THE METABOLISM OF C\(^{14}\)-GLYCEROL IN THE INTACT RAT*

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Considerably more work has been carried out on the formation of carbohydrate from glycerol than on its incorporation into lipides. Thus, conversion of glycerol to glucose (1–4) and to glycogen (5–7) has been repeatedly studied \textit{in vivo} and \textit{in vitro} (6). More recently there has been work on the incorporation of C\(^{14}\)-glycerol carbon (8–10) and deuterioglycerol (11) into liver glycogen.

With regard to lipides, Karnovsky and Gidez (12, 13) and Doerschuk (8) have demonstrated the incorporation of C\(^{14}\)-glycerol into the neutral fats (acetone-soluble lipides) and phosphatides (acetone-insoluble lipides) of the rat. Glycerol has been shown by Kennedy to double the synthesis of phosphatides by isolated washed rat liver mitochondria (14). Similarly Balmain et al. (15, 16), Bloch (17), and Karnovsky (18) have reported a stimulation of acetate incorporation into fatty acids by glycerol.

The present paper contains the results of a study on the formation of CO\(_2\), carbohydrate, and lipide from C\(^{14}\)-glycerol. In most of the data to be discussed the incorporation of glycerol into the various metabolic fractions has been followed with time. Preliminary results of this work have been reported previously (13).

EXPERIMENTAL

\textit{Synthesis of Radioactive Glycerol}—The C\(^{14}\)-glycerol used in these experiments was prepared by the method described by Gidez and Karnovsky (19).

Normal male rats of the Wistar strain were used. All the animals were fasted for 24 hours prior to their use. During the first 12 hours they were given 5 per cent glucose \textit{ad libitum}, and then water for the remainder of the time. Rats weighing 180 to 220 gm. were selected; their weights dropped to 160 to 200 gm. after the fast.

One series of experiments was carried out in a metabolism train. Respiratory CO\(_2\), expired during definite periods, was collected for 6 hours af-
ter intraperitoneal, intravenous, or intragastric administration of varying amounts of \(\alpha\) or \(\beta\)-\(\text{C}^{14}\)-glycerol dissolved in 1 ml. of physiological saline. At the end of 6 hours the animals were injected intraperitoneally with 20 mg. of Amytal. When they reached light surgical anesthesia they were killed by decapitation and exsanguination. Blood and various organs were collected for isolation of lipides and determination of their specific activities. The amount and specific activity of the \(\text{C}^{14}\text{O}_2\) expired in each period were also determined. The radioactivity of collected urine and feces was counted, and constituted usually about 1 to 5 per cent of the counts administered.

In a second series of experiments approximately 1 mM (92 mg.) of \(\alpha\)-\(\text{C}^{14}\)-glycerol dissolved in 1 ml. of saline was injected intraperitoneally into each of several rats. The animals were put in individual wire cages placed in well ventilated hoods, without access to food or water. Prior to the injection, 0.1 ml. of tail blood was collected for blood glucose determination. At the end of 15 or 30 minutes, 1, 2, 4, or 6 hours, animals were stunned by means of a blow at the base of the brain, and then killed by decapitation and exsanguination. Blood was collected in heparinized tubes; the livers were removed immediately and one-half was used for glycogen determination, while the remainder was saved for lipide isolation. The lipides were fractionated, hydrolyzed, and oxidized with periodate as described under "Methods."

In a third series of experiments 1 mM of \(\alpha\)-\(\text{C}^{14}\)-glycerol was administered to each of nine rats under the conditions above. Five animals were sacrificed after 1 hour, and the other four after 6 hours. The livers in each group were pooled and the total lipide extracted and fractionated. Pure tribenzoin and barium glycerophosphate were prepared from the neutral fat and phosphatide, respectively. A comparison was made of the activities of formaldimedon obtained after direct periodate oxidation of the aqueous hydrolysate fractions and those obtained after isolating the pure derivatives.

### Methods

**CO\(_2\) Analyses**—The \(\text{Na}_2\text{C}^{14}\text{O}_3\)-\(\text{NaOH}\) solutions from the metabolism train were diluted, and aliquots were used to determine CO\(_2\) manometrically by the method of Van Slyke and Neill (20).

**Glucose and Glycogen Analyses**—Blood glucose was determined by the method of Nelson (21) and Somogyi (22). Liver glycojen was isolated by the method of Good et al. (23). After hydrolysis of the glycogen, the glucose formed was determined as above (21, 22).

**Isolation of Lipides**—The method of Bloor (24), with modifications, was employed for the extraction of tissue lipides. The tissue was ground with
purified sand and alcohol, and the resulting mince was extracted with hot alcohol for 1 hour on a steam bath. After filtering, the tissue was reground and extracted twice with alcohol-ether (2:1) at 30°. The solutions were filtered and the combined filtrates were taken to dryness in vacuo on a water bath at 60°. The residue was extracted thrice with low boiling petroleum ether and filtered. This solution was washed with three successive portions of water (3 ml.) to remove any free glycerol that might have been present.

Lipides from plasma and whole blood were obtained by extraction with alcohol-ether (3:1) (25). Red blood cells were ground with MgSO₄ and extracted in a Soxhlet apparatus with chloroform.

The phosphatide content of the lipides was estimated by total phosphorus analyses (26, 27). The lipides were fractionated into acetone-soluble and acetone-insoluble components by the addition of 5 to 6 volumes of acetone and 4 to 6 drops of a saturated alcoholic solution of MgCl₂ to 1 to 3 ml. of a petroleum ether solution of the lipide (24). After standing in the cold for 1 hour or longer, the solution was centrifuged. The supernatant acetone was decanted, and the residue was washed with fresh acetone.

Phosphorus analyses were carried out on the acetone solutions. If the acetone-soluble material was found to contain more than 2 per cent phosphatides, a second fractionation was made. This process was repeated until there was 2 per cent or less phosphatide contamination.

Hydrolysis of Lipides

Neutral Fat—The neutral fat was saponified and the unsaponifiable fraction separated by the method of the Society of Public Analysts (28), modified to apply to samples of 30 to 50 mg. of fat. Alcoholic NaOH was used instead of KOH in cases in which periodate oxidation was later to be carried out. After removal of the unsaponifiable fraction, the aqueous solution was acidified with H₂SO₄, and the fatty acids were extracted with low boiling petroleum ether. The remaining solution was chilled, filtered, and treated with periodic acid (29), and the resulting formaldehyde steam-distilled. Formaldimeden was precipitated from the distillate by the addition of 2 ml. of 1 M sodium acetate and 1 ml. of an alcoholic solution of dimedon (5,5-dimethylcyclohexane-1,3-dione) (29). The precipitate was washed with water and recrystallized from aqueous alcohol.

Phosphatides—Samples of phosphatide (50 to 100 mg.) were refluxed for 2 hours with 1 ml. of 95 per cent alcohol and 5 ml. of 6 N HCl. Water was added and the fatty acids were extracted with four 15 ml. portions of low boiling petroleum ether. The aqueous fraction was chilled, filtered, and adjusted to pH 3, and then shaken with 4 ml. of Amberlite IR-100-H for 10 minutes. Preliminary tests had shown that this treatment effects the
complete removal of ethanolamine and serine from solution. The resulting solution showed a negative ninhydrin reaction on paper, and was filtered, made to pH 8, and treated with periodic acid. The formaldehyde was collected, precipitated, and recrystallized as above.

Preparation and Degradation of Lipide Glycerol Derivatives

In the third series of experiments described above, the neutral fats and phosphatides were hydrolyzed, tribenzoin was prepared from the aqueous fraction of the neutral fat hydrolysate, and barium glycerophosphate from the aqueous fraction of the phosphatide hydrolysate, after the ion exchange treatment described above. The methods of preparation of these derivatives were as used by Doerschuk (8). Tribenzoin was saponified, the benzoic acid separated by filtration after acidification and chilling, and the filtrate periodate oxidized. The formaldehyde representing the glycerol α-carbons was collected as above. The formic acid formed was oxidized with HgO, and the glycerol β-carbons obtained as barium carbonate (19). The barium glycerophosphate was burned and counted as BaCO₃. Samples were also converted to the sodium salt and were oxidized with periodate. Formaldehyde was collected as above.

Measurement of Radioactivity

All the samples to be counted were mounted on stainless steel disks which had a plating area of 1.54 sq. cm. A gas flow counter was used throughout the work (30).

Expired C¹⁴O₂—The expired C¹⁴O₂ was counted as BaC¹⁴O₃.

Glucose and Glycogen—To the solutions of glucose from the blood filtrate and the hydrolyzed glycogen, 10 mg. of carrier glucose were added, and the phenylosazones were precipitated. These osazones were recrystallized from 50 per cent ethanol before being mounted for counting.

Lipides—The lipides were dissolved in CHCl₃ and drops of the resulting solution were put on disks of lens paper placed on the plating surface of the planchet. In some instances (unsaponifiable material and fatty acids) ether or petroleum ether solutions were added to the planchet. Solvents were removed in vacuo until the planchets reached constant weight.

α-Carbons of Lipide Glycerol—The formaldimedon from the α-carbons of the glycerol moiety of the triglycerides and phosphatides was plated and counted as such (19).

Self-Absorption—All the counts were corrected with appropriate self-absorption factors. BaCO₃ factors determined by Robinson were used.¹ In the case of the glucosazones, lipides, and formaldimedon, the appropriate corrections were determined experimentally and applied.

¹ Robinson, C. V., unpublished data.
Correction Factors to Counts As BaCO₃—Under the counting conditions employed in these laboratories, good agreement between activities obtained by counting organic substances, or by converting these to BaCO₃, and counting the latter were obtained. Results of the determination of the various small factors involved will be reported elsewhere.

Fig. 1. Specific activities of expired CO₂ after administration of C¹⁴-glycerol, 112.2 mg. of α-C¹⁴-glycerol given intraperitoneally (●), intragastrically (○), and intravenously (△); 59.7 mg. of β-C¹⁴-glycerol given intragastrically (■), and 177.2 mg. of β-C¹⁴-glycerol given intraperitoneally (□). The specific activity of the α-labeled glycerol was 630,000 c.p.m. per mM, that of β-labeled glycerol 103,000 c.p.m. per mM. All the counts were normalized to 1 × 10⁵ c.p.m. administered.

RESULTS AND DISCUSSION

Oxidation of Glycerol to CO₂—Three specific factors were studied with regard to their influence on glycerol oxidation in vivo, as reflected by the expiration of C¹⁴O₂: (1) effect of the route of administration; (2) effect of the position of the label in the glycerol molecule; and (3) effect of the amount of glycerol administered.

Fig. 1 shows the changes in specific activity of expired CO₂ with time after the administration of α- or β-C¹⁴-glycerol, given intraperitoneally, intragastrically, or intravenously. There are apparently no significant differences in the patterns observed.

Fig. 2 demonstrates the relationship of the amount of glycerol oxidized to the amount administered. Where less than 30 mg. of glycerol was given,
the proportion converted to CO₂ was greater than at levels above 30 mg., and bore an inverse relationship to the amount administered. At levels above 30 mg. an approximately constant proportion of the administered dose appeared to be oxidized. The mode of administration or position of label did not appear to affect the cumulative expiration pattern.

*Synthesis of Blood Glucose and Liver Glycogen from C₁₄-Glycerol*—In Table I are data on the formation of glucose and glycogen from glycerol. Net increases of blood glucose were demonstrable only during the first 30 minutes. On the other hand there was no net formation of glycogen until after 15 minutes, and maximal synthesis was not reached until after 1 to 2 hours.

Fig. 3 shows the percentages of total glucose and glycogen carbon derived from glycerol carbon. These data were obtained from specific activity measurements of the glucose phenylazones derived from blood glucose and liver glycogen. Peak values for both carbohydrates occurred at 1 hour, one-third of the glucose and one-fifth of the glycogen carbon being derived from glycerol at that time. From the percentages in Fig. 3 and the chemical data in Table I, the values given in Fig. 7 for the percentages of administered glycerol converted to blood glucose and liver glycogen were obtained.

The values in Fig. 3 show the percentage of total carbohydrate carbons
derived from glycerol, but these data do not yield information on the percentage of *newly formed* glucose or glycogen arising from the administered glycerol. These figures may be calculated from the data of Table I and Fig. 3 for those times when there was *net synthesis* of glucose or glycogen. In the case of glucose, the data are for blood only, and not total extracellular glucose; for the calculations a blood volume of 4.5 ml. per 100 gm. of rat was used (31). In the case of glycogen, the initial levels were based on a fasting glycogen content of 0.046 per cent. The results are presented in Table II. The increases in glucose and glycogen are apparently greater

### Table I

*Synthesis of Blood Glucose and Liver Glycogen at Varying Times after Administration of Approximately 1 mM of C¹⁴-Glycerol*

<table>
<thead>
<tr>
<th>Time (hrs.)</th>
<th>No. of rats</th>
<th>Blood glucose concentration</th>
<th>Liver glycogen, final†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mg. per cent</td>
<td>mg. per cent</td>
</tr>
<tr>
<td>0.5</td>
<td>2‡</td>
<td>63 (±4.5)</td>
<td>94 (±14)</td>
</tr>
<tr>
<td>1.5</td>
<td>5</td>
<td>73 ± 14</td>
<td>100 ± 9</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
<td>68 ± 12</td>
<td>65 ± 6</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>66 ± 7</td>
<td>63 ± 4</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>75 ± 1.3</td>
<td>66 ± 3</td>
</tr>
<tr>
<td>6</td>
<td>2‡</td>
<td>47 (±14)</td>
<td>67 (± 9)</td>
</tr>
</tbody>
</table>

* An accurately determined amount of α-C¹⁴-glycerol (range 87.7 to 89.4 mg.) was administered; specific activity 1.085 × 10⁶ c.p.m. per mm.
† The initial fasting level was 0.046 ± 0.007 mg. per 100 mg. of liver.
‡ The figures in parentheses represent the average deviation from the mean. In all other cases, since sufficient animals were available, the standard error of the mean has been calculated.

than can be accounted for by the glycerol that is converted to these metabolites. However, introduction of glycerol into the animal could presumably cause formation of new glucose and glycogen by direct conversion of C¹⁴-glycerol to carbohydrate, or by a sparing action of oxidized C¹⁴-glycerol on C¹² intermediates, which may then be diverted for synthetic reactions.

**Incorporation of C¹⁴-Glycerol into Blood and Liver Lipides**—The lipides isolated from whole blood were counted. Fig. 4 shows these activities and the activities of the corresponding liver lipides. As will be seen in Table V, probably all of the activity of blood lipides resides in the plasma since there were negligible counts in the red blood cells.

**Incorporation of C¹⁴-Glycerol into Liver Phosphatides and Neutral Fat**—The specific activities of liver phosphatides and neutral fats counted as lipide are plotted in Fig. 5.
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The lipides that were counted to give the curves of Fig. 5, i.e. phosphatides and neutral fat, were, however, not homogeneous fractions. The several chemical species in each fraction might have incorporated C\textsuperscript{14} from C\textsuperscript{14}-glycerol at different rates. Determination of the changes in activity of a defined chemical entity would be of greater interest. Table III represents such data obtained for the glycerol moieties of the neutral fat and

![Graph showing percentages of blood glucose carbon and liver glycogen carbon derived from glycerol carbon.](http://www.jbc.org/)

Table III

<table>
<thead>
<tr>
<th>%</th>
<th>15 min.</th>
<th>30 min.</th>
<th>1 hr.</th>
<th>2 hrs.</th>
<th>4 hrs.</th>
<th>6 hrs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood glucose</td>
<td>30</td>
<td>24</td>
<td>18</td>
<td>12</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Liver glycogen</td>
<td>30</td>
<td>24</td>
<td>18</td>
<td>12</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>

Table II

\textit{Newly Formed Blood Glucose and Liver Glycogen Derived from Administered C\textsuperscript{14}-Glycerol}

The values are given in per cent.

<table>
<thead>
<tr>
<th>Time</th>
<th>Blood glucose</th>
<th>Liver glycogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 hr.</td>
<td>70</td>
<td>39</td>
</tr>
<tr>
<td>2 hrs.</td>
<td>100</td>
<td>23</td>
</tr>
<tr>
<td>4 hrs.</td>
<td>23</td>
<td>21</td>
</tr>
<tr>
<td>6 hrs.</td>
<td>21</td>
<td>15</td>
</tr>
</tbody>
</table>

phosphatide above. The activities of formaldehyde obtained by direct periodate oxidation of aqueous hydrolysate fractions agree reasonably well with results obtained after passing through the defined derivative. Statistical analysis of the results indicated that the differences were on the border line of significance. Table III indicates a rapid drop in the activity of neutral fat glycerol $\alpha$-carbons from 1 hour to 6 hours, and a much slower decline in the activity of phosphatide glycerol $\alpha$-carbons.

Determination of the activity of the $\beta$-carbon of neutral fat glycerol, carried out on the tribenzoin glycerol, showed randomization of activity into this carbon to the extent of 2 per cent of the total glycerol activity at
1 hour, and 5.5 per cent at 6 hours. Randomization is thus small, and, at least in 1 hour, of questionable significance (19). Doerschuk (9) obtained a value of 18 per cent for rat carcass neutral fat glycerol 24 hours after ad-

**Fig. 4.** Specific activities of liver lipides (O) and whole blood lipides (●) after administration of approximately 1 mm of α-C\textsuperscript{14}-glycerol. All the points are the means of three experiments, except at 15 minutes and 6 hours, when only two experiments were carried out. Other conditions were as in Table I. All the results were normalized to 1 × 10\textsuperscript{8} c.p.m. administered.

**Fig. 5.** Specific activities of liver phosphatides (●) and neutral fat (O), after administration of approximately 1 mm of α-C\textsuperscript{14}-glycerol. The number of animals in each group and the other conditions were as in Table I. All the counts were normalized to 1 × 10\textsuperscript{8} c.p.m. administered. Assumed molecular weight for phosphatide is 744; for neutral fat 840.
administration of α-C¹⁴-glycerol, and it has been shown (32) that 12 to 14 per cent of the activity of glycerol of the hen's egg resides in the β-carbon, after administration of acetate-1-C¹⁴.

**Table III**

*Specific Activities of Formaldehyde Obtained on Periodate Oxidation of Hydrolysates and Pure Glycerol Derivatives*

85.2 mg. of glycerol, specific activity 1,443,333 c.p.m. per mM administered to each rat; specific activity (c.p.m. per mM).*

<table>
<thead>
<tr>
<th></th>
<th>Neutral fat</th>
<th>Phosphatide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 hr.</td>
<td>6 hrs.</td>
</tr>
<tr>
<td>Formaldehyde, after</td>
<td></td>
<td></td>
</tr>
<tr>
<td>direct periodate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>oxidation.............</td>
<td>118,100 ± 610</td>
<td>31,200 ± 294</td>
</tr>
<tr>
<td>α-Carbon of tribenzoin glycerol†</td>
<td>114,000 ± 1640</td>
<td>27,500 ± 700</td>
</tr>
<tr>
<td>α-Carbon of Ba-glycerophosphate‡</td>
<td>33,800 ± 582</td>
<td>24,250 ± 1715</td>
</tr>
</tbody>
</table>

* The values are the means of three to five planchets. The standard error of the mean is given. All counts were normalized to 1 × 10⁶ c.p.m. administered.
† Melting point and mixed melting point with authentic tribenzoin 71.5-72°.
‡ Per cent carbon = 11.4 and 11.8; theory 11.7 per cent.

**Fig. 6.** Specific activities of the formaldehyde obtained on direct periodate oxidation of hydrolysates of liver phosphatides (●) and triglycerides (○). All the conditions were as in Table I. The counts were normalized to 1 × 10⁶ c.p.m. administered.
An attempt was made to calculate the randomization of activity in the barium glycerophosphate from the phosphatide by using the values for the whole molecule, obtained by combustion, and those of the α-carbons. The differences fell within the counting error, and no figure could be assigned.

**Table IV**

*Specific Activity of Liver Fatty Acids and Unsaponifiable Fractions after Administration of Approximately 1 mm α-C14-Glycerol*

The conditions are the same as in Table I.

<table>
<thead>
<tr>
<th>Time (hrs.)</th>
<th>Neutral fat</th>
<th>Phosphatide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unsaponifiable fractions</td>
<td>Fatty acids</td>
</tr>
<tr>
<td></td>
<td>c.p.m. per mg.</td>
<td>c.p.m. per mg.</td>
</tr>
<tr>
<td>½</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>1</td>
<td>16.5</td>
<td>9.3</td>
</tr>
<tr>
<td>2</td>
<td>2.1</td>
<td>8.8</td>
</tr>
<tr>
<td>3</td>
<td>1.8</td>
<td>2.6</td>
</tr>
<tr>
<td>4</td>
<td>3.3</td>
<td>2.6</td>
</tr>
<tr>
<td>6</td>
<td>1.7</td>
<td>0.9</td>
</tr>
</tbody>
</table>

**Table V**

*Incorporation of C14 into Lipides of Liver and Extrahepatic Tissues 6 Hours after Administration of C14-Glycerol*

In all cases the counts have been normalized to $1 \times 10^6$ counts administered, and liver lipide then arbitrarily set at 100.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Lipide</th>
<th>Given counts incorporated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>c.p.m. per mg.</td>
<td>per cent</td>
</tr>
<tr>
<td>Liver</td>
<td>100</td>
<td>5.9</td>
</tr>
<tr>
<td>Brain</td>
<td>13</td>
<td>0.26</td>
</tr>
<tr>
<td>Kidney</td>
<td>13</td>
<td>0.16</td>
</tr>
<tr>
<td>Spleen</td>
<td>12</td>
<td>0.06</td>
</tr>
<tr>
<td>Whole blood</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Heart</td>
<td>10</td>
<td>0.07</td>
</tr>
<tr>
<td>Lung</td>
<td>9</td>
<td>0.09</td>
</tr>
<tr>
<td>Stomach</td>
<td>6</td>
<td>0.05</td>
</tr>
<tr>
<td>Mesenteric fat</td>
<td>3</td>
<td>0.09</td>
</tr>
<tr>
<td>Testes</td>
<td>3</td>
<td>0.07</td>
</tr>
<tr>
<td>Small intestine</td>
<td>2</td>
<td>0.04</td>
</tr>
<tr>
<td>Depot fat</td>
<td>1</td>
<td>0.16</td>
</tr>
<tr>
<td>Pancreas</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>0.95</strong></td>
</tr>
</tbody>
</table>
The randomization is, however, of the same order as that in the neutral fat glycerol.

The data of Table III and the low degree of randomization suggest that Fig. 6 represents a reliable picture of changes in neutral fat and phosphatide glycerol activities. At the time of peak incorporation, about 20 per cent of triglyceride and 7 per cent of phosphatide glycerol α-carbons were derived from the α-carbons of administered glycerol. The fact that the curves of Fig. 6 do not satisfy the precursor-product criteria of Zilversmit et al. (33) might indicate that phosphatide glycerol and neutral fat glycerol are independently derived from the administered free C^{14}-glycerol (cf. (34)).

The fatty acids and unsaponifiable material of the liver lipides were also counted. The data appear in Table IV. There appeared to be maximal activity in the three fractions at 30 minutes. The specific activities of the neutral fat fatty acids were slightly higher than the activities of the phosphatide fatty acids, a finding in keeping with the results of Pihl and Bloch (35).

**Incorporation of C^{14}-Glycerol into Extrahepatic Lipides**—Table V shows the specific activities of lipides from various tissues. In lipides from extrahepatic tissues the phosphatide fractions were 3 to 5 times as active as the neutral fats.
Radioactivity Balance

In the case of one rat killed 6 hours after injection of C\textsuperscript{14}-glycerol, an attempt was made to strike a radioactivity balance, even though the experiment had not been specifically designed for the purpose. The results appear in Fig. 7. It will be noted that approximately 80 per cent of the activity was recovered, even though the "water-soluble fraction" of the liver and other organs (containing amino acids, carbohydrate intermediates, etc.) was not obtained, since these organs were used for isolation of glycogen and lipides. This fraction probably constituted an important percentage of counts, as did the same fraction obtained from the carcass. Fig. 7 also serves to summarize the disposition of radioglycerol carbon in different fractions at various time intervals after administration.

SUMMARY

1. The metabolism of C\textsuperscript{14}-glycerol in the intact rat has been studied with the aid of \(\alpha\)-C\textsuperscript{14}- and \(\beta\)-C\textsuperscript{14}-glycerol.
2. Insignificant differences were found when the glycerol was given intraperitoneally, intragastrically, or intravenously.
3. From 30 to 238 mg. of glycerol administered, an approximately constant proportion was converted to CO\textsubscript{2}.
4. As a result of glycerol administration, there were net syntheses of blood glucose and liver glycogen. 70 to 100 per cent of the new glucose was derived directly from glycerol, whereas only 15 to 39 per cent of the glycogen was newly formed from glycerol.
5. The specific activity-time curve of blood lipides paralleled that of liver lipides; however, the C\textsuperscript{14} content of the latter lipides was several times as great.
6. Liver triglyceride glycerol rapidly reached a specific activity about 3 times that of phosphatide glycerol, and declined in activity much more rapidly than the latter. Randomization of activity was low.
7. Activity was found in the lipides of most tissues examined. It is of interest that brain lipides were rather active.

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