activity be detected though the unfractionated proteins to start with had a slight activity of the order reported in literature. 6.


Table 1. Acetylation of carnitine by particles and by particle-free supernatants from different rat organs. The organs were homogenized in 10 volumes 10 % sucrose containing 0.005 M Tri-a-buffer (pH 7.4) and 0.005 M EDTA. The particle fraction was isolated with centrifugation at 25 000 \( \times g \) for 45 min. Acetyl-CoA was generated in the incubation mixture by means of acetylphosphate, 20 \( \mu \)moles; GSH, 2.5 \( \mu \)moles; CoA, 0.15 \( \mu \)moles, and phosphotransacetylase, 50 \( \mu l \) (Transacytase purchased from Nutritional Biochemical Co.). Other additions: Tri-a-maleate-buffer (pH 7.5), 20 \( \mu \)moles, DL-carnitine, 10 \( \mu \)moles; palmitoylcarnitine, 1 \( \mu \) mole; potassium chloride, 30–40 \( \mu \)moles and particles or particle-free supernatant derived from 2.5–10 mg of fresh tissue. Total incubation volume was 1 ml. The acetylcar- nitine formed was assayed according to Friedman and Fraenkel. 7. The values in the table represent \( \mu \)moles acetyl carnitine formed per 10 mg of tissue per hour at 30°.

<table>
<thead>
<tr>
<th>Organ</th>
<th>Particles</th>
<th>Particle-free supernatant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kidney</td>
<td>0.62</td>
<td>0.05</td>
</tr>
<tr>
<td>Liver</td>
<td>0.38</td>
<td>0.14</td>
</tr>
<tr>
<td>Heart</td>
<td>2.65</td>
<td>0.16</td>
</tr>
<tr>
<td>Muscle</td>
<td>0.70</td>
<td>0.03</td>
</tr>
<tr>
<td>Brain</td>
<td>0.34</td>
<td>0.00</td>
</tr>
</tbody>
</table>

The Organ and Cellular Distribution of Acetyl carnitine-CoA Acetyltransferase

Kaae R. Norum

Institute of Clinical Biochemistry, Rikshospitalet, University of Oslo, Oslo, Norway

When mitochondria are incubated with carnitine and pyruvate, acetyl carnitine is formed. In the presence of catalytic amounts of succinate, the acetyl group of acetyl carnitine is completely oxidized by mitochondria. Acetyl-CoA is the donor of the acetyl group in the acetylation of carnitine (Table 1).

Because of the impermeability of the mitochondrial membrane, acetyl-CoA formed in the mitochondria most likely cannot leave these particles. We suggest therefore that carnitine acts as a carrier of activated acetyl groups between the mitochondrial and the extramitochondrial compartments of the cell. This hypothesis requires the presence of acetyl carnitine-CoA acetyl transferase both intra- and extramitochondrially.

Table 1 shows that this enzyme indeed is found in the particle-free supernatant, but apparently in smaller amounts than in the mitochondria. This low activity in the supernatant is possibly due to the presence of inhibitory factors or to rapid inactivation of the enzyme.


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