INDICES OF LIPID METABOLISM IN EXPERIMENTAL THYROTOXICOSIS

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The results of investigation of some indices of lipid metabolism in experimental thyrotoxicosis are described. Changes in most of these indices are shown to be reversible.

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An excess of thyroid hormones lowers the glycogen content of the liver and thus stimulates mobilization of fat from its depots and its subsequent deposition in the liver. This causes depression of the main hepatic functions and, in severe cases, fatty degeneration of the liver.

The object of the present investigation was to study the dynamics of changes in some indices of lipid metabolism in the blood serum of animals during development of thyrotoxicosis and also in the recovery period, after discontinuing the action of the toxic agent.

EXPERIMENTAL METHOD

Experiments were carried out on 250 male rats weighing 200-220 g, receiving the same diet and kept under identical conditions. The animals were divided into four groups: 1) control, 2) rats receiving thyroid extract for 15 days, 3) rats receiving thyroid extract for 30 days, and 4) rats receiving thyroid extract for 30 days and investigated one month after the end of its administration. Thyroid was given by mouth, with an initial dose of 0.2 g and a final dose of 0.7 g daily. The development of thyrotoxicosis was assessed from the characteristic appearance and behavior of the animals, changes in their body weight, and their serum concentration of protein-bound iodine (PBI).

The total lipid content in the serum of the experimental and control animals was determined by Searsy's method [6], the β-lipoprotein concentration by Ledvina's turbidimetric method [4], phospholipids by the method of Fiske and Subbarow [3], ketone bodies by the Engfeld–Pinkussen iodometric method as modified by Leites and Odinov [2], and nonesterified fatty acids (NEFA) by Dole's colorimetric method [5].

EXPERIMENTAL RESULTS

As Table 1 shows, the serum lipid concentration in animals with thyrotoxicosis for 15 days (230.4± 0.09 mg%) and 30 days (221.1±0.4 mg%) was slightly reduced below that in the control animals (237.98± 0.35 mg%). The serum β-lipoprotein level fell sharply during development of thyrotoxicosis. Its mean value in the control animals was 79.61±0.73 mg%, falling to 45.06±0.72 mg% in animals with thyrotoxicosis for 15 days and to 25.35±1.02 mg% in those with thyrotoxicosis for 30 days.

The serum phospholipid concentration in animals receiving thyroid for 15 days (142.4±4.8 mg%) was not significantly different from that in the controls (141.9±0.85 mg%), but in animals with thyrotoxicosis for 30 days it was sharply reduced below the control level, with a mean value of 75.8±2.53 mg%.

The serum NEFA concentration of the experimental animals showed changes in the opposite direction. Their concentration after thyroid feeding for 15 days increased from 0.78±0.05 (in the controls) to 1.04± 0.12 meq/ml, while in rats with thyrotoxicosis for 30 days the increase was more marked still, to 1.35± 0.02 meq/ml. Finally, marked changes occurred in the blood concentration of ketone bodies in rats receiving thyroid for 30 days, rising from 6.09±0.16 (controls) to 18.78±1.26 mg%, although the concentration

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TABLE 1. Serum Concentrations of Total Lipids, β-Lipoproteins, Phospholipids, NEFA, and Ketone Bodies in Experimental Thyrotoxicosis (M±m)

Group of animals	Total serum lipids (in mg%)	Serum β-lipoproteins (in mg%)	Serum phospholipids (in mg%)	NEFA (in meq/ml)	Ketone bodies (in mg%)
Control (1)	237,97±0,79 $n=14$	79,61±2.1 $n=18$	141,9±15,3 $n=14$	0,78±0,05 $n=26$	6,09±0,32 $n=14$
Thyrotoxicosis for 15 days (2)	230,4±0,07 $n=15$ $P<0,001$	45,06±2,4 $n=17$ $P<0,001$	142,4±21,2 $n=17$ $P>0,5$	1,04±0,12 $n=30$ $P<0,02$	7,52±0,82 $n=14$ $P>0,1$
Thyrotoxicosis for 30 days (3)	221,4±1,2 $n=17$ $P<0,001$	25,35±1,8 $n=17$ $P<0,001$	75,8±3,4 $n=20$ $P<0,001$	1,35±0,1 $n=19$ $P<0,001$	18,78±2,5 $n=12$ $P<0,001$
Recovery (4)	242,94±1,1 $n=10$ $P<0,01$	63,48±2,6 $n=11$ $P<0,1$	128,77±4,5 $n=9$ $P>0,5$	0,96±0,066 $n=9$ $P<0,02$	5,19±0,54 $n=7$ $P=0,05$

of ketone bodies after administration of thyroid for 15 days (7.52±0.28 mg%) differed only slightly from the control.

Since the liver is the principal source of β-lipoproteins and phospholipids of the blood serum, a decrease in the serum concentration of these compounds is evidence of disturbance either of their formation or of their elimination from the liver. Which of these mechanisms is responsible can be discovered by determining these compounds in the liver tissue. In the case of NEFA and ketone bodies in the blood, results of their investigation showed that an excess of thyroid hormones stimulated both the elimination of fat from the tissue fat depots and also its oxidation in the liver.

A special series of investigations was carried out to determine the serum concentrations of these compounds in rats 30 days after the end of administration of thyroid for one month.

The results of this series of experiments (Table 1) show that all these indices have returned partly to their normal level. It can thus be concluded that the changes in lipid metabolism during experimental thyrotoxicosis produced in these experiments are reversible in character. Similar results were obtained in this laboratory with respect to protein metabolism [1].

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