The Metabolic Fate of Fatty Acids Derived from Dietary Triglycerides

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Abstract

The metabolic fates of dietary tricaprylin, trimyristin, tripalmitin, triolein, and trilinolein at the 15% level were followed with tracer doses of the corresponding C^{14}-labeled acids. Distribution of the label in respiratory $C^{14}O_2$ and in fatty acids of adipose tissue and liver lipids as well as the fatty acid composition of these unfractionated tissue lipids led to the following conclusions:

Tissue fatty acid compositional homeostasis is limited mainly by the degrees to which dietary fatty acids can be converted to endogenous fatty acids. Other factors, such as their effects on lipogenesis and the relative degrees to which they are catabolized and stored, also play roles.

Introduction

The addition of fat to the diet of most animals causes the body fat to take on a fatty acid composition similar to that found in the diet (1). This effect can be attributed to the deposition of fatty acids derived from the dietary fat. However, as was pointed out by Di Giorgio et al. (2), the fatty acid composition of adipose tissue never actually duplicates that of the dietary fat.

Tove and Smith (3) found that, even though they were able to raise the level of linoleic acid in the depot fat of mice from 5 to 50%, the total level of unsaturated acids remained essentially the same. In a more recent study Tove and Smith (4) were unable to increase the oleic acid level of the depot fat above 67% regardless of the amount fed and therefore proposed that the deposition of fatty acids was enzymatically controlled by selective esterification of the triglyceride molecule. Recently we have found about 80% oleic acid in the epididymal fat of adult rats fed triolein for three months (5).

Other factors also may directly or indirectly exert some influence on the fatty acid composition of depot fat after feeding a high fat diet. These are the amount and rate of ingested fatty acid oxidation to CO_2 and the amount of fatty acid synthesis either *de novo* or by interconversions.

Short-chain fatty acids, such as butyric and caprylic, are oxidized to CO_2 at a much faster rate and higher degree than longer-chain fatty acids such as lauric and palmitic (6), or, since these short-chain acids are not deposited in tissue as such, they may be converted into longer-chain acids and then deposited. Mead et al. (7) observed that in mice the relative amount of 24-hour $C^{14}O_2$ excretion of orally administered 1-C^{14}-methyl esters was oleate>linoleate>stearate. No allowance was made for degree of absorption. Bernhard et al (8) found no difference between the rates or extent of oleic and linoleic oxidation. In a different study in this laboratory (as yet unpublished) in which $C^{14}O_2$ was collected over a 72-hour period, oleate was found to be 64% excreted as $C^{14}O_2$ as compared to 52% of linoleate, in general agreement with Mead (7) but in disagreement with the present results. No doubt several factors, such as the levels in the tissues and the diet, influence the relative degrees of combustion of various fatty acids.

Another factor regulating the quantity and type of fatty acids deposited may be the influence of dietary fat on fatty acid synthesis. Hill et al. (9) observed that dietary fat per se inhibits in vitro fatty acid synthesis by liver tissue of rats. Bottino et al. (5) have shown this to be true in vivo for adipose tissue as well. Reiser et al. (10) have shown that inhibition of fatty acid synthesis in the liver is greater during unsaturated than during saturated triglyceride ingestion. Di Giorgio et al. (2) have demonstrated also that inhibition of lipogenesis by adipose tissue in vivo is not solely dependent on the quantity of diet fat, but that the nature of the fatty acids in the dietary fat is also a factor.

The present investigation was undertaken to further elucidate the metabolic fates of caprylic, myristic, palmitic, oleic, and linoleic acids derived from the diet and to clarify the quantitative effects of fatty acid oxidation, *de novo* synthesis, and interconversions on the composition of tissue fat.

Experimental

Five groups of Holtzman strain male rats (6 per group) 150 ± 10 g were fed a basal diet (Fat-Free Diet, Nutritional Biochemicals Corp., Cleveland, Ohio; further fortified with vitamins) to which was added 15% (85 g basal diet + 15 g of triglyceride) tricaprylin, trimyristin, tripalmitin, triolein, or trilinolein for one month. One percent safflower oil was added (99 + 1) to diets containing tricaprylin, trimyristin, and tripalmitin to meet the essential fatty acid requirement. A control group of 15 animals was fed the 1% safflower oil diet. The fatty acids used in the synthesis of the dietary triglycerides were fractionally distilled as their methyl esters, using a 4 ft × 1 in. column packed with glass helices and a pressure of 4–10 μ. The distilled methyl esters were converted to the respective simple triglycerides according to the method of Konen et al. (11). Table I shows the fatty acid composition of the triglycerides fed.

After feeding the respective diets for one month, 200 to 300 mg of 1-C^{14}-fatty acid methyl esters (0.1 to 0.2 mc), identical to the fatty acid of their dietary triglycerides, were administered by stomach tube to each animal. The control group was divided into 5 subgroups of 3 animals each and given the 5 tracer acids, respectively. The time of administration was approximately 9:00 AM, since preliminary experiments had shown that the stomachs of the animals contained food at that hour, a condition considered normal for a study of the fate of dietary constituents.

Immediately after administration of the 1-C^{14}-fatty acid methyl esters, the animals were placed in a respirometer (12). Respiratory CO_2 was collected over 8 consecutive 30-minute periods. The amounts of CO_2 trapped by 0.5 N NaOH solution during each 30 minute period were determined by titration of the excess alkali after precipitation of the carbonate as the barium salt with a 30% $BaCl_2$ solution. The

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TABLE I

Fatty Acid Composition of the Dietary Triglycerides

Dietary triglycerides	Fatty acids[a]									
	6:0	8:0	10:0	12:0	14:0	16:0	16:1	18:0	18:1	18:2
	Percent									
Caprylate	2.0	95.0	3.0							
Myristate				2.0	97.0	1.0				
Palmitate				1.0	2.0	91.0		6.0		
Oleate							11.0		86.0	3.0
Linoleate							1.0		3.0	96.0
Safflower Oil						8.0		3.0	15.0	74.0

[a] The number before the colon indicates the number of carbon atoms; the number after the colon, the number of double bonds.

$BaCO_2$ samples were dried, weighed, and assayed for C^{14} using the gel-scintillation method of Nathan et al. (13).

Immediately after the 4-hour collection of CO_2 the animals were killed by exsanguination. Gastrointestinal tracts were removed and washed free of the remaining C^{14}-fatty acids with water and petroleum ether. The radioactivtiy in the lipid extracted from these washes was used to calculate the amounts absorbed. All results are based on the amounts of radioactivity absorbed.

Carcass (including washed GI tracts) and liver lipids were extracted using the method of Folch et al. (14). The extracted but unfractionated lipids were saponified with alcoholic KOH and the fatty acids were removed by extraction of the acidified mixture with petroleum ether. Methyl esters were prepared with either diazomethane (15) or boron trifluoride-methanol (16). Mixed lipids, fatty acids, or their methyl esters were assayed for radioactivity in 20 ml of a toluene solution containing 4.0 g of 2,5-diphenyloxazole (PPO) and 100 mg of 1,4-di(2,5-phenyloxazoyl)benzene (POPOP) per liter. All radioactivity determinations were made with a Packard Liquid Scintillation Spectrometer.

Analytical gas-liquid chromatography (GLC) (Katharometer; 6 in. x 1/4 in. column packed with 20% diethylene glycol succinate (DEGS) on 60–80 mesh Chromosorb-W; helium flow rate of 70–80 ml/min; sample size of approximately 1 mg) was used for the determination of all fatty acid composition. Preparative GLC Katharometer; 9 ft x 5/8 in. column packed with 30% DEGS on 60–80 mesh Chromosorb W; helium flow rate of 500–600 ml/min; sample size of 100–300 mg fatty acid methyl esters) was used for separation and collection of individual fatty acid methyl esters. To protect the preparative column from the possibility of C^{14} contamination through ester exchange, the columns were flushed before each collection with a 100 mg sample of unlabeled methyl caprylate. The eluting aerosols were coalesced and collected (80–90% of the theoretical) by heating (300C) about 1/4. in of a 1/4 in. x 6 in. glass collection U-tube with a nichrome wire spiral a few inches from the collection port. A Research Specialties gas chromatograph equipped with an argon-ionization detector was used to check the purity of the samples. Two mutually contaminated adjacent eluting esters such as stearate and oleate, could be corrected for specific activities by the use of simultaneous equations since their composition and mixed specific activities were known.

TABLE II

Effect of Dietary Triglycerides on Final Body Weight, Total Carcass Lipids, and Total Liver Lipids

Dietary groups[a]	Number of animals	Weight gain[c]	Total carcass lipids[d]	Total liver lipids[d]
		g	%	%
Tricaprylin[b]	6	206±27[e]	15.0±1.4[e]	5.4±0.6
Trimyristin[b]	6	150±10	11.6±0.9	5.0±0.2
Tripalmitin[b]	6	168±19	11.0±1.1	5.3±0.5
Triolein	6	203±24	11.9±1.5	6.9±2.1[e]
Trilinolein	6	146±21	11.8±2.3	5.8±0.8
Low-fat[b]	15	155±32	13.4±1.7[e]	4.7±0.5

[a] The triglycerides were present in the diet at a level of 15%.
[b] 1% (w/w) safflower oil added to the diet.
[c] Mean weight gain of rats with standard deviation. Initial weights 150±10 g.
[d] Expressed as percentage of wet tissue.
[e] Significantly different ($P<0.01$) when compared to the other dietary groups.

Since the methyl stearate fraction usually contained oleate plus an unlabeled oleic acid isomer (probably vaccinic acid) (17) this fraction had to undergo further purification as the mercuric acetate derivatives (18). The total radioactivity of any particular fatty acid in the carcass or in a tissue was calculated from percentage composition data, the total weight of the fatty acid in the tissue, and its specific activity. Although corrections were made according to Horrocks et al. (19), this calculation involved a 2–5% error due to variable detector response for the wide range of molecular weights present in a sample. Therefore, some probable sources of error in this phase of the experiment are: (a) calculation of individual fatty acid concentrations from GLC data; (b) incorporation of activity into minor fatty acids (15:0, 17:0, 17:1, etc.) which were not collected and measured for radioactivity; and (c) possible ester exchange of fatty acid methyl esters with succinic acid esters of the column. Although a certain percentage of the C^{14} radioactivity remained unaccounted for, the data were

TABLE III

Effect of Dietary Triglycerides on the Percentage Composition of Liver and Carcass Fatty Acids[a]

Dietary group[b]	Tissue	Total fatty acids[d]	Fatty Acids[e]							
			14:0	16:0	16:1	18:0	18:1	18:2	20:3[g]	20:4[h]
		g	%	%	%	%	%	%	%	%
Tricaprylin[c]	Liver[j]	0.396±0.049	0.5±0.3	25±4.7[i]	7.3±0.8	18.0±2.9	33±1.7	4.6±1.4	5.1±2.3	4.4±1.7
	Carcass	45.0 ±4.6	1.7±0.2	31±2.3	12.0±1.2	3.2±1.0	49±3.1	2.1±0.6		
Trimyristin[c]	Liver	0.340±0.028	6.0±1.4[i]	25±1.9[i]	5.6±0.6	17.0±2.1	32±2.1[i]	4.3±1.0	3.8±1.4	4.8±0.9
	Carcass	26.0 ±2.2	25.4±4.6[i]	24±2.0	11.0±3.0	2.6±0.6	31±4.4	3.5±0.5		
Tripalmitin[c]	Liver	0.315±0.027	0.4±0.1	28±4.7[i]	8.1±1.7	14.0±3.7	35±2.9	3.9±1.7	4.2±1.3	4.3±2.0
	Carcass	29.0 ±2.5	1.6±0.2	38±2.5[i]	11.0±1.1	3.8±0.7	41±1.0[i]	3.0±0.9		
Triolein	Liver	0.621±0.196	0.4±0.2	15±1.9[i]	4.4±1.3[i]	10.0±3.3[i]	50±3.1[i]	6.3±1.2	3.1±1.0	7.0±1.0
	Carcass	36.0 ±4.8	1.1±0.3	18±1.6[i]	7.0±1.9[i]	2.0±0.5	67±2.6[i]	3.3±0.3		
Trilinolein	Liver	0.409±0.074	0.4±0.1	25±7.2[i]	1.9±1.2[i]	18.0±2.2	11±2.3[i]	30.8±7.6[i]	2.3±0.7	11.4±2.4[i]
	Carcass	27.0 ±5.8	1.5±0.2	26±2.6	5.0±1.8[i]	3.6±0.6	20±3.8[i]	42.9±4.2[i]		
Low-fat[c]	Liver	0.302±0.036	0.5±0.3	22±2.3	7.2±2.1	19.0±2.1	34±4.0	5.4±2.5	4.6±3.3	6.5±3.1
	Carcass	34.0 ±4.4	1.6±0.4	28±2.0	11.0±1.6	3.1±1.6	51±4.2	4.6±3.1		

[a] The low-fat dietary group contained 15 animals, all others contained 6.
[b] The triglycerides were present in the diet at a level of 15%.
[c] 1% safflower oil added to the diet.
[d] The mean total concentration of liver carcass fatty acids with standard deviation.
[e] GLC fatty acid percentage composition based on the total area of the chromatogram.
[g] 5,8,11-eicosatrienoic acid.
[h] 5,8,11,14 eicosatetraenoic acid.
[i] Significant difference ($P < 0.01$) when compared to the values obtained for the fatty acid composition of the low-fat group.
[j] The liver from animals fed tricaprylin contained 1.4% of 8:0–12:0 fatty acids and the carcasses of the animals fed trimyristin contained 2.5 ± 2.0% 14:1 acid. All other animals had only traces of these acids in their triglycerides.

TABLE IV

The Percent of the Absorbed 1-C^{14} Fatty Acid Methyl Ester Radioactivity Incorporated into Respiratory Carbon Dioxide, and Carcass and Liver Fatty Acids 4 hrs after Administration

Dietary group[a]	Labeled 1-C^{14} methyl ester administered	Number of animals	Absorbed C^{14} esters	Expired[d] $C^{14}O_2$	Carcass fatty acids[d]	Liver fatty acids[d]
			%	%	%	%
Tricaprylin[b]	MeCaprylate	6	38	70	17	5.9
Low-fat[b]	MeCaprylate	3	30	72	19	4.2
Trimyristin[b]	MeMyristate	6	50	37	48	6.9
Low-fat[b]	MeMyristate	3	42	36	56	3.6
Tripalmitin[b]	MePalmitate	6	26	23	62	8.0
Low-fat[b]	MePalmitate	3	57	6	84	2.5
Triolein	MeOleate	5	58	35	49	10.9
Low-fat[b]	MeOleate	3	39	19	74	4.9
Trilinolein	MeLinoleate	5	47	53	33	6.8
Low-fat[b]	MeLinoleate	3		50	34	11.2

[a] The triglycerides were present in the diet at a level of 15%.
[b] 1% safflower oil added to the diet.
[c] Mean values with standard deviation of total carbon dioxide expired expressed as $BaCO_3$.
[d] Mean values with standard deviation based on the amount absorbed.

quite useful in the interpretation of fatty acid synthesis and tissue deposition.

Whenever possible, a statistical evaluation of the mean difference between groups was made using a Student *t*-test (20).

Results

Total Lipids of Carcass and Liver

Animals fed 15% tricaprylin or triolein had significantly ($P<0.01$) greater mean weight gains than animals fed trimyristin, tripalmitin, trilinolien or 1% safflower oil (Table II). The greater final weight of the tricaprylin-fed rats was due to the high percentage ($15.0\pm1.4\%$) of their weight as fat.

Fatty Acid Composition of the Carcass and Liver Lipids

The carcass fatty acid changes effected by the diets were more pronounced than those of the liver (Table III). In general, however, diet myristate, palmitate, and linoleate were deposited at the expense of the tissue monoenes, and diet oleate at the expense of palmitate. There were other minor adjustments.

Diet caprylate was not deposited and did not disturb the purely endogenous proportions in the tissues.

The Absorption of 1-C^{14}-Fatty Acid Methyl Esters

From 30 to 50% of the ingested 1-C^{14}-fatty acid methyl esters (50–150 mg containing 20–100 x 10^3 disintegrations per minute) were absorbed within 4 hr after administration. Total recovery of radioactivity in these experiments was calculated to be within the range of 80–90%. Although the values of the percentages of administered label absorbed during the 4 hr may appear small, it should be recalled that this was after only 4 hr. The reasons for the higher degrees of absorption in the test than in the control animals except the palmitate, may only be speculated upon, but may be due to the relatively small amount of lipid material in the intestine.

Oxidation of 1-C^{14}-Fatty Acid Tracers as Measured by $C^{14}O_2$ Production

The percentages of absorbed labeled acids catabolized were calculated from the amounts of C^{14} fed, the amounts unabsorbed, and the amount excreted as $C^{14}O_2$. The amounts of absorbed labeled acids incorporated in the liver and carcass were similarly calculated (Table IV).

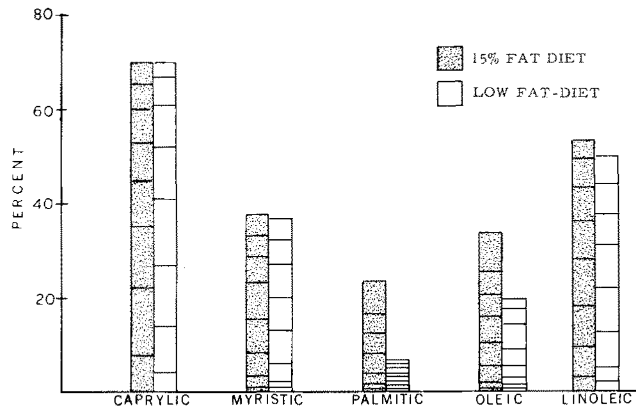

FIG. 1. Per cent of absorbed 1-C^{14} labeled acids excreted as $C^{14}O_2$ in ½ hour periods for 4 hours.

During the first 4 hr after administration to animals on the fatty diets, tracer short-chain caprylic acid was 70% oxidized to $C^{14}O_2$, medium-chain myristic acid 35%, and the longer-chain palmitic acid only 23% (Table V). Oleic acid was 35% oxidized and linoleic 50%. Thus the rates of catabolism of the acids varied inversely with their chain length but directly with their degree of unsaturation. In contrast to the fatty diets only 6–19% labeled palmitate and oleate, respectively were oxidized when given to the animals on the control diets.

The rates of oxidation of labeled caprylate, myristate, and linoleate were maximal within the first 2 hr (Fig. 1) after administration. Labeled palmitate and oleate did not reach their maximum rate of oxidation until the third and fourth hours.

Incorporation of Absorbed 1-C^{14}-Fatty Acid Activity into the Fatty Acid Activity of Carcass and Liver Lipids

In conformity with classical observations, practically all of the label of ingested caprylate was found in carcass acids other than caprylic (Table V). In the carcasses of the test animals approximately 64, 89, and 75% of the label of dietary myristic, oleic and linoleic acids respectively were present there unchanged. The distribution of the remainder indicates both elongation and oxidation to acetate followed by resynthesis.

With the possible exception of palmitate there was little difference between the degrees of deposition of labeled acids in the carcass fat of the test and control groups. Less labeled diet palmitate appeared in tissue palmitate of the control group than of the test group, 65 and 81%, respectively.

Liver fatty acids contained lower percentages of the diet acids than the carcass, and had a wider distribution of the administered tracer than those of the carcass, indicating a higher level of metabolic activity. An especially interesting phenomenon is that after C^{14}-myristic acid administration only 3% of liver C^{14} was in the myristic acid of the control group compared to 35% in the test group. The difference made its appearance in the liver palmitic acid.

Labeled oleic acid, like myristic, was deposited to a higher degree in the test than in the control livers, the difference being found in palmitic acid.

Discussion

In general, the results of the study show that the extent to which diet fatty acids modify tissue fatty acid composition depends upon the degrees to which

TABLE V

The Distribution of C^{14}-Activity Among the Fatty Acids of Liver and Carcass 4 hrs after Administration of 1-C^{14} Fatty Acid Methyl Esters

Dietary group[b]	Tissue	Labeled acid given	Number of animals	Fatty acids 14:0	14:1	16:0	16:1	18:0	18:1	18:2	20:3[d]	20:3[e]	20:4[f]	Percent recovered[g]
				Percentage of total activity incorporated in the individual acids										
TriCAP[c]	Liver	MeCAP[c]	6	0.1		37.1	3.9	17.3	15.2	—	0.5	0.1	0.3	74.5
	Carcass	"	6	3.5±1.8		50.7±10.6	9.5±3.5	4.9±2.5	17.9±9.9					86.8±10.2
Low-fat	Liver	"	3	0.3		43.2	5.8	19.3	24.3	—	0.9	—	0.4	94.1
	Carcass	"	3	1.4±0.4		59.5±17.4	5.0±1.8	2.4±1.2	13.9±4.9					82.8±10.1
TriMYR	Liver	MeMYR	6	35.1	0.8	32.1	4.1	9.4	8.2	—	0.8	0.1	1.4	91.9
	Carcass	"	6	64.1±6.0	3.8±1.9	6.6± 3.5	1.7±1.2	0.7±0.5	2.2±0.9					78.9± 5.3
Low-fat	Liver	"	3	3.0		51.2	1.9	3.2	7.0	—	0.6	—	0.2	67.1
	Carcass	"	3	63.0±2.9	0.5±0.5	5.1± 1.9	1.6±1.1	2.4±2.3	4.1±2.8					76.9± 7.5
TriPAL	Liver	MePAL	6	0.2		53.8	2.0	8.3	7.6	—	0.8	—	0.1	77.8
	Carcass	"	6	0.5±0.5		81.1± 6.7	6.6±1.8	1.2±1.0	3.4±1.9					92.9± 5.8
Low-fat	Liver	"	3	0.2		58.1	1.0	13.5	7.6	—	0.4	—	1.2	81.8
	Carcass	"	3			65.4± 9.0	11.1±1.7	0.5±0.4	3.6±0.7					85.9± 8.5
TriOL	Liver	MeOL	5	0.4		9.5	0.3	12.7	65.3	—	4.4	0.1	0.6	93.3
	Carcass	"	6	0.2±0.2		1.5± 0.4	0.1±0.1	0.8±0.3	89.6±3.7					92.2± 3.8
Low-fat	Liver	"	3	0.1		45.0	1.6	10.8	29.5	—	0.8	0.1	0.4	88.3
	Carcass	"	3	0.4±0.2		4.8± 2.8	0.3±0.1	0.5±0.2	87.9±9.6					94.0± 4.0
TriLINOL	Liver	MeLINOL	5	0.2		27.2	0.8	7.4	4.5	41.9	0.5	1.0	3.2	86.8
	Carcass	"	5	0.4±0.6		3.4± 1.4	0.1	0.4±0.4	0.9±0.7	78.8±6.4				83.9± 5.5
Low-fat	Liver	"	3	0.3		23.2	0.9	3.3	4.7	32.7	3.2	—	1.7	70.0
	Carcass	"	3	0.8±1.0		4.3± 1.1	0.4±0.2	0.6±0.1	5.2±3.7	69.9±5.1				81.1± 4.6

[a] The carcasses triglycerides were analyzed individually, but the liver triglycerides were pooled for analyses. Thus standard deviation is given for the carcass values only.
[b] The triglycerides were present in the diet at a level of 15%; low-fat diet contained 1% safflower oil.
[c] Tri- designates triglyceride; Me-, methyl; CAP, caprylate; MYR, myristate; PAL, palmitate; OL, oleate; LINOL, linoleate.
[d] 5,8,11 eicosatrienoic acid.
[e] 8,11,14 eicosatrienoic acid.
[f] 5,8,11,14 eicosatetraenoic acid.
[g] Percentage of total tissue fatty acid C^{14}-activity recovered after preparative GLC.

the diet acids are catabolized, stored unchanged, and converted to other acids. The effect of the diet acids on lipogenesis is also an important factor. The 8-carbon caprylic acid does not upset the fatty acid relationships of endogenous fat because it is mainly catabolized to CO_2, does not inhibit fatty acid synthesis (3), is not a normal end product of endogenous fatty acid[1] biosynthesis, and is not directly deposited after being fed. As used here "endogenous fatty acids" are those produced from nonfatty precursors.

Dietary palmitic acid also has little effect on the endogenous fatty acid composition of tissue lipids, but for different reasons. In contrast to caprylic acid it does inhibit fatty acid biosynthesis and it is deposited to a very high degree. Also, it is directly convertible to stearic, oleic, and other endogenous fatty acids. Therefore, diet palmitic acid simply replaces endogenous palmitic acid. The level fed (15%) might exceed that endogenously produced since the adipose tissue level was greater (38%) on the tripalmitin than on the low-fat control diet (28%). However, this might also reflect a difference in triglyceride structure. Endogenous rat fat approaches randon distribution (21). Feeding a fat of different structure, such as tripalmitin, might affect depot fatty acid composition since endogenous fat is resynthesized 80% back to its original structure (22). These influences on adipose tissue fatty acid composition cannot as yet be assessed.

Dietary myristic, oleic, and linoleic acids, in contrast to caprylic and palmitic, upset the normal endogenous pattern of tissue fatty acid composition, but each for different reasons. Myristic acid, like palmitic acid, is convertible to endogenous fatty acids. However, it is not the major end product of fatty acid biosynthesis (23, 24), its elongation to palmitic evidently being limited. Different from caprylic, it may be stored, so that those fractions not elongated or burned to CO_2 are deposited in the tissues at a higher level (25%) than is present in purely endogenous fat (2%) (Table III).

Oleic acid, in contrast to myristic, is not directly converted to other endogenous acids except to a very limited degree. It does inhibit fatty acid biosynthesis (3). Therefore, it accumulates in the tissues while the constituent palmitic and the other endogenous acids gradually disappear through metabolic utilization.

Like oleic acid, dietary linoleic is not converted to endogenous acids and prevents endogenus fatty acid synthesis. It therefore accumulates. However, it is oxidized to CO_2 to a greater degree than oleic[1] and its level in tissues does not reach values as high as oleic when similarly fed.[1] Any differences between oleic acid and linoleic acid deposition are products of the differences in their metabolic patterns. (a) Oleic acid is an endogenous acid and linoleic is not. (b) Linoleic acid is incorporated into phospholipids to a greater degree than oleic (5), probably a reflection of its essential functions. (c) Linoleic acid is incorporated differently than oleic into triglycerides, though these relationships are not yet clear.

[1] In another study under different conditions (5) more labeled oleic acid than linoleic was excreated as $C^{14}O_2$.

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