TABLE V

Glyceride Type Distribution of Beef Fat

Glyceride type	SSS	SUS	SSU	USU	UUS	UUU
Locality of fat						
Bottom back rear	14.6	25.2	16.9	4.9	29.7	8.8
(random calculated)	17.1	13.7	27.4	11.0	22.0	8.8
Back bottom rear[a]	13.9	27.2	14.0	3.5	30.6	7.6
(random calculated)	13.1	12.7	25.4	12.3	24.6	11.9
Perinephric fat	20.2	32.8	15.1	2.8	26.3	4.5
(random calculated)	35.3	14.6	29.2	6.1	12.1	2.5
Visceral pericardial fat	15.4	41.1	10.4	1.7	27.2	4.5
(random calculated)	25.1	14.7	29.4	8.6	17.2	5.0
Rib cavity	13.7	21.6	18.8	6.4	29.4	10.1
(random calculated)	15.2	13.3	26.6	11.6	23.2	10.1

[a] From another animal.

TABLE VI

Glyceride Type Distribution in Pork Fat

Anatomical position	SSS	SUS	SSU	USU	UUS	UUU
Bottom rear back	8.0	3.4	31.4	30.8	13.3	13.0
(random calculated)	9.9	11.6	23.0	13.6	27.2	15.4
Kidney	7.7	8.9	37.3	44.7	4.3	5.1
(random calculated)	12.1	12.7	25.4	12.6	25.2	12.8
Top rear back sirloin	4.9	1.1	38.4	46.6	8.6	10.5
(random calculated)	8.6	10.9	21.8	13.8	27.6	17.4
Flank fat	5.9	2.3	29.5	36.3	11.6	14.3
(random calculated)	8.1	10.6	21.2	13.8	27.6	18.3
Back fat layer (shoulder) Upper 1 cm	2.6	0.7	23.0	51.4	6.8	15.2
(random calculated)	5.9	9.3	18.6	14.5	29.0	22.7
Back fat layer (shoulder) Lower 1 cm	7.7	7.7	22.6	18.2	23.9	18.3
(random calculated)	7.9	10.5	21.0	14.0	28.0	18.6
Back top[a]	4.5	2.1	26.2	37.5	12.2	17.9
(random calculated)	6.5	9.7	19.3	14.4	28.7	21.4
Back bottor[a]	5.8	1.6	30.8	41.2	8.8	11.7
(random calculated)	8.5	10.8	21.7	14.0	28.0	17.6

[a] Different animal.

triunsaturated triglyceride composition of sheep fat.

In contrast to the recent report of Barford et al. (13) the results obtained in the present study indicate that considerable differences exist between the percentages of any given fatty acid located in the 2- position of the glycerol moiety among the various tissues examined. A comparison of the results obtained when the glyceride distribution was calculated from pancreatic lipase hydrolysis data and that which would be expected from a random distribution indicates that the depot fats examined here are distributed in a nonrandom manner.

TABLE VII

Glyceride Type Distribution in Sheep Fat

Glyceride type	SSS	SUS	SSU	USU	UUS	UUU
Locality of fat:						
Outer side of back fat layer						
(1 cm depth)	14.4	24.0	17.6	5.3	29.4	8.9
(random, calculated)	15.7	13.6	26.8	11.5	23.0	9.8
Inner side of back fat layer						
(1 cm depth)	17.7	37.6	12.2	0.2	25.9	4.9
(random, calculated)	21.8	14.4	28.8	9.5	19.0	6.3
Fat attached to rib inside of rib cavity	20.4	34.6	14.0	4.1	23.8	4.1
(random, calculated)	23.7	14.6	29.2	9.0	18.0	5.5

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[Received April 7, 1965—Accepted August 2, 1965]

Dietary Fatty Acids: Their Metabolic Fate and Influence on Fatty Acid Biosynthesis[1]

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Abstract

Adult rats fed a low-fat diet or diets containing 15% of either tripalmitin, triolein or trilinolein were injected intraperitoneally with H^3-labeled acetate. Those which received fat were also given by mouth, simultaneously with acetate, the 1-C^{14}-labeled sodium salt of the respective dietary fatty acid. The fate of the tagged material was followed by time-spaced biopsies of subcutaneous adipose tissue and by collection of the expired $C^{14}O_2$.

After 72 hr, 51, 64, and 52% of the dietary palmitate, oleate, and linoleate, respectively, were catabolized, as indicated by the corresponding percentages of the label having been excreted as $C^{14}O_2$. Dietary linoleate was relatively less incorporated into body triglycerides than palmitate and oleate. Animals ingesting diets of 15% triolein had only about one-half the amount of phospholipids in their tissues as had the other groups.

The distribution of both the C^{14} and H^3 labels in the tissue triglycerides showed that all diets containing fats decreased fatty acid synthesis but did not inhibit conversion of palmitate to oleate. Conversions of oleate or linoleate appeared to be through acetate. As a result of these factors, the fatty acid composition of the tissue triglycerides after 3 months' ingestion of tripalmitin was essentially the same as that of the low-fat group, whereas the ingestion of triolein produced triglycerides with a very high content of oleic acid. Trilinolein ingestion produced effects similar to triolein but to a less pronounced degree.

Both the respiratory $C^{14}O_2$ and the C^{14}- and H^3-labeled fatty acids in subcutaneous adipose tissue exhibited a second rise in specific activity 12 to 24 hours after the administration of the label.

[1]Presented at the AOCS meeting, Chicago, 1964.

Introduction

THE OBJECTIVE OF THIS STUDY was to obtain information on the differences if any, in the metabolic fates of typical saturated, monounsaturated, and diunsaturated dietary fatty acids. The specific phenomena studied were (a) the relative degrees of catabolism and storage of dietary fatty acids; (b) the conversion of dietary fatty acids to other acids and (c) the influence of dietary acids on the synthesis of fatty acids from acetate. The fates of the dietary fatty acids were determined by the use of tracer amounts of carbon^{14}-labeled acids, while fatty acid synthesis was measured by following the incorporation of tritium-labeled acetate in the triglycerides of the same animals.

Experimental

Sixteen male, one-year-old albino rats, reared on a stock ration and weighing from 373 to 409 g each, were fed a low-fat diet (1) until the level of linoleic acid in their depot fat was reduced to around 5%. The animals were then distributed into four groups. Group 1 was kept for another 3 months on the low-fat diet and groups 2, 3, and 4 were given diets containing 15% by weight of tripalmitin, triolein, or trilinolein, respectively, until a constant body weight was attained. Under this condition, the amount of fat in the depots may be considered constant, thus enabling the determination of the biological half-lives of their component fatty acids. Tripalmitin and triolein were given for 3 months. Since the animals were adults and had about 5% linoleic acid in their depots, supplementation of the diets with linoleic acid was not considered necessary. Trilinolein was given for a month. Both the preparation of feed and cleaning of feeders were performed daily.

During the feeding period the digestibility of the tripalmitin, mixed as reprecipitated fine powder with the low-fat diet, was tested by extracting the fat from the feces and comparing the amounts in the fecal fat with the amounts consumed.

At 9 am on the day of the test, the tripalmitin fed group was given by stomach tube approximately 0.14 mc of sodium palmitate-1-C^{14} per kg of body weight, in aqueous solution. The triolein fed group was given 0.12 mc of sodium oleate-1-C^{14} per kg, and the trilinolein fed group 0.36 mc of sodium linoleate-1-C^{14} per kg. Immediately afterwards, the animals were injected intraperitoneally with approximately 2 mc of sodium acetate-2-H^3 per kg of body weight. The low-fat control group was administered 0.38 mc/kg of the H^3-labeled acetate but no labeled fatty acids. After the acetate injection and during the remainder of the experiment except for periods of CO_2 collection, the animals were allowed access to their respective diets ad libitum. Biopsy samples of approximately 200 mg of adipose tissue were taken for fatty acid assay from the subcutaneous inguinal region of each animal at 0.5, 1, 2, 4, 8, 24, and 48 hours after the labeled acetate injection.

The animals which had received the labeled fatty acids orally were kept for approximately one-half hour in a respirometer (2) at approximately 1, 2, 4, 8, 14, 24, 32, 48, 56, and 72 hours after the administration of the labeled materials, and the respiratory $C^{14}O_2$ was collected. Immediately after the last $C^{14}O_2$ collection period the rats were sacrificed with chloroform and their livers and a portion of their adipose tissue removed. The weighed carcasses were then frozen and ground, and the fat quantitatively extracted from 10 g samples. In view of the small proportions of lipids other than triglycerides in adipose tissue, no effort was made to fractionate the lipids of that tissue. However, lipids extracted from the ground carcasses were fractionated by the silicic acid slurry procedure (3) into two fractions: (a) triglycerides, fatty acids, cholesterol, cholesterol esters and partial glycerides, and (b) phospholipids.

The fatty acids of the biopsy samples taken during the 72 hours were converted into methyl esters. From each sample an aliquot was taken for the determination of radioactivity, and the remainder was fractionated by preparative gas-liquid chromatography (GLC). Samples of the palmitic and oleic acids of groups 1, 2 and 3 and of palmitic, oleic and linoleic acid of group 4, were collected separately. Each of the fractions isolated was tested for C^{14} and H^3 radioactivity and for purity by analytical GLC. All determinations were made in duplicate.

TABLE I

Influence of Dietary Simple Triglycerides on the Amount of Total Lipid, Triglycerides, and Phospholipids in the Adult Rat[a]

Dietary fat	Body lipid[b]	Composition of Body Lipid[b]			
		Triglycerides[c]		Phospholipids	
	g/100g[d]	%	g/100g[d]	%	g/100g[d]
Tripalmitin	9.9 (8.5–13.4)	91.0 (89.1–92.9)	9.0 (7.7–11.9)	9.0	0.9 (0.4–1.5)
Triolein	10.7 (9.8–11.3)	95.0 (93.9–95.9)	10.2 (9.2–10.8)	5.0	0.5 (0.4–0.6)
Trilinolein	8.1 (7.9– 8.2)	89.7 (88.6–90.8)	7.3 (7.0– 7.5)	10.3	0.8 (0.8–0.9)
None	11.9 (8.3–14.0)	91.0 (86.8–93.0)	10.9 (7.2–12.8)	9.0	1.0 (0.8–1.2)

[a]Triglycerides were given at 15% (w/w) level in the diet to adult rats previously fed a low-fat diet. Tripalmitin and triolein were fed for 3 months, trilinolein for one month, and the low-fat diet for more than 3 months.
[b]Figures in parentheses indicate the range of the individual values.
[c]This fraction probably includes minor proportions of cholesterol, cholesterol esters, free fatty acids and partial glycerides (10).
[d]Grams per 100 g of carcass.

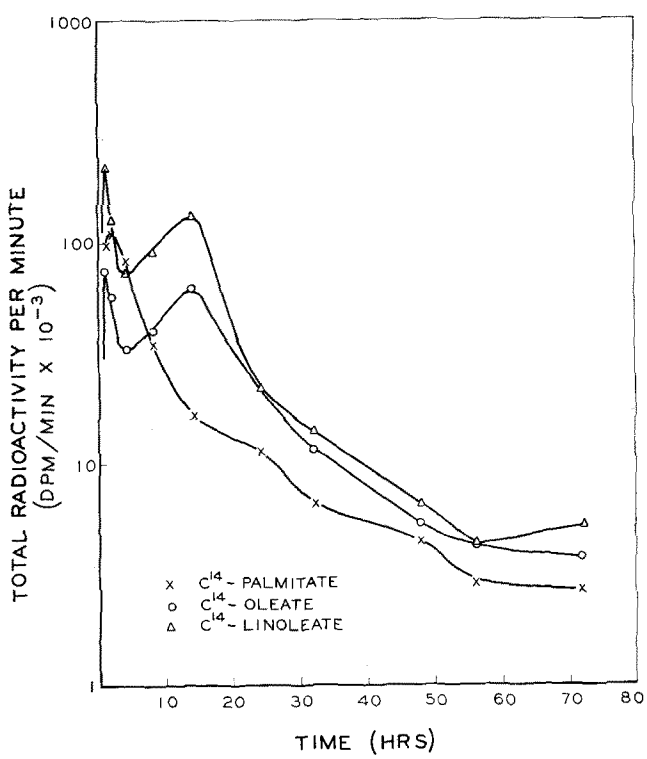


FIG. 1. $C^{14}O_2$ expired over a 72-hour period after the ingestion of either 1-C^{14}-labeled palmitate, oleate or linoleate.

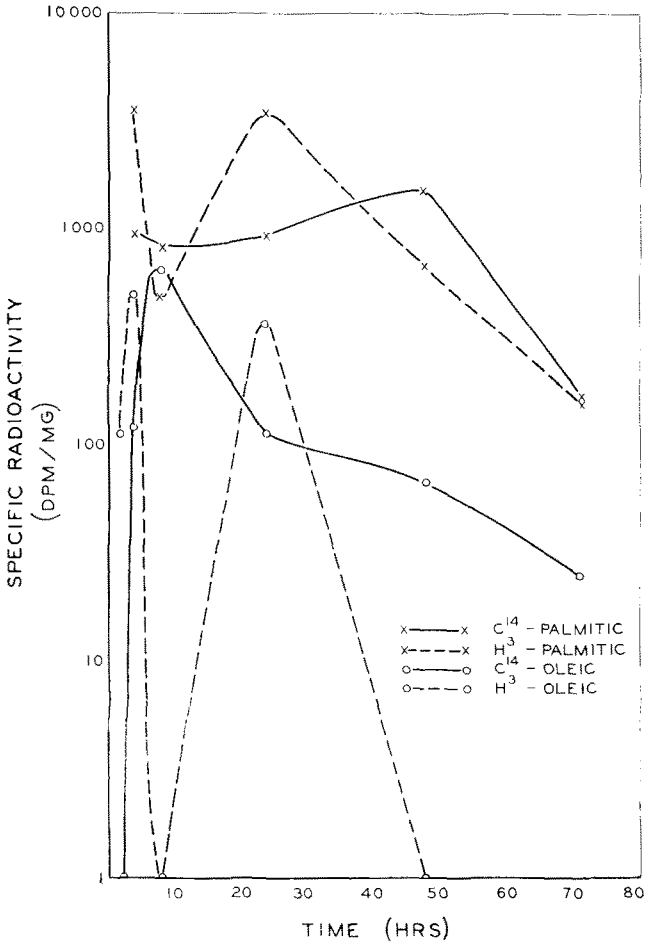
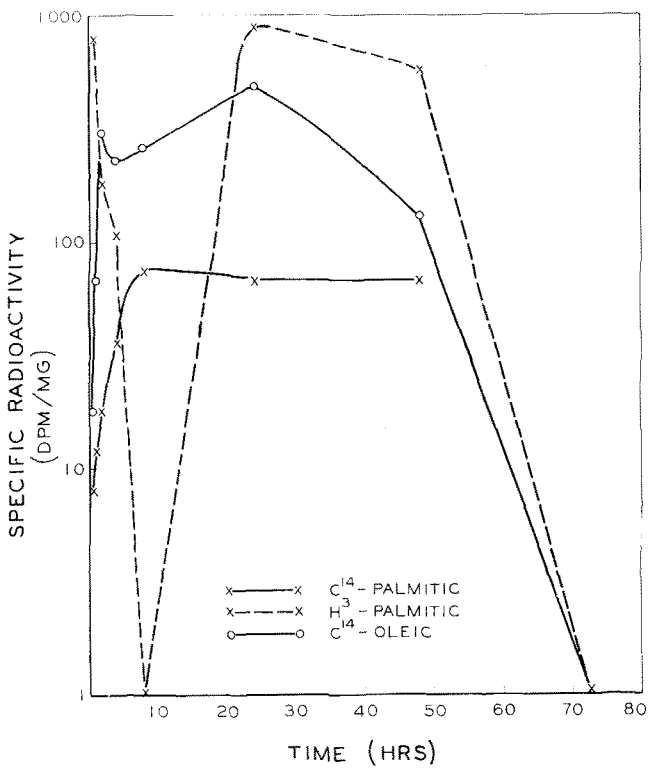


FIG. 2, A–D. Profile of the specific radioactivity curves of adipose tissue fatty acids over a 72-hour period following the administration of the labels.

2A. Rats fed 15% tripalmitin; 1-C^{14}-palmitate given orally, 2-H^3-acetate injected.



2B. Rats fed 15% triolein; 1-C^{14}-oleate given orally, 2-H^3-acetate injected.

Analytical Methods

Tripalmitin was prepared from commercial palmitic acid which had been crystallized from absolute methanol, ground and sieved through a No. 30 sieve. The palmitoleic, palmitic, and shorter chain fatty acids were partially removed from commercial oleic acid by fractional distillation of the methyl esters and subsequent short path distillation of the still-pot fraction. Methyl esters of both the fatty acid and fat samples were prepared by refluxing with a 2% solution of sulfuric acid in absolute methanol. Triglycerides were prepared by transesterification of the methyl esters with triacetin, using sodium methoxide as a catalyst, with a usual yield of about 80% triglycerides. No effort was made to separate the accompanying nonreacted methyl esters. Extractions of fat from tissues were performed by the method of Folch et al. (4). Fecal fat was determined by the method of van de Kamer et al. (5).

The fatty acid compositions of the dietary triglycerides and adipose tissue samples were determined by GLC on a 6 ft × ¼ in. column packed with 20% ethylene-glycol succinate polyester on Chromosorb W. An ionization detector was used. Preparative runs were performed with a similar apparatus in which a less sensitive thermal conductivity detector was substituted for the ionization detector. The amounts of methyl esters that were collected from the gas-liquid chromatograph and later counted were estimated according to the method of Bragdon (6).

C^{14} and H^3 radioactivities were determined simultaneously by means of a liquid scintillation spectrometer (Packard Tri-Carb, Model 314M), using a mixture of 2,4-diphenyloxazole and 1,4-bis-2-(4-methyl-5-phenyloxazolyl)-benzene in toluene. The exhaled $C^{14}O_2$ was trapped in 0.5 N NaOH and precipitated as $BaC^{14}O_3$ with 30% $BaCl_2$. The excess NaOH was determined by titration. The radioactivity of the $BaC^{14}O_3$ was determined in a scintillation spectrometer after dispersion in thixotropic gel (Cab-O-Sil, Packard Instruments Co.).

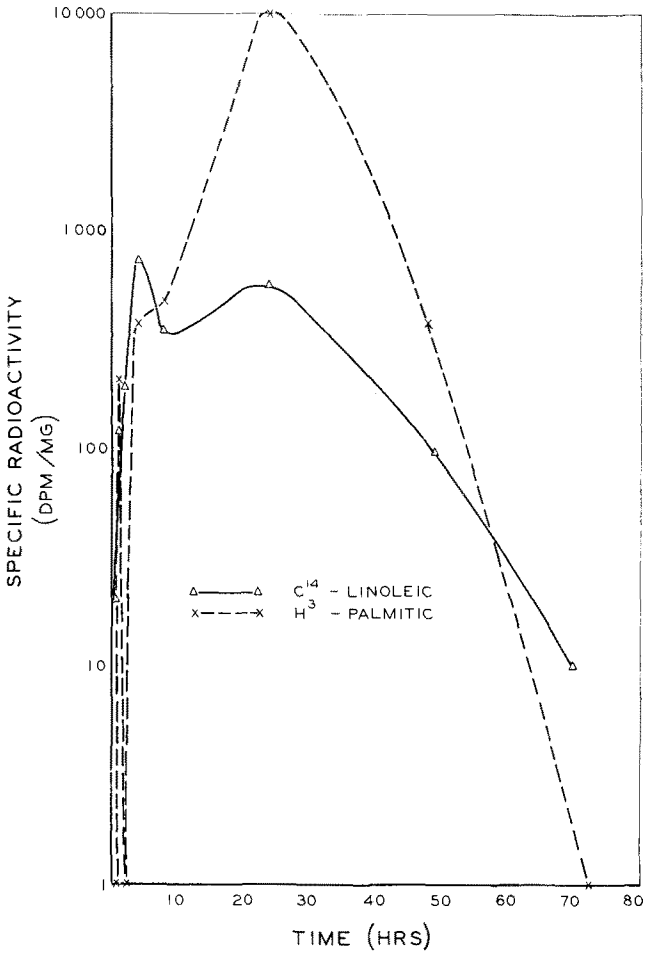
Results and Discussion

Absorption of Simple Triglycerides

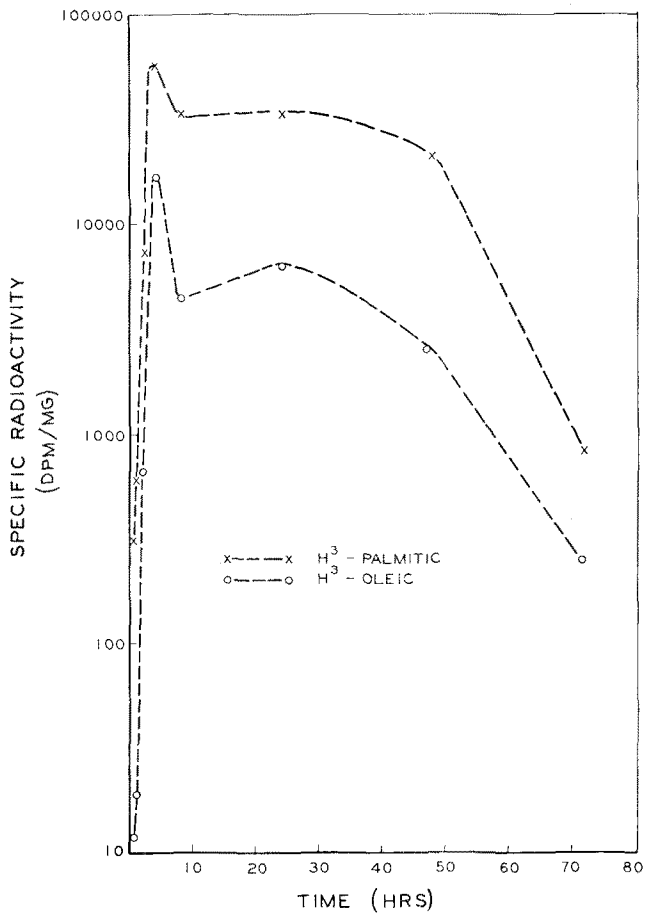
The tripalmitin in the diet was found to be 82% absorbed, in agreement with absorption values obtained by other methods (7,8). The lower figure of 46% found by Thomasson et al. (9) may be attributable to the much higher proportion of fat in their diet or, possibly, to the use of a less finely divided preparation. Triolein and trilinolein have been demonstrated to be well absorbed (9) and consequently were not tested for absorption in the present study. The daily consumption of diets containing tripalmitin, triolein, and trilinolein was found to be 14.8, 14.4, and 13.4 g, respectively.

Effect of Dietary Simple Triglycerides on Amounts of Body Lipids

The data in Table I depict the influence of the nature of the dietary fat on the amount and composition of body lipid in the adult rat. There was a lower amount of triglycerides in the carcasses of the trilinolein fed animals. As will be shown below and as reported previously to occur in liver (11), trilinolein feeding reduced fatty acid synthesis to very low levels. Also, as shown by the 26% level reached by linoleic acid in the depots of the corresponding



2C. Rats fed 15% trilinolein; 1-C^{14}-linoleate given orally, 2-H^3-acetate injected.



2D. Rats fed the low-fat diet; 2-H^3-acetate injected.

group (Table IV) and as have been repeatedly demonstrated in this laboratory (12,13) dietary linoleate is incorporated only to a limited degree in the depots. It then follows that an adult rat ingesting trilinolein as the only fat should have a lower percent of fat in the body (14) and consequently gain little weight (15). The former is apparent from the data in Table I, and the latter is shown by the shorter length of time required by the trilinolein fed group to reach a constant body weight: 1 month, as compared to 3 months by both the tripalmitin and triolein fed groups.

It has been reported (16,17) that tripalmitin also causes a depletion of body fats when fed to rats. Support for this cannot be drawn from the present data.

The administration of triolein resulted in a ratio of 19:1 of neutral fat to phospholipids in the carcass as compared to a ratio of 10:1 produced by the tripalmitin, trilinolein and the low-fat ration. Consideration of the amounts of neutral fat and phospholipid shows that the cause of the difference in ratio was the presence of half the amount of phospholipids in the body of the triolein fed animals as compared to the other groups, and not, as might be expected, the presence of more triglycerides. A possible explanation for this surprising observation is in the report of Bjorntorp et al. (18) who found in vitro that the presence in the medium of relatively high concentrations of oleic acid depressed oxidative phosphorylation, which has been demonstrated (19,20) to be necessary for phospholipid synthesis.

Relative Degrees of Storage and Conversion of Dietary Fatty Acids

The portion of radioactivity administered as C^{14}-labeled fatty acids but not exhaled as $C^{14}O_2$ was presumed to be incorporated into all the body tissues since the amount of fat excreted in the feces is comparatively very small (21,22).

The radioactivity (expressed as disintegrations per minute) in the $C^{14}O_2$ expired during each collection period cannot be integrated against time to obtain the radioactivity expired over the entire 72-hour period. However, if the radioactivity expired as $C^{14}O_2$ during each collection period is calculated as dpm per minute of collection, and this value is plotted against time, the integration becomes possible. Time in Figure 1 is the number of hours elapsed between the ingestion and halfway through each particular collection period. The area under the curve is thus a measure of the total radioactivity exhaled. The difference between this value and the radioactivity administered gives the portion of the label which was, theoretically, incorporated into the body. Thus calculated, the portions of dietary palmitate, oleate and linoleate incorporated into the body during a 72 hour period following ingestion were 49, 36, and 48% of the amount given, respectively. In general, these values are high when compared to those obtained by others on fasted (23,24) or very young animals (25,26). The explanation may be the sparing action of dietary carbohydrates on fat oxidation (23,27) since the animals in the present study were kept continuously in the presence of a 15% fat, 51% sucrose diet. Kirschner and Harris (8) have found that young rats under experimental

TABLE II

Incorporation of the Label of Dietary Tracer Acids Into Carcass Triglyceride Fatty Acids[a]

Dietary tracer acid	Tissue fatty acid		
	Palmitic	Oleic	Linoleic
		%	
Palmitate-1-C^{14}	16.2	5.7	....[b]
Oleate-1-C^{14}	0.2	14.9	....[b]
Linoleate-1-C^{14}	0.2	0.2	2.1

[a]Percent incorporation of the dietary tracer into carcass triglycerides calculated from the maximum specific activity in the adipose tissue. These calculations were based on the assumptions given in the text.
[b]Not determined.

conditions very similar to the present ones exhaled in 48 hours as much as 49.5% of the radioactivity given as oral palmitate-1-C^{14}. The value obtained in the present experiment for palmitic acid incorporation is consistent with the 44% recovery of dietary palmitic acid achieved by Stetten and Schoenheimer (28) in depot fat after 8 days.

The relative degree to which each of the fatty acids fed was deposited into triglycerides, either directly or after conversion to other acids, may be calculated as follows: $\%D = \frac{C \cdot A \cdot S}{T}$ where %D indicates percentage of the administered radioactive acid present in the body at the time of maximum incorporation; C, percentage of the acid in depot triglycerides; A, amount of total body triglycerides (mg); S, maximum specific activity reached by the acid in depot triglycerides (dpm/mg); T, total amount of radioactivity administered (dpm).

The value of %D may be used as a measure of the relative degrees of storage since the curves of deposition of the three acids have approximately the same shape (Fig. 2). This calculation admittedly gives only a rough approximation, since it assumes that all the body triglycerides had the same activity as the sample taken by biopsy. That this is not strictly true is shown by the fact that the specific activities of the palmitic and oleic acids in the carcass fat were approximately twice those in the subcutaneous fat taken at 72 hours. Benjamin et al. (29) have observed that various depot pools differ in metabolic activity, the subcutaneous pool being slower than others.

The values calculated as indicated above are presented in Table II. It can be seen that dietary labeled palmitic, oleic and linoleic acids at the times of their greatest incorporation were deposited in the carcass triglycerides at the relative levels of 16, 15, and 2%, respectively. The very low incorporation of dietary linoleic acid into depot triglycerides as compared to its incorporation into the entire body fat can only be explained by its being diverted into complex lipids and cholesterol esters, not examined in the present study. Hanahan et al. (30) have given C^{14}-palmitic and C^{14}-linoleic acids orally to rats and have found that they were preferentially incorporated in phospholipids rather than into triglycerides.

TABLE III

Incorporation of Injected H^3-labeled Acetate into Palmitic and Oleic Acids of Carcass Triglycerides[a,b]

Dietary Fat	Tissue fatty acids	
	Palmitic	Oleic
	%	
Tripalmitin	2.8	0.4
Triolein	0.2	0.0
Trilinolein	3.2	0.2
None	40.6	20.9

[a]No determination of linoleic acid was made as there is no synthesis of linoleic acid from acetate.
[b]Percent incorporation of the label into the carcass triglycerides calculated from the maximum specific activity in the adipose tissue. These calculations were based on the assumptions given in the text.

TABLE IV

Fatty Acid Composition of Carcass Triglycerides from Rats Fed Simple Triglycerides[a]

Fatty acid[b]	Tripalmitin		Triolein		Trilinolein		Low Fat
	Dietary fat	Adipose tissue	Dietary fat	Adipose tissue	Dietary fat	Adipose tissue	Adipose tissue
	%	%	%	%	%	%	%
12:0	1.2	0.4	....	trace	....	trace	0.2
14:0	2.1	2.1	....	0.5	....	1.6	2.0
14:1	....	0.3	....	trace	....	0.4	0.6
16:0	91.0	38.4	1.4	8.0	....	18.9	28.3
16:1	....	13.2	3.6	5.5	1.1	8.7	13.0
16:2?	....	trace	....	1.2	....	trace	trace
18:0	5.7	3.9	1.5	1.0	....	2.0	2.9
18:1	....	34.7	90.0	79.7	2.8	42.5	48.2
18:2	....	7.0	3.5	4.1	96.1	25.9	4.8

[a]See footnote *a*, Table I.
[b]The number before the colon denotes chain length. The number after the colon indicates the number of double bonds.

The presence of C^{14}-oleic acid in the depot fat of the tripalmitin-fed group equivalent to 6% of the C^{14}-palmitate given (Table II), confirmed observations by others (31,32) on the conversion of palmitic into oleic acid. On the other hand, conversion of dietary oleate or linoleate into other acids seemed to have not taken place except to the slight degree expected to occur through acetate, in agreement with the conclusions of Bernhard et al. (33) and of Stein and Stein (34,35).

Fatty Acid Synthesis as Affected by the Diet

De novo synthesis was determined by the incorporation of acetate-2-H^3 into the fatty acids of body triglycerides, in the same manner indicated above for the C^{14}-data. The resulting values are given in Table III.

There was little fatty acid synthesis, though not complete inhibition, in the animals that received fat, and a high degree of synthesis in those which did not, as expected. Also as expected there was more label incorporated in palmitic than in oleic acid.

The claim by Rou and Farvarger (36) that dietary palmitate favors the synthesis of palmitic acid could not be confirmed with the present data. However, there seemed to be an indication of higher palmitic acid synthesis in the tripalmitin- and trilinolein- than in the triolein-fed group.

Effect of Dietary Triglycerides on the Fatty Acid Composition of Carcass Triglycerides

The influence of the three dietary acids on the fatty acid composition of carcass triglycerides (Table IV) is clear reflection of the differences in their intermediary metabolism.

Dietary tripalmitin produced a relatively minor increase in palmitic acid and a decrease in oleic acid as compared to the low-fat diet. Dietary triolein, on the contrary, resulted in an increase in oleic from 48 to 80% and decreases in palmitic from 28 to only 8%, or in palmitoleic acid from 13 to 5%. Linoleic acid in the diet likewise was deposited at the expense of palmitic, palmitoleic, and oleic, though not to as great a degree as oleate. This dissimilar degree of deposition can be attributed only in part to the shorter feeding period of trilinolein, since the limited deposition of dietary linoleic acid in tissue has been repeatedly experienced in this laboratory (12,13).

The explanation for these observations may be as follows: All diet fats inhibit synthesis. Since palmitic acid is a precursor of palmitoleic, stearic and oleic acids, when fed it simply replaces endogenous palmitic and the balance of the normal endogenous acids remain relatively undisturbed. Exogenous oleic acid, on the other hand, is not converted into other acids so that they are gradually replaced by the oleate. Linoleic acid also is not the precursor of saturated or monoenoic fatty acids. Like oleic acid, therefore, it tends to replace the endogenous acids, but to a different degree. Moreover, it is more avidly incorporated into lipids other than triglycerides.

Comparison of the fatty acid composition of the triglycerides of various depots, such as subcutaneous, epididymal, and carcass fat (not presented in the table) showed no significant differences in confirmation of results obtained in humans (37).

Specific Activity-Time Relationships

The specific radioactivity-time curves for the expired $C^{14}O_2$ and for the labeled fatty acids in the subcutaneous adipose tissue are presented in Figures 1 and 2. A common feature of most of these curves is their biphasic nature. Analogous two-component curves have been reported in liver fatty acids (38–42) and in carcass fat (43) following injection with labeled acetate. In the present experiment the phenomenon is found in the curves depicting $C^{14}O_2$ production, fatty acid synthesis *de novo* in the adipose tissue, and incorporation of dietary acids into adipose tissue.

Tove et al. (40), in discussing similar biphasic curves in liver fatty acids after acetate injection, advanced the hypothesis that the second peak could be the result of the recycling of radiocarbon into the hepatic fatty acids from some other metabolite previously derived from the injected acetate. Later, Tove (44) suggested that it could be due to different speeds of esterification of the alcohol groups in the glyceryl moiety of the lipids.

It has been shown that various adipose tissues have different turnover rates (29) and conceivably, different degrees of affinity for newly absorbed or newly synthesized fatty acids. Exchange between different adipose tissues could thus explain the second rise in the more slowly metabolizing subcutaneous tissue examined in the present study.

In spite of the fact that the biphasic nature of the curves almost prevented the calculation of biological half-lives, some rough estimate could be made. In general, approximated half-lives of 5 to 22 hours were calculated from the slope of the second peak in the specific activity-time curves. The comparison of these values with the much higher ones recorded in the literature (reviewed in ref. 34) suggested that no complete equilibrium among the cytoplasmic and droplet compartments of the adipose cell (37,42, 45–48) was attained during the 3-day experimental period, or, in other words, that very little of the label was incorporated at that time in the very slow "fat droplet" compartment, not revealed in the above curves.

In considering the speed of incorporation of the label into the expired $C^{14}O_2$ and depot fatty acids, it has to be kept in mind that the label was administered as sodium salts of C^{14}-fatty acids. This could explain the presence of sharp peaks of radioactivity very shortly after the administration. Even under these conditions, the occurrence of a first peak of radioactivity in palmitic, oleic, and linoleic acids as early as 4 hours after the oral administration of the respective labeled acid, points to a rapid incorporation and turnover.

The appearance of both C^{14}-palmitic and C^{14}-oleic acid peaks within 8 hours after the administration of C^{14}-palmitate (Fig. 2a) indicates a rapid conversion of the incorporated C^{14}-palmitate into oleic acid taking place probably in the fast compartment (subcellular particles?) mentioned above.

ACKNOWLEDGMENTS

Neofat 16, 95% palmitic acid, by Armour Chemical Company, Chicago, Illinois. Oleic acid by A. Gross and Company, New York, N.Y. Linoleic acid by the Northern Regional Research Laboratory, Peoria, Illinois.

Supported in part by a grant from the Robert A. Welch Foundation.

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[Received March 22, 1965—Accepted September 2, 1965]