The Influence of Dietary Fat on the Lipogenic Activity and Fatty Acid Composition of Rat White Adipose Tissue

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The in vivo fatty acid synthesis rate, selected enzyme activities and fatty acid composition of rat white adipose tissue from animals fed semisynthetic diets of differing fat type and content were studied. All animals were starved for 48 hr and then refed a fat-free (FF) diet for 48 hr. They were then divided into three groups. One group was continued on the FF diet for 48 hr. Another group was fed a diet containing 44% of calories from corn oil (CO). The final group was fed a diet containing 44% of calories from completely hydrogenated soybean oil (HSO). The animals on the FF diet had a marked increase in adipose tissue fatty acid synthesis during the 96-hr feeding period (as measured by ^3H incorporation into adipose fatty acids). Addition of either CO or HSO to the diets did not significantly inhibit fatty acid synthesis in dorsal or epididymal adipose tissue. The activities of the enzymes' fatty acid synthetase, ATP-citrate lyase and glucose-6-phosphate dehydrogenase increased on the FF diet and generally were not inhibited significantly by the addition of either fat to the diets. Linoleic acid was the major polyunsaturated fatty acid (ca. 22%) in adipose tissue. Monounsaturated fatty acids (palmitoleic, oleic, *cis*-vaccenic) made up ca. 38% of the total adipose fatty acids, while saturated fatty acids accounted for about 32% (myristic, palmitic and stearic). White adipose tissue in mature male rats was a major depot for n-3 fatty acids. There were differences in the fatty acid composition of epididymal and dorsal adipose tissue, particularly in their content of long chain, polyunsaturated fatty acids with epididymal tissue containing more of these compounds than dorsal fat. The fatty acid composition of the white adipose tissue did not change significantly during fasting or 96 hr of refeeding the FF diets. The addition of HSO to the diet for 48 hr had little influence on the adipose tissue fatty acid composition, but the addition of CO to the diet caused a 7% increase in the dorsal adipose tissue linoleate content (as percentage of total dorsal adipose tissue fatty acids) within 48 hr compared to animals fed the stock diet and those starved for 48 hr. The fatty acid synthesis data indicated that adipose tissue in the rat can continue to be a source of de novo fatty acid synthesis in animals consuming high-fat diets.
***Lipids 22,* 338-344 (1987).**

Adipose tissue is a major site of fatty acid synthesis in rats (1-4) as well as other mammalian species (5-8). However, the regulation of fatty acid synthesis in adipose tissue is extremely complex as it is influenced by many intrinsic and extrinsic factors, such as diet (3,4,5,9-12), age (13,14), hormones (15-17) and heredity (18). There is uncertainty about the role of dietary fat on the suppression of fatty acid synthesis in adipose tissue (19-21).

The fatty acid synthetic rates in white adipose tissue have been measured using various fatty acid precursors such as ^{14}C-glucose (22,23), ^{14}C-acetate (24) and tritiated water (25,26). This latter substance has become the label of choice to measure fatty acid synthesis because it avoids the confounding influence of the various metabolic pools and the recycling phenomenon of the carbon precursors (27,28). Nevertheless, when studying fatty acid synthesis in the whole animal, one encounters the problem that adipose tissue, while actively synthesizing fatty acids, is a repository for fatty acids synthesized de novo in the liver (29) as well as for fatty acids of exogeneous origin. Some investigators have attempted to resolve this matter by suppressing liver fatty acid synthesis by diet (24) or drugs (29), or by using short incubation periods (25).

In rats fed stock diets ad libitum, six major fatty acids (30)—palmitic, palmitoleic, stearic, oleic, *cis*-vaccenic and linoleic—constitute about 90% of the total fatty acids present in white adipose tissue. Linoleic acid is the major polyunsaturated fatty acid of rat white adipose tissue (31). Under appropriate conditions dietary fatty acids can influence the fatty acid composition of adipose tissue (32-34).

We have previously reported the effect of feeding fat-free (FF) and fat-containing diets on liver and plasma fatty acid composition (35), as well as the activity of liver lipogenic enzymes in rats fed these diets (36). The objective of this research was to investigate some of the parameters affecting the regulation of white adipose tissue fatty acid metabolism by dietary fat in mature rats fed a semisynthetic diet. Here we present data on the synthetic rate, some enzymes of the fatty acid synthetic pathway and the fatty acid composition of epididymal and dorsal adipose tissue as a function of time on the FF and high-fat diets. Under the conditions used in this study it was found that high-fat diets, both saturated and polyunsaturated, did not suppress fatty acid synthesis in adipose tissue.

MATERIALS AND METHODS

Materials, animals and diets. Male, Sprague-Dawley rats, 100 to 150 g, were purchased from Bantin and Kingman (Fremont, California). They were fed a stock diet from Ralston-Purina (Richmond, Indiana) until they had grown to 400 to 500 g in weight and were 120 to 140 days old. The animals were then segregated into three groups of 12 to 22 rats each, starved for 48 hr and then refed a FF diet (75% of calories from carbohydrates) for 48 hr. After that, one group was continued on the FF diet for another 48 hr. A second group was placed on a synthetic diet in which 44% of the calories was supplied by corn oil (CO). The remaining group was fed a diet in which 44% of the calories was supplied by completely hydrogenated soybean oil (HSO), a gift of Durkee Foods (Cleveland, Ohio) for 48 hr. The composition of the three diets has been reported previously (35). The fatty acid composition of the diet is listed in Table 1. Animals had access to the diets ad libitum, except that initially the animals were

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TABLE 1

Fatty Acid Composition of Experimental Diets (Wt % of Total Fatty Acids)

Fatty acid[a]	Corn oil	Hydrogenated soybean oil
14:0	0.12	0.12
16:0	11.23	10.72
16:1(n-7)	0.10	—
17:0	0.14	0.25
18:0	2.06	86.37
18:1(n-9)	25.55	0.32
18:1(n-7)	1.04	—
18:2(n-6)	55.02	—
18:3(n-3)	0.42	—
20:0	0.46	0.55
20:1(n-9)	0.25	—
20:4(n-6)	0.09	—
22:0	0.16	—
24:0	—	0.11
Sum of trace components	2.96	1.21

[a]Fatty acids are designated by carbon chain length, number of double bonds and position of the first double bond from the methylene end of the molecule.

given approximately 3 ml of each diet by stomach tube. All animals consumed approximately the same number of calories regardless of diet group and appeared to find the diets palatable.

The animals were killed at 0, 24, 48, 72 and 96 hr after refeeding was begun. All animals received intraperitoneally tritiated H_2O (0.5 mCi/100 g body weight) one hr before they were killed. The tissues were removed and handled as previously described (37).

All organic solvents were obtained from Burdick and Jackson (Muskegon, Michigan). Purified fatty acid methyl ester reference standards were purchased from Nu-Chek Prep (Elysian, Minnesota).

Lipid extraction. Hydroquinone was added to all samples during processing. Three adipose tissue samples from each dietary group were extracted using $CHCl_3$-MeOH (2:1, v/v) by the procedures described previously (37) after being lyophilized to remove all traces of free tritiated water. This procedure was used to determine the percentage of fat present in the tissue based on the wet weight of the tissue. The total lipid extracts (TLE) were either prepared for transmethylation immediately or stored under nitrogen at −20 C until further processing. The remaining samples, which were the bulk, were lyophilized and transmethylated directly as dried tissue samples as described below.

Transmethylation. The transmethylations of the TLE to obtain the fatty acid methyl esters (FAME) were carried out using methanolic HCl (7%, w/w) (37) as previously described. For the direct transmethylation of the freeze-dried adipose tissue sample, the following procedure was used: Ca. 25 to 50 mg of dry tissue was placed directly in a 30-ml screw-capped culture tube to which a reflux condenser and drying tube were attached. Methanolic HCl (5 ml) was then added to the vial, and it was heated under reflux at 85 C for 2 hr. It was cooled to room temperature and processed similarly to the transmethylation of an aliquot of the TLE (37).

After transmethylation the FAME were extracted into hexane, purified and diluted in hexane to an appropriate concentration as described previously (37). They were then stored under N_2 at −20 C until analyzed by gas liquid chromatography (GLC) or by liquid scintillation count of the ^3H in the fatty acid moiety.

GLC of FAME. The FAME samples were analyzed as described previously (37) on fused-silica capillary columns coated with SP-2340 (Supelco, Bellefonte, Pennsylvania). Samples were chromatographed on a Perkin-Elmer Sigma 2000 coupled to a P-E 7500 computer loaded with a Chrom 3 data analysis program.

The quantitative accuracy of the GLC procedures was evaluated by using either purified single FAME or reference mixtures selected to cover the range of FAME present in the experimental samples. Accuracy of the analysis was estimated to be within 5% for the major components (greater than 10% of the total FAME in the sample) and within 10% for the minor components in the samples.

The cholesterol extracted into the hexane washes of the transmethylation procedure was not separated from FAME prior to injection of the samples in the chromatograph. However, cholesterol is largely decomposed during acidic transmethylation (38), and free cholesterol does not elute as a discrete peak from an SP-2340 column under the conditions used in this work (37).

The compositions (wt %) of the FAME in the rat samples were derived from the area percentages of the chromatograms as described previously (37). A chromatogram had between 30 and 50 discrete FAME peaks. Most minor peaks individually contributed less than 0.1% to the total area and were not identified with confidence. The major identified components usually comprised 97% to 99% of the total FAME present in the sample. For convenience, only selected major fatty acids are listed in the tables. The minor fatty acids are collectively presented as "sum of trace components," which varies from 4-6% of the total fatty acids present in the samples. Both identified and unidentified components are grouped together in this category.

Enzyme analysis. Adipose tissue samples were homogenized at 0 C in 4 volumes of HEPES (0.2 M)/EDTA (0.1 mM)/2-mercaptoethanol (10 mM) buffer, pH 7.4. The homogenates were centrifuged at 20,000 × g for 1 hr at 4 C. Supernatants recovered after this centrifugation were used for enzyme and protein determinations. Activities of fatty acid synthetase (FAS) were determined as previously reported (39). A unit of FAS is taken as the activity of enzyme required to synthesize 1 nmol of palmitic acid (equivalent to the oxidation of 14 nmol of NADPH)/min at 30 C. The activity of ATP-acid citrate lyase (ACL) was determined by the method of Linn et al. (40). One unit is defined as the activity necessary to catalyze the oxidation of 1 μmol NADH/min. Glucose 6-phosphate dehydrogenase (G6PDH) activity was determined by the procedures described previously (39). One unit of G6PDH represents the activity of enzyme necessary for the production of 1 μmol NADPH/min at 30 C. Activities of all enzymes are expressed as mU/mg protein.

Protein contents of tissue homogenates were determined by the method of Lowry et al. (41). All statistical

analysis of the data were done using the two-tailed student's t-test.

RESULTS

Starving the animals for 48 hr suppressed fatty acid synthesis and lipogenic enzyme activities in white adipose tissue compared to the animals fed the stock diet ad libitum, but had no influence on the fatty acid composition of the adipose tissue. Refeeding a FF diet stimulated adipose tissue lipogenic activity markedly by 48 hr to levels above that found in rats fed the stock diet and again without influencing the fatty acid composition of this tissue. Figure 1 presents the time course of fatty acid synthesis in rat dorsal adipose tissue. The animals on the FF diet exhibited an almost linear increase in fatty acid synthesis during the course of the study. No significant change in the rate of synthesis in the dorsal fat was detected when either corn oil or hydrogenated soybean oil was added to the diet at 48 hr. However, the variance in the experimental values was much larger in the groups receiving the high-fat diets. Nevertheless, there were no significant differences between the means of the fatty acid synthesis rates among any of the three diet groups at 72 or 96 hr.

Figures 2, 3 and 4 show the level of activity of the three enzymes measured in dorsal adipose tissue for the same time points as in Figure 1. Again, while individual variations were large, generally no significant differences were detected between the three dietary groups at 72 and 96 hr with the exception that animals on the HSO diet had suppressed FAS activity at 96 hr compared to animals on the FF and CO diets. All three enzymes showed increased activity after refeeding a FF diet compared to animals on the stock diet or those starved for 48 hr.

Epididymal adipose tissue enzymes were also investigated and showed approximately the same pattern as found in the dorsal tissue except that the activity levels were somewhat lower than those observed in the dorsal fat. The incorporation of ^3H into the fatty acids of epididymal fat was also about 50% lower than that observed

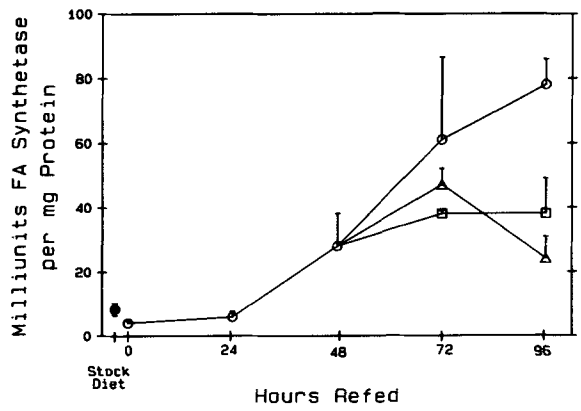

FIG. 2. Effect of different diets on the fatty acid synthetase activity in rat dorsal adipose tissue as a function of time on the diets. Symbols are the same as those in Fig. 1. The only difference that was statistically significant from the fat-free diet group was the 96-hr point for the hydrogenated soybean oil fed group, where $p < 0.01$.

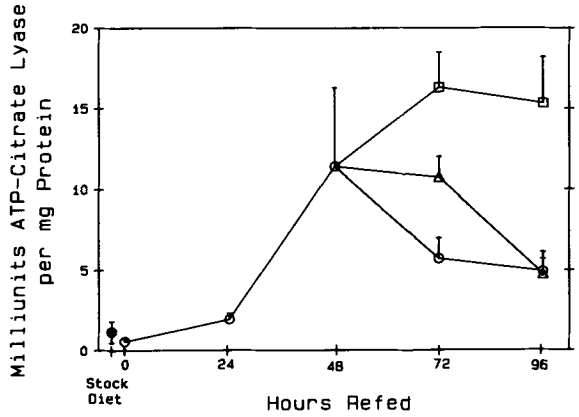

FIG. 3. Effect of different diets on ATP-citrate lyase activity in rat dorsal adipose tissue as a function of time on the diets. Symbols are as given in Fig. 1. Trends did not reach statistical significance during the 96-hr feeding period.

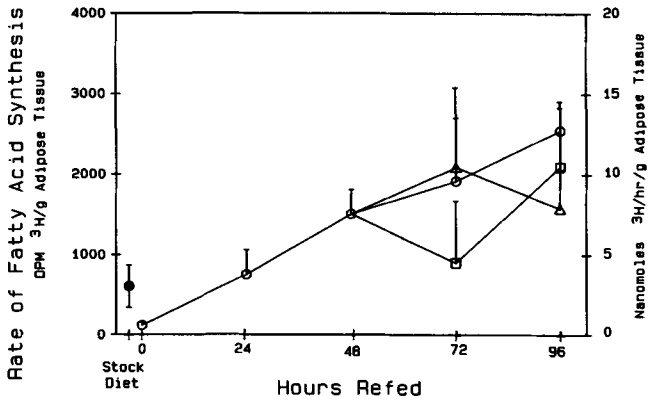

FIG. 1. Time course of fatty acid synthesis rate in dorsal adipose tissue from rats fed the three experimental diets. Symbols used are ●, stock diet; ○, fat-free diet; □, corn oil diet; △, hydrogenated soybean oil. Points are the means ± S.E.M. for groups of four to six animals. No statistically significant differences were detected between synthetic activities in animals on the fat-free diet and those on either the corn oil or hydrogenated soybean oil diets.

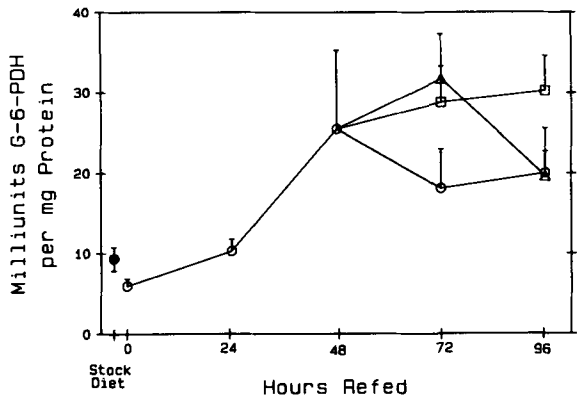

FIG. 4. Effect of different diets on the glucose-6-phosphate dehydrogenase activities in rat dorsal adipose tissue as a function of time on the diets. Symbols are the same as given in Fig. 1. No significant differences in the levels of enzyme activity were found among the tissues from animals in the three dietary groups.

in dorsal fat at equivalent time points. Actual data for the epididymal fat are not shown.

Table 2 gives the fatty acid composition of rat dorsal fat as a function of time and type of diet. There is relatively little change in the fatty acid composition between the animals starved for 48 hr and those refed a FF diet for 96 hr. There is a trend toward a reduction in the proportion of linoleate in adipose tissue after 96 hr on the FF diet, but this trend did not reach statistical significance. The only statistically significant change in any of the fatty acids in dorsal fat between the control animals and those on the FF diet was the 0.7% increase in *cis*-vaccenic acid at 96 hr.

When corn oil was added to the animals' diet after 48 hr on the FF diet, there was a statistically significant increase in the proportion of linoleate in dorsal fat. After 96 hr (48 hr on the CO diet) the linoleate level was 9% above that observed in the dorsal fat of the animals on the FF diet and 7% above the level in the dorsal fat of the animals starved for 48 hr. Other fatty acids showed statistically significant changes at 48 hr on the CO diet as well (see Table 2). Conversely, when saturated fat was added to the diet, there was little or no change in the fatty acid composition of the dorsal fat. Slight increases in the mean values for stearate, oleate and *cis*-vaccenic acids were noted in the dorsal fat of the animals fed saturated fat compared to animals on the stock diet or those starved for 48 hr. However, except for *cis*-vaccenic acid, the values were not significantly different at 96 hr.

It can also be observed from Table 2 that adipose tissue in the rat contains less than 1% arachidonic acid. Indeed, there is generally less arachidonic acid in adipose tissue than 22:6(n-3).

Table 3 lists the fatty acid composition of epididymal fat as a function of time and type of diet. Epididymal adipose tissue contains significantly larger proportion of polyunsaturated fatty acids than dorsal fat. In animals fed stock diets, the ratio of archidonic acid to the sum of n-3 20 and 22-carbon fatty acids is 0.34, while in epididymal fat this ratio is 0.25. Thus, there is almost 30% more n-3 fatty acids in epididymal fat proportionally to arachidonic acid than in dorsal fat.

DISCUSSION

It is generally agreed that dietary linoleic acid suppresses liver fatty acid synthesis (1,4,9,42-45), but several previous reports suggest that saturated fat does not suppress liver fatty acid synthesis (9,10,21,43,45-47). We recently reported (36) that in starved refed rats, high saturated-fat diets suppressed liver fatty acid synthesis equivalent to linoleate-containing diets. Some investigators, using various feeding periods (9,48), have suggested that fatty acid synthesis in adipose tissue is not inhibited by dietary fat. Reports on the enzymes of fatty acid synthesis indicate that rat adipose tissue fatty acid synthetic enzymes (usually measured in the epididymal fat pad) are not suppressed by dietary fat (5,10,17,21,49), although these studies used feeding periods and animals of ages different from those in this work. No consistent suppression of fatty acid synthesis was noted here in either dorsal or epididymal adipose tissue, even though linoleic acid was found to be rapidly incorporated into adipose fat. This suggests that other modulators of fatty acid synthesis, such as blood glucose levels, may play an important role in triggering fatty acid synthesis in adipose tissue.

Recently Gandemer et al. (25), using tritiated water and short incubation times to minimize the contribution from fatty acids synthesized de novo by the liver, indicated that in rats on a low-fat diet (0.3%) only 27% of the total fatty acids was synthesized in total body adipose tissue, whereas 42% of the fatty acid synthesis took place in the liver. Earlier work by Clarke and coworkers (4,9) and Romsos and Leveille (19) in meal-fed rats suggested that as much as 70% of the de novo fatty acid synthesis in the rat took place in the adipose tissue. High levels of dietary fat almost totally suppress liver fatty acid synthesis (9,21,36,46,50), and there is little if any transport of de novo synthesized fatty acids in the circulation by this time (35). Thus, the contribution of fatty acids synthesized in the liver to adipose tissue fatty acid stores should be minimal in rats on a high-fat diet.

It may be that a fat-fed animal synthesizes fatty acids primarily in the adipose tissue while those on a very low or fat-free diet synthesize fatty acids primarily in the liver with, perhaps, a substantial synthetic contribution from the adipose tissue. Factors including diet, age, sex, strain and caloric intake probably all affect the site of fatty acid synthesis, so considerable caution must be exercised in assigning the relative contribution of the liver or adipose tissue to de novo fatty acid synthesis in the rat. Meal feeding vs ad libitum feeding will also affect this ratio (48).

The fatty acid compositional data shown in Tables 2 and 3 were obtained by direct transmethylation of the lyophilized tissue. No compositional differences in the adipose fatty acid data could be detected between portions of the same adipose tissue sample processed by direct transesterification of the lyophilized tissue or by transesterification of the TLE from that tissue. The direct method was simple and quantitative, and saved considerable time and effort by avoiding the initial extraction step using $CHCl_3$/MeOH. This method works well for adipose tissue probably because adipose tissue is about 90% triglyceride and, hence, there are few proteins, carbohydrates or other nonlipoidal substances present to interfere with the reaction. Other investigators have also recently proposed one-step transesterification procedures (51,52) applicable to a variety of tissues. However, no attempt was made in this work to determine if this method was applicable to other tissues.

Palmitic, palmitoleic, stearic, oleic, *cis*-vaccenic and linoleic acids constitute almost 90% of total fatty acids (30,32,53) in adipose tissue. This may be true regardless of diet (30,31,54), which suggests that there are regulatory mechanisms controlling the fatty acid composition of this tissue other than the fatty acid composition of the diet. By comparing Tables 2 and 3 one can find subtle differences between the fatty acid compositions of the dorsal and epididymal fat. They may be due to differences in the amounts of phospholipids present in the two tissues, but any physiological significance of these differences remains obscure.

While dietary linoleic acid causes a rapid increase in the linoleic acid content of white adipose tissue, a high level of stearic acid in the diet has little influence on the content of this fatty acid in adipose tissue. The stearic acid level in adipose tissue is low, presumably due to the active Δ-9 dehydrogenase present in adipose tissue (33,54).

TABLE 2

Fatty Acid Composition of Rat Dorsal Adipose Tissue (Wt % of Total Fatty Acids)

Fatty acid[a]	Ad-libitum fed stock diet (n = 5)	48 Hr starved 0 Hr (n = 4)	Fat-free, high carbohydrate diet				Corn oil diet (after 48 hr on FF diet)		Saturated fat diet (after 48 hr on FF diet)	
			24 Hr (n = 5)	48 Hr (n = 5)	72 Hr (n = 4)	96 Hr (n = 4)	24 Hr (n = 4)	48 Hr (n = 4)	24 Hr (n = 6)	48 Hr (n = 6)
14:0	1.98 ± 0.13	1.96 ± 0.13	1.92 ± 0.11	2.03 ± 0.25	1.84 ± 0.08	1.97 ± 0.13	1.72 ± 0.10	1.45 ± 0.32[b]	1.94 ± 0.14	1.89 ± 0.10
16:0	25.92 ± 1.86	25.24 ± 0.78	24.71 ± 1.19	25.89 ± 1.86	25.25 ± 0.44	25.78 ± 1.08	24.31 ± 1.80	22.33 ± 2.48[b]	24.92 ± 1.21	25.33 ± 1.33
16:1(n-7)	5.13 ± 1.26	3.85 ± 0.43	4.42 ± 1.05	5.07 ± 0.91	4.78 ± 0.68	5.73 ± 0.42	4.91 ± 0.97	3.30 ± 1.11[b]	5.31 ± 0.90	5.09 ± 0.94
18:0	4.85 ± 1.26	5.10 ± 0.55	4.97 ± 0.49	5.06 ± 0.33	5.08 ± 0.24	4.57 ± 0.18	4.70 ± 0.31	4.74 ± 0.13	5.06 ± 0.37	5.06 ± 0.61
18:1(n-9)	29.63 ± 1.61	29.17 ± 1.08	28.72 ± 1.20	29.85 ± 0.93	29.50 ± 1.30	29.60 ± 0.49	31.57 ± 1.32	29.89 ± 0.88	30.71 ± 1.31	30.31 ± 0.23
18:1(n-7)	3.58 ± 0.22	3.89 ± 0.12	4.07 ± 0.21	4.03 ± 0.29	4.05 ± 0.19	4.34 ± 0.24[b]	3.90 ± 0.15	3.42 ± 0.50	4.33 ± 0.53	4.30 ± 0.47
18:2(n-6)	21.19 ± 3.96	22.71 ± 0.46	22.49 ± 2.16	19.98 ± 2.92	21.35 ± 0.99	19.76 ± 1.06	22.24 ± 2.88	28.76 ± 4.87[b]	20.49 ± 2.01	20.60 ± 1.70
18:3(n-3)	1.36 ± 0.34	1.29 ± 0.14	1.24 ± 0.14	1.13 ± 0.18	1.22 ± 0.15	1.16 ± 0.05	1.00 ± 0.10	0.90 ± 0.24[b]	1.03 ± 0.17	1.07 ± 0.09
20:4(n-6) + 22:1(n-11)	0.43 ± 0.11	0.42 ± 0.09	0.44 ± 0.11	0.40 ± 0.07	0.47 ± 0.13	0.46 ± 0.07	0.37 ± 0.12	0.48 ± 0.10	0.42 ± 0.09	0.44 ± 0.07
20:5(n-3)	0.27 ± 0.06	0.16 ± 0.05	0.20 ± 0.10	0.18 ± 0.03	0.19 ± 0.08	0.20 ± 0.05	0.09 ± 0.05	0.10 ± 0.05[b]	0.11 ± 0.05	0.11 ± 0.03
20:5(n-3)	0.37 ± 0.08	0.37 ± 0.10	0.41 ± 0.17	0.33 ± 0.09	0.42 ± 0.16	0.37 ± 0.07	0.23 ± 0.09	0.19 ± 0.12[b]	0.32 ± 0.16	0.31 ± 0.17
22:6(n-3)	0.62 ± 0.24	0.66 ± 0.15	0.79 ± 0.20	0.61 ± 0.16	0.74 ± 0.31	0.79 ± 0.19	0.38 ± 0.13	0.34 ± 0.21	0.55 ± 0.23	0.64 ± 0.17
Sum of trace components	4.67 ± 1.24	5.24 ± 0.96	5.62 ± 0.85	5.44 ± 1.06	5.11 ± 0.89	5.27 ± 1.29	4.58 ± 1.13	4.62 ± 0.96	4.81 ± 0.87	4.85 ± 0.92

[a]Fatty acids designated by chain length, number of double bonds and position of the first double bond from the methylene end of the molecule.
[b]Values significantly different from those for animals fed the stock diet or starved for 48 hr at $p < 0.05$.

TABLE 3

Fatty Acid Composition of Rat Epididymal Adipose Tissue (Wt % of Total Fatty Acids)

Fatty acid[a]	Ad-libitum fed stock diet (n = 5)	48 Hr starved 0 Hr (n = 4)	Fat-free, high carbohydrate diet		Corn oil diet (after 48 hr on FF diet	Saturated fat diet (after 48 hr on FF diet)
			48 Hr (n = 5)	96 Hr (n = 4)	48 Hr (n = 4)	48 Hr (n = 6)
14:0	1.74 ± 0.27	1.51 ± 0.14	1.82 ± 0.27	1.63 ± 0.12	1.38 ± 0.10[b]	1.62 ± 0.10
16:0	24.42 ± 1.92	23.04 ± 1.80	23.21 ± 1.33	23.64 ± 0.42	21.68 ± 0.78[b]	23.17 ± 0.87
16:1(n-7)	5.34 ± 1.82	4.53 ± 0.88	4.67 ± 1.77	4.95 ± 0.65	3.57 ± 0.61[b]	5.14 ± 0.96
18:0	4.73 ± 0.44	4.58 ± 0.51	5.01 ± 0.44	4.80 ± 0.39	5.01 ± 0.40	4.85 ± 0.55
18:1(n-9)	28.00 ± 0.57	27.96 ± 0.62	28.10 ± 1.39	28.88 ± 1.14	28.66 ± 0.64	28.96 ± 0.83
18:1(n-7)	3.88 ± 0.36	4.15 ± 0.16	4.08 ± 0.54	4.82 ± 0.25[b]	3.91 ± 0.31	4.58 ± 0.35
18:2(n-6)	21.45 ± 2.19	22.62 ± 1.20	22.58 ± 1.59	21.59 ± 1.14	26.50 ± 1.56[b]	22.50 ± 1.10
18:3(n-3)	1.41 ± 0.18	1.41 ± 0.10	1.45 ± 0.08	1.33 ± 0.08	1.16 ± 0.21[b]	1.32 ± 0.08
20:4(n-6) + 22:1(n-11)	0.77 ± 0.17	0.86 ± 0.11	0.74 ± 0.19	0.63 ± 0.09	0.75 ± 0.14	0.67 ± 0.10
20:5(n-3)	0.52 ± 0.12	0.51 ± 0.09	0.48 ± 0.21	0.36 ± 0.10	0.28 ± 0.10[b]	0.30 ± 0.08
22:5(n-3)	0.69 ± 0.18	0.87 ± 0.15	0.70 ± 0.24	0.54 ± 0.12	0.51 ± 0.13[b]	0.53 ± 0.11
22:6(n-3)	1.58 ± 0.41	1.94 ± 0.36	1.57 ± 0.56	1.26 ± 0.34	1.15 ± 0.34	1.25 ± 0.25
Sum of trace components	5.47 ± 1.31	6.02 ± 1.01	5.54 ± 1.21	5.57 ± 1.06	5.44 ± 1.02	5.06 ± 0.91

[a]Fatty acids designated by chain length, number of double bonds and the position of the first double bond from the methylene end of the molecule.
[b]Values significantly different from those for animals fed the stock diet or starved for 48 hr at $p < 0.05$.

It is possible that poor intestinal absorption of stearic acid is the reason that the hydrogenated fat diet had little influence on the fatty acid composition of the adipose tissue. However, all the animals in this study consumed approximately the same number of calories and gained about the same amount of weight. Additionally, 40% of the calories consumed in the hydrogenated fat diet were in the form of stearic acid, so that even a relatively poor absorption rate for this compound would still provide a significant amount of stearic acid for metabolic use as reported by Clarke et al. (9).

Most previous reports on the fatty acid composition of adipose tissue have not included data on the 20- and 22-carbon polyunsaturated fatty acids present in this tissue (30,34,55). In this work we observed significant amounts of 20:4(n-6), 22:5(n-3) and 22:6(n-3), and small, but measurable, amounts of 20:5(n-3) in both dorsal and epididymal fat. Indeed, the 22-carbon, n-3 polyunsaturated fatty acids constitute more of the adipose tissue fatty acids than arachidonic acid, an unusual circumstance in the rat, whose tissues usually contain relatively large amounts of arachidonic acid (35,56,57).

Adipose tissue is a major reservoir of fatty acids in the mature rat (22,57); indeed, the adipose tissue pool of fatty acids may be larger than all other carcass fatty acid pools combined (14,22,58). If all the individual long chain n-3 fatty acids in rat adipose tissue are summed, they represent between 3% and 4% of the total adipose fatty acids. This is in contrast to the arachidonic acid pool which, although much larger than the n-3 pool in rats, is concentrated in more physiologically active tissue, such as heart muscle (57,59), liver (35,37,60), nerve (61) and various others (53,57,61,62). The accumulation of n-3 fatty acids by adipose tissue may be an aging phenomenon as the 22-carbon, n-3 fatty acids are not precursors of physiologically active eicosanoids (63) and may accumulate in the adipose tissue with age.

There was no inhibition of the lipogenic enzymes, measured in white adipose tissue during the refeeding period of this study. When we fed corn oil to the animals, linoleic acid levels rose markedly in the adipose tissue within 48 hr, whereas feeding saturated fat had no influence on the fatty acid composition of the adipose tissue in the same time period. Feeding these two types of fat for longer periods could clarify whether these differences were due to slower absorption and/or transport of saturated fat or to different pathways for the metabolism of saturated versus polyunsaturated fatty acids in the rat.

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[Received November 10, 1986]