CHARACTERISTICS OF THE LIPID METABOLISM IN RATS WITH EXPERIMENTAL ANTHYROIDISM AND HYPOTHYROIDISM

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Changes in lipid metabolism of rats with hypothyroidism (feeding with Mercazolyl) and athyroidism (thyroidectomy) are characterized by a decrease in mobilization of fat from the fat depots, an increase in the formation and output of β -lipoproteins, and a decrease in the autolipolytic activity of the liver.

In previous investigations [1, 4] the writer found certain disturbances of lipid metabolism in rats with experimental thyrotoxicosis. In the present investigation, indices of lipid metabolism were accordingly studied in animals with a deficiency of thyroid hormones.

EXPERIMENTAL METHOD

Athyroidism was induced in rats by total thyroidectomy (group 1) and hypothyroidism by inhibition of thyroid function by feeding with the thyrostatic drug Mercazolyl in a dose of 0.0015 g daily for 45-50 days (group 2). The initial weight of the animals of group 1 was 110-140 g, and of group 2 50-60 g. The animals of group 1 were used in the experiment 20 days after the operation. Control animals were examined at the same time. The rats of group 2 were used in the experiment after growth had ceased over a period of two weeks.

The following were investigated in all the experimental and control animals: a) the state of fat mobilization from adipose tissue, as judged from the lipolytic activity of the adipose tissue and the content of higher free nonesterified fatty acids (NEFA) [5], the total serum lipids, and the lipids of the liver (gravimetrically); b) the state of removal of fat from the liver, as reflected by the concentrations of β -lipoproteins and phospholipids [3, 6] in the liver and in the blood serum, and c) autolipolysis of the liver, i.e., the hydrolysis of triglycerides in the liver with liberation of higher free NEFA.

EXPERIMENTAL RESULTS

The total content of lipids in the liver of the thyroidectomized rats and also of the rats fed with Mercazolyl was indistinguishable from their total content in the control animals (Table 1).

Mobilization of fat from the adipose tissue of the animals with athyroidism and hypothyroidism was sharply reduced (both the lipolytic activity of the adipose tissue and the serum NEFA concentration were lowered) compared with the animals of the control group. The intensity of removal of fat from the liver also was changed in the experimental animals. For instance, the content of β -lipoproteins in the liver tissue and blood serum of the thyroidectomized animals and of the animals receiving Mercazolyl were significantly higher than in the animals of the control groups. The level of phospholipids in the liver and in the blood serum of

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TABLE 1. Indices of Lipid Metabolism in Control and Thyroidectomized Animals and in Animals Receiving Mercazolyl ($M\pm m$)

Serum Liver phos- Autolipol- phospholip- pholipids ysis of liver ids (in mg %) in μeq/ml/	(8	$\begin{array}{c} 5.88 \pm 0.04 \\ n = 10 \\ 4.557 \pm 0.248 \\ P < 0.001 \\ n = 18 \\ 5.16 \pm 0.028 \\ n = 20 \\ n = 20 \\ n = 3.8 \pm 0.14 \\ P_1 < 0.001 \\ n = 35 \end{array}$
Liver phos-pholipids (in mg %)		$\begin{array}{c} 4.05\pm0.22\\ n=10\\ 4.16\pm0.2\\ P>0.5\\ n=25\\ 4.0\pm0.38\\ n=26\\ 3.85\pm0.43\\ P>0.5\\ n=35\\ n=35\\ n=35\\ n=35\\ \end{array}$
Serum phospholip- ids	in g %	$ \begin{array}{c} 78,4\pm 0,028 \\ n=12 \\ 85,65\pm 0,92 \\ P<0,001 \\ n=20 \\ n=30 $
Liver 8 - lipopro- teins		$\begin{array}{c} 1.02\pm0.028\\ n=12\\ 1.288\pm0.04\\ p=0.001\\ n=20\\ 0.938\pm0.038\\ n=26\\ 1.324\pm0.008\\ P_1<0.001\\ n=20\\ \end{array}$
Serum β - liver β - Serum lipids lipopro- teins teins	in mg %	$78,4\pm0,028$ $n=12$ $85,65\pm0,92$ $P<0,001$ $n=20$ $70,3\pm0,66$ $n=26$ $92,7\pm0,52$ $P_1<0,001$ $n=20$
Serum lipids	in m	$220, 4 \pm 0, 840$ $n = 12$ $234, 5 \pm 5, 7$ $p = 0$ $231, 5 \pm 1, 8$ $n = 10$ $231, 5 \pm 1, 8$ $n = 12$ $266 \pm 9, 7$ $P_1 < 0, 0.2$ $n = 12$
	in meq/m1/g	0.94 ± 0.058 n=10 0.440 ± 0.05 0.440 ± 0.01 n=19 0.812 ± 0.812 n=24 0.524 ± 0.053 $P_1<0.001$ n=20
Lipolytic Lipolytic activity of adipose ting %)	in med	$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$
Liver lipids (in g %)		$ \begin{array}{c} 14.0\pm0.96 \\ n = 10 \\ 13.75\pm1.02 \\ P>0.5 \\ n = 14 \\ 14.5\pm0.726 \\ n = 14 \\ 15.2\pm0.770 \\ n = 12 \\ 15.2\pm0.770 \\ n = 12 \\ \end{array} $
Group of animals		Group 1 controlthyroidectomized animals

Note: Significance of differences P and P_1 calculated relative to the corresponding control group; n denotes number of animals.

the animals with hypothyroidism and athyroidism was not significantly changed by comparison with the control. The ability of the liver to hydrolyze triglycerides was sharply reduced in the animals receiving Mercazolyl, and to a lesser degree in the thyroidectomized animals. The investigations thus showed that in animals with athyroidism and hypothyroidism changes in the lipid metabolism were mainly opposite to the changes in thyrotoxicosis and were characterized principally by a decrease in the mobilization of fat from fat depots, an increase in the formation and output of β -lipoproteins, and a decrease in the autolipolytic activity of the liver.

LITERATURE CITED

- 1. L. M. Gol'ber and A. V. Negovskaya, Probl. Éndokrinol., No. 1, 67 (1970).
- 2. S. M. Leites and Chou-Su, Vopr. Med. Khimii, No. 3, 289 (1962).
- 3. M. G. Ledyina, Lab. Delo, No. 3, 13 (1960).
- 4. A. V. Negovskaya, Byull. Éksperim. Biol. i Med., No. 6, 58 (1969).
- 5. W. C. Duncombe, J. Biochem., <u>88</u>, 7 (1963).
- 6. C. H. Fiske and J. Subbarow, J. Biol. Chem., 66, 375 (1925).