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# Tissue fatty acid deposition is influenced by an interaction of dietary oil source and energy intake level in rats

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*To investigate the net tissue fatty acid deposition in response to graded levels of energy restriction and modification of diet fatty acid composition, rats were randomly assigned into four dietary groups and fed for 10 weeks diets containing 40% as energy of either fish, safflower, or olive oil, or beef tallow, consumed ad libitum or energy restricted to 85% or 68% of ad libitum intake by reducing diet carbohydrate content. An additional eight rats were killed before the diet regimen, to provide baseline data from which fatty acid deposition rates were calculated. Body weight, and heart, liver and fat mass gains were decreased with energy restriction ($P < 0.001$). Olive oil feeding resulted in higher body weight gain ($P < 0.03$) than tallow feeding, whereas fish oil feeding was associated with highest ($P < 0.007$) liver weight and lowest ($P < 0.03$) fat mass gains. Energy deficit-related differences in the deposition of stearic, linoleic, arachidonic, and docosahexaenoic acids in heart and palmitic and docosahexaenoic acids in liver were dependent on the dietary oil consumed ($P < 0.03$). Similarly, interactive effects of restricted food intake and dietary oil type were found in the gain of palmitic, stearic, oleic, and linoleic acids in adipose tissue ($P < 0.01$) when expressed in relation to the amount of each fatty acid consumed. These data suggest that energy intake level can influence the deposition pattern, as well as oxidation rate, of tissue fatty acids as a function of tissue type, fatty acid structure, and dietary fatty acid composition.*  (J. Nutr. Biochem. 7:650–658, 1996.)

**Keywords:** fatty acid deposition; dietary fat; energy restriction; liver; heart; adipose; rat

## Introduction

Increasing evidence suggests that tissue fatty acid deposition is influenced by the feeding state of the animal. Food restriction in rats induced increases in percentage of linoleic and arachidonic acids in liver while palmitic, palmitoleic and oleic acids decreased markedly.[1–3] Linoleic acid-enriched triacylglycerol species were quantitatively increased in liver and serum lipids during fasting.[4] Carbohydrate restriction increased unsaturation indices of triacylglycerol fatty acids in liver by dramatically increasing linoleic acid content while decreasing oleic acid content.[5] These studies collectively suggest structure-specific alterations in oxidation rate of individual fatty acids subsequent to energy deficiency. The selective partitioning of body fatty acids between either accumulation or utilization for energy has also been shown to be dependent on dietary fatty acid composition. Humans consuming diets rich in polyunsaturated fatty acids (PUFA) and trans fatty acids tend to have more of these fatty acids deposited in their adipose tissue,[6,7] whereas animal studies have shown that dietary lipid profiles determine organ[8] and adipose tissue,[9,10] as well as membrane phospholipid[11,12] fatty acid composition. Modifications in membrane structural fatty acid composition induce changes in enzyme activity[13,14] and membrane function[12] and influence several metabolic processes, in-

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cluding glucose transport,[15] protein turnover,[16] insulin binding,[17] and glucose tolerance.[18]

These observations suggest that the selective deposition of body fatty acid depends on both the energy status of the animal and diet fatty acid composition individually. Recently, we reported an interactive heterogeneity of tissue fatty acid composition at 10 weeks in response to energy intake level and dietary oil source.[19] However, whether the longer term accumulation of tissue fatty acid was also influenced by the interaction remains unknown. The present study was designed to test the hypothesis that net deposition rates for major fatty acids in different tissues of animals are dependent on both energy intake level and dietary fatty acid composition.

## Methods and materials

### *Animal and diets*

Adult male Sprague Dawley rats (193 ± 9.1 g) were purchased from Biobreeding Laboratories (Ottawa, Ont.), housed individually in stainless steel hanging cages with a 12-hr light-dark cycle and given free access to Purina rat chow for the first 7 days after delivery. Animals were then randomly divided into four dietary oil groups of 24 rats each. Each group was assigned a nutritionally adequate diet containing fish, safflower, or olive oil or beef tallow where the fat comprised 40% of energy consumed. The beef tallow was supplemented with 1% safflower oil to maintain adequate intake of linoleic acid. An additional eight rats were killed before the dietary regimen to serve as a baseline for calculating deposition rates. Animals within each oil group were given either free access to the control diet ($n$ = 8), or food restricted by reducing calories supplies to 85% ($n$ = 8) or 68% ($n$ = 8) of ad libitum daily intakes. Daily determination of food intake of ad libitum-fed animals enabled precise control of amounts fed to each of the energy-restricted groups. The control diet contained 22.4% casein, 19% fat, and 42% carbohydrate with adequate amounts of minerals and vitamins. Diets consumed by the food-restricted animals were adjusted to supply equal quantities of all nutrients each day compared to the control diet, with the exception of carbohydrate.[19] Within each dietary oil group, the fatty acid contents in the food restriction diets were proportionally increased in order to maintain the fatty acid supply (*Table 1*). Food intakes were recorded daily. Body weights were monitored on the starting day of the feeding trial and before sacrifice. At the end of the 10-week feeding treatment, all animals received i.p. injection of 1 mL $^2H_2O$. Two hours later, animals were anaesthetized and blood samples collected by heart puncture. Animals were then killed, liver, heart and nape adipose tissue collected, weighed and immediately frozen in liquid $N_2$, then stored at −70°C until further analysis. The weight gains of liver and heart were calculated as weight differences at sacrifice between animals in each treatment group and those in the baseline group.

### *Lipid extraction and gas chromatography*

Liver, heart, and nape fat samples from each treatment group and from the baseline group animals were extracted using $CHCl_3$-MeOH (2:1 v/v) by the procedures described previously.[20] The extracts were trans esterified to form methyl esters as described by Al Makdessi et al.[21] Fatty acid methyl ester composition was analyzed using gas-liquid chromatography (model 5890, Hewlett Packard, Palo Alto, CA, USA) equipped with a 30 m × 0.2 mm SP 2330 column (Supelco, Bellefonte, PA, USA), flame ionization detectors, and automated injection. Fatty acid methyl esters were detected and identified against authentic standards. Total fatty acid and each individual fatty acid contents per organ basis were determined, and depositions were calculated using the following formula:

Fatty acid deposition = values derived from each animal in each treatment group − values derived from the average of baseline group. (1)

### *Body composition determination*

Serum samples were prepared for isotopic analysis using vacuum techniques and analyzed by isotope ratio mass spectrometer as previously described.[22] Total body fat was measured by body water volume determination using the deuterium dilution space.[23] Total body water was calculated as:

$$\text{Total body water} = (F_1{*}N_1 - \text{corr.})/F_2 - \text{new water} \quad (2)$$

where $F_1$ is the mole fraction of deuterium in the dose, $N_1$ is the deuterium dose, "corr." enables correcting the dose for the amount of isotope needed to bring the new water up to the predose isotopic abundance of body water, $F_2$ is the mole fraction of the isotope in serum, and "new water" is the water present in deute-

**Table 1** Major fatty acid composition of the experimental diets

| Fatty acid | Dietary oil source | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Fish Energy intake group | | | Safflower Energy intake group | | | Olive Energy intake group | | | Beef tallow Energy intake group | | |
| | 100% | 85% | 68% | 100% | 85% | 68% | 100% | 85% | 68% | 100% | 85% | 68% |
| | (g/100g diet) | | | | | | | | | | | |
| 16:0 | 3.2 | 3.8 | 4.8 | 0.5 | 0.6 | 0.7 | 2.3 | 2.8 | 3.4 | 4.8 | 5.7 | 7.1 |
| 18:0 | 0.9 | 1.1 | 1.3 | 0.4 | 0.5 | 0.6 | 0.7 | 0.8 | 1.0 | 0.4 | 0.5 | 0.6 |
| 18:1 (n − 9) | 2.3 | 2.7 | 3.4 | 2.8 | 3.3 | 4.1 | 13.4 | 15.8 | 19.7 | 7.3 | 8.6 | 10.7 |
| 18:2 (n − 6) | 0.9 | 1.1 | 1.3 | 14.9 | 17.5 | 21.9 | 2.2 | 2.6 | 3.2 | 0.3 | 0.4 | 0.5 |
| 20:4 (n − 6) | 0.4 | 0.5 | 0.6 | —[a] | — | — | — | — | — | — | — | — |
| 22:6 (n − 3) | 2.1 | 2.5 | 3.1 | — | — | — | — | — | — | — | — | — |

[a]Trace amount.

rium dose entering the body. Lean body mass was considered to consist of 27% protein and 73% water.[24] Thus,

$$\text{Lean body mass} = \text{total body water}/0.73 \quad (3)$$

$$\text{Total body fat} = \text{body weight} - \text{lean body mass} \quad (4)$$

Body fat mass gain was determined by subtracting baseline values from the calculated total body fat.

### *Statistical analysis*

Data were analyzed by a two-way analysis of variance using a SAS general linear model program (SAS Version 6. SAS Institute Inc. Cary, NC, USA). When the interaction of the two main effects for a parameter was not significant, the means were pooled across a main effect to test the group differences within another main effect. Means of variables were separated by Fisher's protected least significant difference procedure (Fisher's protected LSD).[25] Differences between means were considered to be significant at $P < 0.05$. The data are expressed as means ± SEM.

## Results

Food consumption was not significantly different among groups fed different dietary oil within each food intake level. Body weight, heart, liver, and fat mass weight gain data are shown in *Figure 1*. Cumulative body weight gain was proportionally decreased with progressive food restriction ($P < 0.001$). Similar findings were observed in heart, and fat mass gains ($P < 0.001$). Liver weight was decreased only in the 68% restricted group ($P < 0.001$). Fat mass was the most sensitive tissue in response to energy restriction as shown by 53% less weight gain in animals consuming 68% of ad libitum food intake as compared to the gain of control animals.

Dietary oil source had no effect on the increments of body and heart weight ($P > 0.15$). However, when data were pooled across the three food intake levels, higher body weight gains were found in olive oil-fed animals as compared to the beef tallow group ($P < 0.03$). In contrast, to

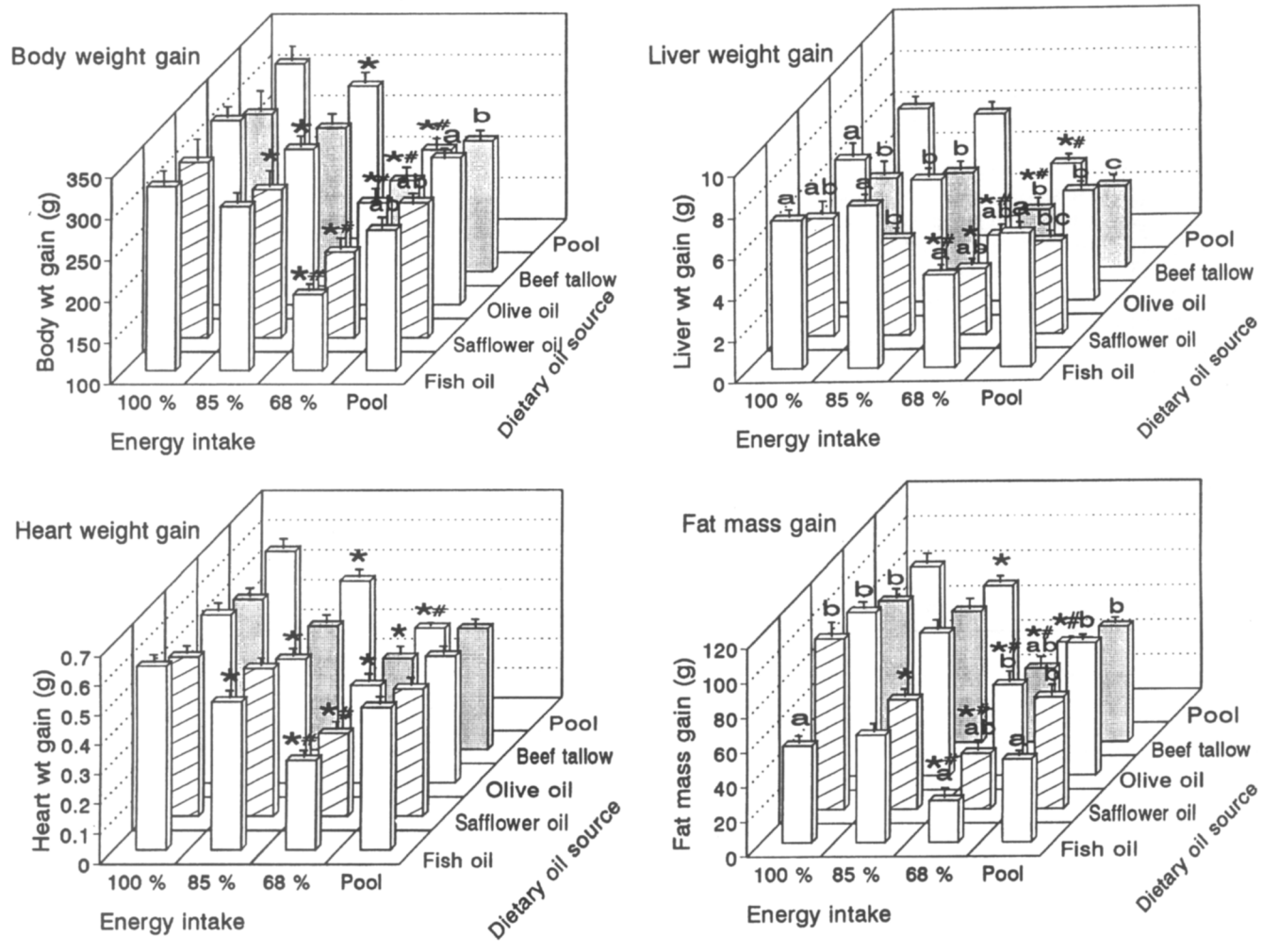


**Figure 1** Body, heart, liver weight, and fat mass gains of rats consuming diets varying in energy intake level and oil source for 10 weeks. Energy restriction influenced the accretions of body weight and all the tissue investigated ($P < 0.001$). Dietary oil source had no effect on body and heart weight gains ($P \geq 0.16$), while significantly influencing liver and adipose weight gains ($P < 0.001$). Values are means ± SEM ($n = 8$). Values within each column not associated with the same superscript letter are significantly different (Fisher's protected LSD, $P < 0.05$). Within each row, values with * superscript differ significantly with ad libitum (100%) fed group; values with # superscript differ with 85% of ad libitum fed group.

body weight and heart weight gains, dietary fatty acid composition significantly influenced liver and adipose tissue accretions ($P < 0.001$). Liver weight gain was highest in fish oil- and lowest in beef tallow-fed groups, whereas no difference was found between those fed safflower and olive oil. Fat mass gain was lower in fish oil-fed animals compared with other dietary groups ($P < 0.03$).

Total and major fatty acids depositions in hearts of animals consuming diets varying in energy level and oil source are shown in *Table 2*. Food restriction significantly reduced the total fatty acid increment per heart ($P < 0.004$). When data were pooled across the diet oil groups, fatty acid deposition in hearts of 68% food intake group was significantly lower than that of the ad libitum-fed ($P < 0.002$) and 85% intake groups ($P < 0.02$). No interactive effect of fat type and energy restriction was found ($P = 0.48$). Fish oil feeding was associated with a higher total fatty acid deposition as compared with tallow feeding ($P < 0.03$).

Food restriction decreased ($P < 0.04$) the deposition of palmitate in heart while not affecting the gain of oleate ($P = 0.10$). Feeding fish oil was associated with higher palmitic ($P < 0.001$) and lower oleic ($P < 0.05$) acid gains compared with other oil groups. Depositions of most major fatty acids (stearic, linoleic, arachidonic, and docosahexaenoic acid) in heart were affected by an interactive effect between dietary fat and energy restriction ($P < 0.05$). Deficit in caloric intake caused less stearic acid deposition in heart of animals fed fish, safflower oils and beef tallow while this was not found with olive oil feeding. Both animal oil feeding and food restriction decreased ($P < 0.05$) linoleic acid levels in fish and tallow group. In contrast, the amount of linoleic acid remained constant in the safflower oil group or increased in the olive oil group in a manner associated with energy restriction. Arachidonic acid deposition remained unchanged across the three energy intake levels in olive oil-fed animals while decreasing in animals consuming other dietary oils. A zero or negative gain of docosahexaenoic acid was observed across all oil groups independent of energy intakes, except for fish oil group in which the cumulative gain of this fatty acid was lower in 68% restricted group as compared with the control ($P < 0.05$) or 85% restricted group ($P < 0.02$).

Total and major fatty acids depositions in livers of rats fed diets varying in energy level and oil source are shown in *Table 3*. Both energy restriction ($P < 0.001$) and diet fatty acid composition ($P < 0.001$) independently influenced total fatty acid deposition in liver without interactive effect ($P = 0.32$). Food restriction to 68% of ad libitum intake decreased liver total fatty acid gain as compared with control animals ($P < 0.001$) and to those restricted to 85% ($P < 0.01$) across dietary oil groups. When data were pooled across varied food intakes, fish and olive oil feedings were

**Table 2** Depositions of total and major fatty acids in hearts of rats fed fish, safflower, olive oil or beef tallow at graded levels of energy intake[a]

| Fatty acid | Energy intake | Dietary oil consumed | | | | Pool |
|---|---|---|---|---|---|---|
| | | Fish | Safflower | Olive | Beef tallow | |
| | | | | (mg/heart) | | |
| Total | 100% | 37.0 ± 7.6 | 33.6 ± 4.0 | 24.7 ± 0.7 | 27.1 ± 2.4 | 30.9 ± 2.4 |
| | 65% | 31.3 ± 5.9 | 28.5 ± 3.8 | 24.7 ± 2.2 | 26.6 ± 3.9 | 27.8 ± 2.0 |
| | 68% | 22.6 ± 4.6$^{ab*}$ | 18.4 ± 3.2$^{ab*}$ | 25.7 ± 4.9$^{a}$ | 13.7 ± 1.8$^{b*\#}$ | 19.9 ± 2.0$^{*\#}$ |
| | Pool | 30.8 ± 3.7$^{a}$ | 26.5 ± 2.4$^{ab}$ | 25.0 ± 2.0$^{ab}$ | 22.5 ± 4.4$^{b}$ | |
| 16:0 | 100% | 6.3 ± 2.0$^{a}$ | 2.3 ± 0.2$^{b}$ | 2.9 ± 0.4$^{ab}$ | 2.5 ± 0.3$^{b}$ | 3.5 ± 0.6 |
| | 85% | 5.1 ± 1.1$^{a}$ | 1.8 ± 0.2$^{b}$ | 2.3 ± 0.3$^{b}$ | 2.5 ± 0.8$^{b}$ | 2.9 ± 0.4 |
| | 68% | 3.4 ± 1.3$^{a}$ | 0.6 ± 0.2$^{b*\#}$ | 1.7 ± 0.4$^{b*}$ | 0.4 ± 0.1$^{b*\#}$ | 1.4 ± 0.3$^{*\#}$ |
| | Pool | 5.0 ± 0.9$^{a}$ | 1.6 ± 0.1$^{b}$ | 2.2 ± 0.2$^{b}$ | 1.8 ± 0.4$^{b}$ | |
| 18:0[b] | 100% | 6.1 ± 0.6$^{a}$ | 6.2 ± 0.8$^{a}$ | 2.2 ± 2.1$^{b}$ | 4.6 ± 0.5$^{ab}$ | |
| | 85% | 5.0 ± 0.4$^{a}$ | 5.0 ± 0.4$^{a}$ | 3.3 ± 0.2$^{b}$ | 4.1 ± 0.4$^{ab}$ | |
| | 68% | 3.2 ± 0.4$^{a*\#}$ | 2.8 ± 0.4$^{a*\#}$ | 3.2 ± 0.3$^{a}$ | 2.3 ± 0.3$^{a*\#}$ | |
| 18:1 | 100% | 3.3 ± 1.6 | 5.3 ± 0.8 | 4.9 ± 1.7 | 6.0 ± 0.8 | 4.9 ± 0.6 |
| (n-9) | 85% | 2.7 ± 0.9$^{a}$ | 4.7 ± 1.5$^{ab}$ | 6.3 ± 1.5$^{ab}$ | 7.4 ± 2.0$^{b}$ | 5.2 ± 0.8 |
| | 68% | 1.6 ± 1.0$^{a}$ | 2.9 ± 1.1$^{ab}$ | 5.5 ± 1.6$^{b}$ | 3.2 ± 0.3$^{ab\#}$ | 3.4 ± 0.6$^{\#}$ |
| | Pool | 2.5 ± 0.7$^{a}$ | 4.3 ± 0.7$^{ab}$ | 5.6 ± 0.9$^{b}$ | 5.6 ± 0.8$^{b}$ | |
| 18:2 | 100% | −2.9 ± 0.4$^{a}$ | 4.9 ± 0.6$^{b}$ | −1.1 ± 1.3$^{a}$ | −0.9 ± 0.2$^{a}$ | |
| (n-6)[c] | 85% | −3.7 ± 0.3$^{a}$ | 5.8 ± 0.7$^{b}$ | 0.8 ± 0.2$^{c}$ | −1.5 ± 0.2$^{d}$ | |
| | 68% | −4.6 ± 0.2$^{a*\#}$ | 3.4 ± 0.5$^{b\#}$ | 1.2 ± 0.3$^{c*}$ | −2.0 ± 0.2$^{d*}$ | |
| 20:4 | 100% | 3.8 ± 0.6$^{a}$ | 9.0 ± 1.1$^{b}$ | 3.7 ± 2.6$^{a}$ | 7.0 ± 0.7$^{ab}$ | |
| (n-6)[d] | 85% | 3.4 ± 0.3$^{a}$ | 7.4 ± 0.5$^{b}$ | 6.3 ± 0.5$^{b}$ | 6.5 ± 0.5$^{b}$ | |
| | 68% | 1.0 ± 1.2$^{a*\#}$ | 5.7 ± 0.5$^{b*}$ | 6.4 ± 0.3$^{b}$ | 4.9 ± 0.5$^{b*}$ | |
| 22:6 | 100% | 7.9 ± 0.5$^{a}$ | −3.4 ± 0.0$^{b}$ | −1.6 ± 0.6$^{c}$ | 0.1 ± 0.7$^{d}$ | |
| (n-3)[e] | 85% | 8.4 ± 0.6$^{a}$ | −3.4 ± 0.0$^{b}$ | −0.8 ± 0.5$^{c}$ | −0.7 ± 0.7$^{c}$ | |
| | 68% | 4.6 ± 1.7$^{a*\#}$ | −3.2 ± 0.2$^{b}$ | −0.7 ± 0.5$^{b}$ | −0.9 ± 0.7$^{b}$ | |

[a]Values are means ± SEM ($n$ = 6–8). Means with different letter superscript in the same row are significantly different ($P < 0.05$, Fisher's protected LSD). For total or each fatty acid in the same column, means with * superscript significantly different with 100% fed group; means with # superscript significantly different with 85% fed group.
[b]ANOVA: energy * fat $P < 0.05$.
[c]ANOVA: energy * fat $P < 0.002$.
[d]ANOVA: energy * fat $P < 0.05$.
[e]ANOVA: energy * fat $P < 0.04$.

**Table 3** Depositions of total and major fatty acids in livers of rats fed fish, safflower, olive oil or beef tallow at graded levels of energy intake[a]

| Fatty acid | Energy intake | Dietary oil consumed | | | | Pool |
|---|---|---|---|---|---|---|
| | | Fish | Safflower | Olive | Beef tallow | |
| | | | | (mg/liver) | | |
| Total | 100% | 1360.2 ± 156.4[a] | 994.0 ± 135.0[a] | 1388.6 ± 108.8[a] | 503.0 ± 76.1[b] | 1039.1 ± 93.6 |
| | 85% | 1343.2 ± 157.7[a] | 769.6 ± 247.8[b] | 1363.8 ± 190.2[a] | 585.6 ± 56.1[b] | 1028.6 ± 108.1 |
| | 68% | 768.0 ± 182.9*# | 645.8 ± 127.3 | 709.4 ± 113.2*# | 408.2 ± 94.0 | 642.8 ± 69.2*# |
| | Pool | 1167.0 ± 110.5[a] | 794.8 ± 100.7[b] | 1153.9 ± 54.3[a] | 504.2 ± 44.3[c] | |
| 16:0[b] | 100% | 382.8 ± 60.6[a] | 219.6 ± 30.8[bc] | 314.0 ± 29.0[ab] | 121.0 ± 21.2[c] | |
| | 85% | 328.6 ± 44.4[a] | 117.2 ± 41.0[b] | 348.8 ± 70.0[a] | 110.2 ± 18.0[b] | |
| | 68% | 158.4 ± 42.4*# | 70.6 ± 19.8* | 153.2 ± 35.4*# | 144.0 ± 61.0 | |
| 18:0 | 100% | 75.6 ± 9.2[ab] | 89.8 ± 18.2[ab] | 112.4 ± 18.8[a] | 48.0 ± 13.8[b] | 81.9 ± 9.0 |
| | 85% | 91.6 ± 10.8 | 58.4 ± 20.4 | 89.4 ± 14.4 | 71.8 ± 7.4 | 79.6 ± 6.8 |
| | 68% | 59.0 ± 13.8 | 52.6 ± 5.0 | 40.6 ± 2.6*# | 41.0 ± 4.2 | 49.1 ± 4.1*# |
| | Pool | 76.2 ± 7.2[ab] | 67.8 ± 9.2[ab] | 85.1 ± 10.7[a] | 53.9 ± 6.5[b] | |
| 18:1 | 100% | 286.6 ± 66.0[a] | 334.4 ± 56.0[ab] | 504.2 ± 87.0[b] | 208.4 ± 28.8[a] | 329.9 ± 37.4 |
| (n-9) | 85% | 248.6 ± 41.2[a] | 255.6 ± 96.8[a] | 767.0 ± 166.6[b] | 237.6 ± 41.0[a] | 381.8 ± 65.3 |
| | 68% | 112.2 ± 33.2*# | 134.4 ± 36.2[a] | 397.8 ± 74.6[b] | 223.2 ± 66.0[a] | 207.3 ± 34.3*# |
| | Pool | 208.3 ± 30.2[a] | 242.3 ± 44.3[a] | 576.8 ± 79.8[b] | 223.1 ± 23.9[a] | |
| 18:2 | 100% | 40.8 ± 27.8[a] | 189.2 ± 50.8[b] | 170.4 ± 38.8[b] | -33.4 ± 1.2[a] | 84.3 ± 23.7 |
| (n-6) | 85% | -5.74 ± 6.6[a] | 217.0 ± 53.2[b] | 96.6 ± 23.6[c] | -32.0 ± 2.0[a] | 54.3 ± 21.1 |
| | 68% | -28.6 ± 6.6[a]* | 191.8 ± 47.4[b] | 74.0 ± 23.4[c]* | -37.6 ± 2.2[a] | 56.3 ± 23.1* |
| | Pool | 3.66 ± 11.68[a] | 198.5 ± 27.5[b] | 113.7 ± 18.6[c] | -34.1 ± 1.1[a] | |
| 20:4 | 100% | 85.6 ± 16.2[a] | 184.2 ± 25.6[b] | 210.6 ± 26.0[b] | 71.4 ± 13.4[a] | 137.9 ± 15.3 |
| (n-6) | 85% | 75.4 ± 17.0[ac] | 125.4 ± 29.4[ab] | 175.6 ± 19.0[b] | 61.6 ± 8.2[c] | 110.2 ± 12.9* |
| | 68% | 35.2 ± 9.2[ad]* | 133.0 ± 9.0[b] | 107.0 ± 6.2[c]*# | 24.6 ± 4.0[d]*# | 73.8 ± 10.0*# |
| | Pool | 63.4 ± 9.1[a] | 149.5 ± 14.2[b] | 167.2 ± 14.5[b] | 55.5 ± 7.2[a] | |
| 22:6 | 100% | 274.4 ± 54.2[a] | -28.8 ± 4.4[b] | -5.12 ± 9.96[b] | 16.7 ± 6.4[b] | |
| (n-3)[c] | 85% | 211.4 ± 27.4[a] | -37.2 ± 3.4[b] | 3.14 ± 9.82[b] | 0.76 ± 7.70[b] | |
| | 68% | 127.2 ± 26.0[a]* | -36.4 ± 2.6[b] | -23.4 ± 6.1[b] | -17.4 ± 6.2[b] | |

[a]Values are means ± SEM ($n$ = 6–8). Means with different letter superscript in the same row are significantly different ($P < 0.05$, Fisher's protected LSD). For total or each fatty acid in the same column, means with * superscript significantly different with 100% fed group; means with # superscript significantly different with 85% fed group.
[b]ANOVA: energy * fat $P < 0.04$.
[c]ANOVA: energy * fat $P < 0.02$.

associated with the highest liver fatty acid content, whereas beef tallow feeding were associated with the lowest.

Net retention of palmitic acid in liver in response to energy restriction was specific to the type of oil fed ($P < 0.04$). Specifically, food restriction to 68% of ad libitum intake reduced palmitate deposition in fish, safflower, and olive oil groups, whereas a deficit in food intake had no effect in the tallow-fed group. A similar interactive effect of caloric restriction and diet fat type was found on the deposition of docosahexaenoic acid ($P < 0.02$). The net gain of this fatty acid in liver was decreased with fish oil feeding in response to energy restriction ($P < 0.001$), whereas the deficit in energy intake showed no effect on the accumulation of docosahexaenoic acid in other oil groups.

Energy restriction decreased the deposition of stearic and oleic acid ($P < 0.02$) in liver, as shown by reduced content of these fatty acids in liver in the 68% restricted group as compared with the control group ($P < 0.05$). Arachidonic acid accumulation was progressively reduced in response to increasing energy restriction ($P < 0.03$). The gain of linoleic acid in the safflower oil- and beef tallow-fed groups was not influenced by energy restriction. However, when data were pooled across dietary oil groups, a significant lowered deposition of this fatty acid was found in 68% food restriction group as compared with the control group ($P < 0.05$).

Dietary oil source was found to influence liver oleic and linoleic acid deposition ($P < 0.0002$) in a manner that reflected the fatty acid composition of the oil. Net accumulation of oleic acid was higher ($P < 0.001$) in the olive oil-fed group compared with other groups, whereas the increment of linoleic acid was highest with safflower oil feeding. However, this phenomenon was not found for the depositions of stearic and arachidonic acid. Stearic acid gain was higher in the olive oil-fed group compared with the group consuming beef tallow ($P < 0.02$). Arachidonic acid accumulation was lower in fish oil and tallow feeding ($P < 0.01$) as compared with safflower and olive oil feeding.

Total and major fatty acids depositions in adipose tissue of rats fed diets varying in energy level and fatty acid composition are shown in *Table 4*. Energy restriction influenced fatty acid accumulation in adipose tissue ($P < 0.0002$) as shown by less fatty acid stored in adipose tissue of animals where food intake was restricted to 68% of ad libitum intake compared with animals restricted to 85% ($P < 0.0002$) or animals given free access to food ($P < 0.0002$). There were no differences in fatty acid deposition between the 85% restriction group and controls ($P = 0.12$). Dietary fat type had no influence on total fatty acid gains in adipose tissue ($P = 0.13$), nor were there interactive effects between energy level and oil source ($P = 0.77$).

Food restriction significantly influenced net accumulation of palmitic, stearic, oleic and linoleic acids in adipose

**Table 4** Depositions of total and major fatty acids in adipose tissue of rats fed fish, safflower, olive oil or beef tallow at graded levels of energy intake[a]

| Fatty acid | Energy intake | Dietary oil consumed | | | | Pool |
|---|---|---|---|---|---|---|
| | | Fish | Safflower | Olive | Beef tallow | |
| | | | | (g/fat mass) | | |
| Total | 100% | 63.6 ± 10.0 | 71.5 ± 13.5 | 72.8 ± 8.2 | 73.9 ± 13.5 | 70.4 ± 5.5 |
| | 85% | 58.6 ± 5.9 | 43.8 ± 6.7* | 73.7 ± 7.6 | 65.9 ± 5.9 | 60.5 ± 3.8 |
| | 68% | 20.8 ± 3.7*# | 26.1 ± 8.2* | 38.7 ± 8.0* | 31.2 ± 5.4*# | 29.5 ± 3.3*# |
| | Pool | 47.1 ± 5.9 | 49.7 ± 7.4 | 61.1 ± 5.8 | 56.5 ± 6.7 | |
| 16:0 | 100% | 17.9 ± 3.1[a] | 21.3 ± 4.7[ab] | 25.5 ± 2.8[b] | 16.7 ± 3.2[a] | 20.3 ± 1.7 |
| | 85% | 16.2 ± 1.4[a] | 9.96 ± 1.65[a]* | 24.3 ± 2.4[b] | 13.9 ± 1.4[a] | 16.1 ± 1.3* |
| | 68% | 5.83 ± 1.11[ab]*# | 3.19 ± 1.82[a]* | 11.2 ± 2.4[b]*# | 4.82 ± 1.18[ab]*# | 6.48 ± 1.00*# |
| | Pool | 13.3 ± 1.7[a] | 12.0 ± 2.5[a] | 20.1 ± 2.1[b] | 11.7 ± 1.7[a] | |
| 18:0 | 100% | 2.19 ± 0.48[a] | 3.36 ± 0.69[ab] | 2.93 ± 0.45[ab] | 3.72 ± 0.56[b] | 3.05 ± 0.28 |
| | 85% | 2.18 ± 0.29[a] | 2.04 ± 0.40[a] | 3.21 ± 0.40[ab] | 3.78 ± 0.41[b] | 2.76 ± 0.23 |
| | 68% | 0.87 ± 0.09[a]* | 1.68 ± 0.65[ab]* | 2.23 ± 0.49[b] | 2.82 ± 0.37[b] | 1.96 ± 0.25*# |
| | Pool | 1.79 ± 0.23[a] | 2.45 ± 0.38[ab] | 2.74 ± 0.27[bc] | 3.42 ± 0.27[c] | |
| 18:1 | 100% | 15.3 ± 4.6[a] | 35.0 ± 5.9[bc] | 28.3 ± 3.2[b] | 44.4 ± 7.3[c] | 30.8 ± 3.3 |
| (n-9) | 85% | 9.36 ± 1.17[a] | 21.6 ± 3.0[b]* | 30.3 ± 3.1[bc] | 41.3 ± 3.3[c] | 25.0 ± 2.7 |
| | 68% | 2.82 ± 0.59[a]* | 13.3 ± 4.0[ab]* | 18.1 ± 3.3[b]# | 22.8 ± 3.2[b]*# | 14.7 ± 2.1*# |
| | Pool | 9.46 ± 1.96[a] | 23.9 ± 3.3[b] | 25.3 ± 2.2[b] | 35.9 ± 3.6[c] | |
| 18:2 | 100% | 1.54 ± 0.38[a] | 8.84 ± 0.91[b] | 5.34 ± 0.70[c] | −0.01 ± 0.27[a] | 4.15 ± 0.76 |
| (n-6) | 85% | 0.87 ± 0.30[a] | 7.10 ± 2.04[b] | 5.69 ± 0.68[c] | −0.13 ± 0.19[a] | 3.38 ± 0.82 |
| | 68% | −0.84 ± 0.16[a]* | 4.62 ± 1.25[b]*# | 2.91 ± 0.82[b]*# | −1.31 ± 0.20[a] | 1.32 ± 0.63*# |
| | Pool | 0.46 ± 0.29[a] | 6.86 ± 0.91[b] | 4.59 ± 0.50[c] | −0.51 ± 0.19[a] | |
| 20:4 | 100% | 0.63 ± 0.09[a] | 0.62 ± 0.09[a] | 0.41 ± 0.06[b] | −0.06 ± 0.03[c] | |
| (n-6)[b] | 85% | 0.53 ± 0.05[a] | 0.35 ± 0.09[b]* | 0.32 ± 0.05[b] | −0.09 ± 0.02[c] | |
| | 68% | 0.16 ± 0.03[a]*# | 0.19 ± 0.06[a]* | 0.11 ± 0.04[a]*# | −0.10 ± 0.01[b] | |
| 22:6 | 100% | 5.05 ± 0.63[a] | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | |
| (n-3)[c] | 85% | 4.17 ± 0.37[a]* | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | |
| | 68% | 1.99 ± 0.21[a]*# | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | −0.03 ± 0.00[b] | |

[a]Values are means ± SEM ($n$ = 6–8). Means with different letter superscript in the same row are significantly different ($P < 0.05$, Fisher's protected LSD). For total or each fatty acid in the same column, means with * superscript significantly different with 100% fed group; means with # superscript significantly different with 85% fed group.
[b]ANOVA: energy * fat $P < 0.004$.
[c]ANOVA: energy * fat $P < 0.001$.

tissue ($P < 0.003$), independent of oil fed as shown by decreased deposition of these acids in adipose tissue of 68% food restricted animals as compared with ad libitum-fed and 85% of ad libitum-fed animals ($P < 0.03$). Palmitic acid levels were also lower in the group restricted to 85% than in control animal given free access to food ($P < 0.02$). Adipose tissue content of stearic and oleic acids was highest in beef tallow group ($P < 0.05$), and linoleic acid level was greatest in the safflower oil group ($P < 0.01$), resembling the fatty acid composition in the diet. Similarly, greater gain of docosahexaenoic acid was found in fish oil feeding ($P < 0.05$). However, olive oil feeding associated with lower oleic acid ($P < 0.003$) and higher palmitic acid ($P < 0.0002$) accumulation compared with tallow feeding, inconsistent with fatty acid composition in the diet.

Arachidonic acid and docosahexaenoic acid depositions in adipose tissue were influenced by an interactive effect between whole body energy status and dietary fat type ($P < 0.004$). Energy restriction reduced the arachidonic acid content in animals consuming 68% of ad libitum intake compared with the control animals in fish and olive oil groups ($P < 0.01$), whereas accumulation of this fatty acid was found lower both in 68% and 85% energy restriction groups ($P < 0.05$), as compared with controls fed safflower oil. Food restriction had no effect on the negative gain of arachidonic acid associated with beef tallow-fed animals. The influence of dietary oil source on the deposition of this fatty acid was also found to be dependent on the energy status of the animals. Gains of arachidonic acid were higher in the safflower oil-fed group compared with the animals fed olive oil ad libitum ($P < 0.02$); however, when food intake was restricted to 68% and 85% of ad libitum intakes, no difference was found among dietary groups ($P > 0.37$). The influence of energy restriction on deposition of docosahexaenoic acid was only observed in fish oil feeding, whereas the negative gain of this fatty acid seen with the other oil-fed groups was not affected by energy deficit.

Accumulations of palmitic, stearic, oleic and linoleic acid in adipose tissue were also examined expressed as per gram of individual fatty acid consumed (*Table 5*). In contrast with the net accretions, the depositions relative to consumption of all the four fatty acids investigated were influenced by the interaction between energy restriction and dietary oil type ($P < 0.01$). Palmitic acid accumulation was decreased in response to energy restriction in safflower oil-fed animals ($P < 0.01$) while it remained constant in fish, olive oil- and beef tallow-fed animals. Energy restriction to 68% reduced deposition of linoleic acid in fish, olive oil, and tallow feedings ($P < 0.03$), whereas not affecting the gain of this acid in animals fed safflower oil diet ($P = 0.59$). Depositions of arachidonic and docosahexaenoic acid in adipose in relation to the intakes were not investigated

**Table 5** Depositions of major fatty acids expressed relevent to amount of consumption in adipose tissue of rats fed fish, safflower, olive oil or beef tallow at graded levels of energy intake[a]

| Fatty acid | Energy intake | Dietary oil consumed | | | |
|---|---|---|---|---|---|
| | | Fish | Safflower | Olive | Beef tallow |
| | | | (mg/fat mass/g consumed) | | |
| 16:0[b] | 100% | 328.2 ± 57.4$^{ac}$ | 2055.9 ± 431.6$^{b}$ | 613.1 ± 63.8$^{c}$ | 187.4 ± 31.9$^{a}$ |
| | 85% | 326.8 ± 28.8$^{ac}$ | 1171.7 ± 194.7$^{b*}$ | 607.2 ± 60.7$^{c}$ | 167.6 ± 16.9$^{a}$ |
| | 68% | 102.8 ± 19.5 | 167.9 ± 183.2$^{*\#}$ | 267.9 ± 58.5 | 55.7 ± 13.6 |
| 18:0[c] | 100% | 152.4 ± 33.6$^{a}$ | 445.1 ± 80.1$^{b}$ | 240.6 ± 36.4$^{a}$ | 47.5 ± 6.9$^{c}$ |
| | 85% | 166.0 ± 21.8$^{a}$ | 294.4 ± 58.4$^{b*}$ | 286.0 ± 30.2$^{b}$ | 51.2 ± 5.6$^{c}$ |
| | 68% | 42.3 ± 12.3$^{a*\#}$ | 112.3 ± 41.5$^{ab*\#}$ | 182.6 ± 40.2$^{b}$ | 36.7 ± 4.8$^{a}$ |
| 18:1 | 100% | 217.8 ± 47.5$^{ac}$ | 692.4 ± 100.5$^{b}$ | 119.3 ± 12.8$^{a}$ | 333.0 ± 48.6$^{c}$ |
| (n-9)[d] | 85% | 243.9 ± 31.6$^{ac}$ | 463.8 ± 64.4$^{b*}$ | 132.7 ± 13.3$^{c}$ | 330.9 ± 26.6$^{ab}$ |
| | 68% | 54.4 ± 18.2$^{a*\#}$ | 266.8 ± 80.8$^{b*\#}$ | 76.0 ± 14.0$^{a}$ | 175.0 ± 24.4$^{ab*\#}$ |
| 18:2 | 100% | 104.6 ± 25.5$^{a}$ | 31.7 ± 2.8$^{b}$ | 138.5 ± 17.3$^{a}$ | −5.19 ± 53.9$^{b}$ |
| (n-6)[e] | 85% | 65.3 ± 22.6$^{a}$ | 28.8 ± 8.3$^{a}$ | 153.2 ± 18.3$^{b}$ | −56.0 ± 40.2$^{c}$ |
| | 68% | −54.8 ± 10.5$^{a*\#}$ | 14.6 ± 4.9$^{b}$ | 75.3 ± 21.2$^{b\#}$ | −276.4 ± 35.8$^{c*\#}$ |

[a]Values are means ± SEM ($n$ = 5–8). Means with different letter superscript in the same row are significantly different ($P < 0.05$, Fisher's protected LSD). For each fatty acid in the same column, means with * superscript significantly different with 100% fed group; means with # superscript significantly different with 85% fed group.
[b]ANOVA: energy * fat $P < 0.0002$.
[c]ANOVA: energy * fat $P < 0.01$.
[d]ANOVA: energy * fat $P < 0.01$.
[e]ANOVA: energy * fat $P < 0.0002$.

because these two fatty acids were only detected in fish oil and were absent from other oil diets.

## Discussion

The present study was conducted to determine whether tissue fatty acid deposition rates in response to food restriction vary according to dietary fatty acid composition and tissue type. Specific and interactive effects of both energy restriction and dietary fat type on net tissue fatty acid retention were presently observed. Linoleic acid accumulation was decreased in adipose tissue in response to energy deficit while remaining constant in livers of animals fed safflower oil and increasing in hearts with olive oil feeding.

Energy restriction has been found to influence the pattern of tissue fatty acid retention previously. Chen and Cunnane[4] fasted rats for 24 or 48 hr and then examined liver fatty acids and serum triacylglycerols. Arachidonic, stearic, linoleic, α-linolenic and docosahexaenoic acid were quantitatively increased by food deprivation, whereas oleic, palmitic, and palmitoleic acid decreased, suggesting remodelling of tissue triacylglycerol fatty acid by fasting. Similar results were found by Rojas et al.[5] in energy-restricted mice. In the present study, under conditions of constant fat intake and positive weight gain, energy restriction to 68% affected accumulation of most tissue fatty acids investigated in a manner that exhibited organ and structure specificity.

Decreased deposition of individual fatty acids occurred more frequently with energy restriction in adipose tissue compared with liver and heart. Accretions of saturated fatty acid and PUFA were equally influenced by energy restriction; no differences among tissues were found. Conversely, accumulation of monounsaturated fatty acid in response to food restriction was more tissue-specific. The gain of oleic acid in response to energy deficiency decreased in adipose tissue while not affected in heart, independent of oil fed. Recently, Chen et al[26] reported that after repeated fasting and refeeding, the (n-3) and (n-6) PUFA accumulation in rat adipose tissue declined whereas that of palmitate, palmitoleate, and oleate increased. This higher retention of palmitic and oleic acids was inconsistent with our findings. The possibility that fasting and refeeding cycles stimulate tissue fatty acid synthesis, whereas constant food restriction inhibits it, may explain this discrepancy.

Tissue fatty acid deposition has been shown to be dependent on dietary fatty acid composition. Jandacek et al.[9] fed growing rats diets containing either medium-chain triacylglyceride, or corn, or menhaden oil for 6 weeks. Only small amounts of eicosapentaenoic and docosahexaenoic acids in fish oil-fed, and decanoic and octanoic acids in medium chain oil-fed animals were deposited into adipose tissue, indicating extensive oxidation of these acids associated with consuming these two specific dietary oils. Lin et al. showed similar findings in rabbits.[10] Our data showed that among the four tissue long-chain unsaturated fatty acids investigated, the depositions of linoleic and docosahexaenoic acids were positive correlated to amounts consumed in the diet. Only the fish oil group consumed (n-3) long chain fatty acids and the net depositions of tissue fatty acids were calculated using the tissue fatty acid composition of rat chow fed animal as the baseline. This could explain the negative gains of docosahexaenoic acid in most of the oil feedings observed, with the exception of fish oil group.

There was no quantitative relationship between tissue deposition of oleic and arachidonic acids and the amount consumed. Although there was almost double the amount of oleic acid in olive oil compared with beef tallow, gain of oleic acid in heart tissue was not significantly different between animals fed these two dietary fats. Moreover, the deposition of oleic acid was greater with tallow feeding in

adipose tissue. Lin et al.[10] reported seven times more deposition of oleic acid in rabbit adipose tissue than the amount consumed, in accordance with our findings. This accumulation could be due to the fact that stearic acid can be desaturated to oleic acid, which is deposited in adipose tissue or transported via the circulation to the heart for metabolism.

Only trace amounts of arachidonic acid were present in the four diet oils employed in the present study. However, positive deposition of arachidonic acid was observed across the dietary oil groups as well as tissues investigated, the only exception being adipose tissue with tallow-feeding, suggesting selective synthesis and storage of arachidonic acid. Garg et al.[27] reported that the desaturation-chain elongation of linoleic acid to form arachidonic acid in the small intestine was sensitive to dietary linoleic acid content and energy intake in rats, suggesting the biosynthesis rate of arachidonic acid depends on the substrate fatty acid supply and tissue need. This may explain the discrepancy between dietary supply and tissue deposition.

The results from the present study further demonstrate an interactive influence between energy intake level and dietary fatty acid composition on tissue fatty acid deposition. This interactive effect on tissue fatty acid accretion was also dependent on tissue type and fatty acid structure. For example, food restriction decreased stearic acid deposition in liver only with olive oil feeding, whereas in adipose tissue, reduced retention of this acid was found in fish and safflower oil-fed, but not in olive oil- and tallow-fed animals associated with food restriction. In contrast, with the present findings, we previously reported an increase in the proportion of stearic acid present in liver and adipose tissue in the face of energy deficit independent of oil fed.[19] In considering 50% decreased liver and adipose weight gain and 40% lowered total fatty acid in these two tissues of food restricted animals, the findings between net deposition of stearic acid on a per organ basis and distribution in percentage of total fatty acid could not be the same.

Linoleic acid cannot be synthesized de novo in the absence of 16:2(n-6); therefore, its presence in animal tissue is exclusively from diet. Linoleic acid deposition in liver was proportionally decreased in response to graded levels of energy restriction in fish, olive oil- and tallow-fed animals. Because the absolute intake of linoleic acid within each dietary oil group was equal (*Table 1*), the present findings suggest that more linoleic acid was oxidized or structurally changed to other fatty acids in the face of energy deficit, which resulted in less storage. However, the deposition of linoleic acid in liver with safflower oil feeding remained unchanged across energy intake groups. Similar results were found in heart tissue. Indeed, the gain of linoleic acid in heart was unchanged in response to food restriction with safflower oil, and even increased with olive oil feeding. In contrast, linoleic acid deposition in adipose tissue was decreased in all oil groups with energy restriction, suggesting mobilization of linoleic acid from this major fatty acid storage organ to supply the needs of other tissues. Our previous findings show the percentage of linoleic acid was increased both in liver and adipose of safflower oil-fed animals in facing energy deficiency.[19] Obviously, this proportional increase does not mean that more linoleic acid was deposited in these tissues of energy-restricted animals as compared with the controls. Chen and Cunnane[3] found that rats fed 25% of their ad libitum intake of semi-purified diet with sunflower oil over 4 days, linoleic acid-enriched triglycerides accumulated in liver. The present results demonstrate that this selective retention of linoleic acid during food restriction is dependent on dietary fatty acid composition and exhibits tissue specificity. It means that linoleic acid is preferentially stored in the critical organs such as liver and heart in the face of energy deficit when dietary supply of this fatty acid is sufficient.

Adipose tissue is the major storage organ for body fatty acids. Interestingly, when individual fatty acid accumulation in adipose was expressed as a function of the amount of that fatty acid consumed, a reverse trend between the amount of intake and deposition was observed. For example, consumption of beef tallow higher in palmitic and stearic acid resulted in lower depositions of these two fatty acids compared with other dietary oil groups. Similarly, the accumulation of linoleic acid was lower in safflower oil-fed animals although the intakes were higher compared with other oil feedings. Plentiful supply of an individual fatty acid from the diet may initiate the body to use it as a priority, thus resulting in less storage. Conversely, less intake of a fatty acid may cause preferential retention or synthesis of that fatty acid. In contrast to the fatty acid net accumulation in adipose, when the amount of individual fatty acid intake was equalized across the dietary oil groups, a highly significant interaction between energy restriction and dietary oil source on adipose tissue fatty acid accumulation was observed for all the four fatty acids investigated. This suggests that the qualitative aspect of different fatty acid intake might be a confounding factor when investigating the interactive effect involving dietary fatty acid composition and energy intake.

In the present report, fatty acid accretion of nape fat was used to represent the fatty acid deposition in adipose tissue. A possible concern could be the homogeneity of fatty acid across various regions of body fat. A site-specific difference in fatty acid composition was noted in human subcutaneous adipose tissue.[28,29] Other investigators reported little differences in fatty acid composition over most human adipose depots, with the exception of certain sites.[30] Recently, it was shown that the selective mobilization of tissue fatty acid was similar across retroperitoneal, epididymal, mesenteric, and subcutaneous adipose tissue in rats.[31] Quantification of overall adipose tissue fatty acid composition by sampling a unique site in the present study simplified our analytical procedures and provided us an example of different responses of adipose fatty acid accretion to the different energy intake level and dietary oil type.

In summary, the present study has demonstrated that longer term tissue fatty acid accumulation was influenced by an interaction between energy intake level and dietary fatty acid composition. The pattern of fatty acid deposition in tissues was specific to tissue type, fatty acid structure, and the amount of deposition was not proportional to its intake. Linoleic acid retention in adipose tissue was decreased in the face of energy deficiency, whereas heterogeneous responses were observed in liver and heart varying according to tissue and diet oil type. These results under-

score the importance of considering energy balance when establishing the relative partitioning of dietary fatty acids for utilization versus storage.

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