within the fairly short (8-week) duration of this study, we were able to detect an impact of dietary fat type as well as dietary fat level on increasing body weight gain and lipid deposition in genetically obese Zucker rats, with a high saturated fat diet having the greatest effect. Energy accumulation effects of dietary fat level or type were negligible in the lean Zucker rats within this time frame. Subtle differences would undoubtedly become more readily apparent with a longer duration of high-fat feeding. The obese Zucker rat appears to be a good model for further studies of diet-induced obesity.

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