Short Communications

On the Mechanism of the Intestinal Fat Absorption
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Two different hypotheses\(^1,2\) and much conflicting results\(^3,5\) have been published on the intestinal fat absorption\(^3,4\).

We have studied this process in cats with the aid of C-14 labelled stearic acid.

The material listed in Table 1 (A–C) was given to cats by stomach intubation. One hour later the thoracic duct was cannulated under chloralose anaesthesia and the lymph collected during 5–11 hours in a flask chilled in dry ice. The frozen lymph was lyophilized, the total lipids extracted and fractionated. The different fractions were then saponified and the activity of the fatty acids determined after wet combustion.

In these experiments only 3–12 per cent of the lipids fed were recovered in the lymph, apparently due to the condition of the animal after the operation, most of the fat remaining in the intestine.

After feeding equivalent amounts of glycerides (B) or of free fatty acids alone (C) the collected lymph was pronounced milky for several hours. Fractionation showed that in all cases the composition of the total lipids was approximately the same, the glyceride fatty acids constituting 70–80 per cent of the total fatty acids.

The high specific activities of the glyceride fatty acids leave no doubt that the free fatty acids fed in case C had been

<table>
<thead>
<tr>
<th>Form of fat administered</th>
<th>Collection time in hours</th>
<th>Lymph in g</th>
<th>% of fat fed recovered in lymph</th>
<th>Fatty acids recovered in lymph as</th>
<th>Phospholipid fatty acids</th>
<th>Glyceride fatty acids</th>
<th>Cholesterol fatty acids</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Weight %</td>
<td>Activity %</td>
<td>Weight %</td>
</tr>
<tr>
<td>A. 5 ml corn oil + 25 mg active stearic acid</td>
<td>10</td>
<td>42</td>
<td>12.8</td>
<td>13.7</td>
<td>58.5</td>
<td>79.8</td>
<td>83.5</td>
</tr>
<tr>
<td>B. 5 ml corn oil + 75 mg active glyceride</td>
<td>5</td>
<td>24</td>
<td>3.2</td>
<td>16.1</td>
<td>37.8</td>
<td>76.0</td>
<td>73.5</td>
</tr>
<tr>
<td>5 ml hydrolyzed corn oil + 25 mg active stearic acid</td>
<td>11</td>
<td>112</td>
<td>8.1</td>
<td>16.2</td>
<td>40.0</td>
<td>78.5</td>
<td>65.0</td>
</tr>
</tbody>
</table>

Table 1. The distribution of C\(^14\)-labelled stearic acid in lymph fatty acids after peroral administration to cats. Activity %, specific activity in % of specific activity of administered fatty acids.
used for the synthesis of the lymph glycerides as indicated by earlier work by Munk, Ivy and Tidwell, and in contradistinction to Frazer’s work.

Frazer estimates that 10–30 per cent of the fatty acids of fed triglycerides are set free by lipolysis in the intestinal lumen. Apparently he assumes that these acids are absorbed via the portal vein and that resynthesis plays a minor rôle. From our results (A) it is evident that these free fatty acids are incorporated mainly into triglycerides and to a smaller extent into phospholipids in the intestinal mucosa and transported via the thoracic duct into the systemic circulation.

The phospholipids show another picture: Apparently the phospholipids in the lymph are diluted more than the glycerides by the phospholipids coming from other organs than the intestines. The great difference between A and C requires special comments: When only free fatty acid was given C, the fatty acids in the phospholipids had 15 per cent of the activity of the acids fed. When inactive glyceride was fed together with a small amount of free radioactive acid in amounts such that total hydrolysis would give a mixture of the same composition as in case C, the phospholipid fatty acids, however, are 4 times as active in case A then in C. A possible explanation for this is that only a partial hydrolysis of the inactive triglyceride has taken place, the isotopic acid thus becoming less diluted so that the acid available for phospholipid synthesis had a higher specific activity. That can thus be taken as an experimental support of Frazer’s hypothesis of partial hydrolysis, but we do not wish to place much weight on these findings until we have obtained more results with better recoveries of the fat fed.

Recent work by Chaikoff with intravenous administration of fat emulsions has shown that in fasting dogs the liver is the main supplier of blood phospholipids. Under these conditions the intestine only took up triglycerides from the blood for its own metabolic use and did not contribute significantly to the blood phospholipids.

The results reported here and other work in this laboratory show that during intestinal fat absorption the intestinal wall supplies a quantitatively important part of the blood phospholipids via the thoracic duct when feeding either glyceride or free fatty acids.

The work with radioactive phosphate (Chaikoff 1948) seems to show that the entire fat absorption does not pass via phospholipins in the sense that all the absorbed glycerine or mono- and diglycerides are phosphorylated. Another possibility, suggested by our results, is that the absorbed fatty acids are incorporated into phospholipids and then used for the resynthesis of the glycerides from mono- and diglycerides possibly by some transacylation mechanism. However, the quantitative aspects of these possibilities remain to be elucidated.

A full report will appear in this journal.

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