LIPOLYTIC ACTIVITY OF ADIPOSE TISSUE IN CHILDREN

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Today it may be considered as established that the mobilization of fat from adipose tissue takes place as a result of lipolysis of its triglycerides, followed by the entrance of free (nonesterified) fatty acids (NEFA) into the blood, with their subsequent use as energy material [1, 3, 10].

Insufficient light has been shed in the literature on the problem of the lipolytic activity of human adipose tissue. Engelberg [7] did not detect lipolytic activity in human adipose tissue, while Angervall [5], as well as Nestel and Hevel [9], have shown it. The cause of the discrepancy is evidently the fact that the authors used different methods for the determination of lipolytic activity: Engelberg used an extract of acetone powder of adipose tissue, Angervall used Tween-20 as a substrate, and Nestel and Hevel used an activated emulsion of coconut oil.

We investigated the lipolytic activity of children's adipose tissue according to a modified method of Gordon and Cherkes [8].

PROCEDURE

Ground pieces of adipose tissue weighing 100 mg were placed in 3 ml of incubation liquid (5% human serum albumin in Krebs ringer phosphate buffer, brought to pH 7.3-7.4 by the addition of a 1 N solution of sodium hydroxide). Before incubation, the NEFA content in 1 ml of liquid was determined, after which the sample was covered with foil and placed in a water bath in a Warburg apparatus at 37° for 150 min under constant stirring. At the end of the incubation the NEFA content in 1 ml of the incubated mixture was determined once more. The lipolytic activity was characterized by the difference between the NEFA content after incubation and that before incubation and was expressed in micro-equivalents of NEFA per gram of tissue. The NEFA content was determined by the method of Dole [6], permitting the calculation of the natural lipolytic activity of adipose tissue.

Adipose tissue in the groin region taken from children operated upon for inguinal hernia served as the object for the investigation; only one of the children was operated upon for a rupture of the abdominal linea alba, and the adipose tissue was taken from this region. The children had no other diseases. The ages of the children ranged from one-and-a-half to 13 years. Of the 19 examined, 15 were boys and four girls.

RESULTS

As is evident from the data presented in the table, lipolytic activity was detected in the adipose tissue of all the children in the range 1.4-4.0 μ -equiv/g (an average of 2.2±0.19 μ -equiv/g). These indexes were lower than the values we obtained using the same method on rats (4.85±0.59 μ -equiv/g) and came close to the indexes of lipolytic activity in rabbit adipose tissue (2±0.28 μ -equiv/g).

As was established in experiments on rats [1] the addition of glucose to the incubation medium inhibits lipolytic activity of adipose tissue. The same inhibition (an average of 50%) was detected upon the addition of glucose in a concentration of 100 mg % to an incubation medium with children's adipose tissue (see table).

Characteristics of Lipolytic Activity in Adipose Tissue of Children

| Observation No. | Name | Age . (in years) | Ht. (in cm) | Wt. (in kg) | Lipo- lysis | addition | absorption | Lipolysis after the ad- dition of adrenalin in a dose of 0.248/ml |
|---|---|--|---|--|--|-------------------------|------------|---|
| 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 | Vova S. Igor' I. Sergei M. Valya S. Vova A. Lena M. Vasya M. Misha Z. Ira S. Gena M. Misha U. Misha I. Sasha F. Sveta S. Sayar E. Vitya Sh. Vova A. Tolya K. Borya E. | 11/ ₂ 11/ ₂ 2 31/ ₂ 41/ ₂ 5 5 6 6 7 71/ ₂ 9 11 13 | 61 79,5 80,0 93 102 95 114 112 110 119 113 112 113 118 119 130 139 167 | 11,6 12,5 11,5 11,1 12,5 16,3 20,0 23,0 20,0 20,5 21,0 20,0 20,0 17,0 23,5 34,5 33,0 62,0 | 2,2 1,5 2,0 4,0 2,0 1,4 1,4 2,8 1,4 2,8 1,4 2,2 1,4 2,2 3,6 3,6 2,8 2,0 | 2,2 | 15 | 4,2 |
| Average $(M\pm m)$ | | | | | | $1,0\pm0,21$ P<0,001 | 19,0±3,33 | $ \begin{array}{c} 3,0\pm0,2 \\ P < 0,01 \end{array} $ |

Glucose absorption by adipose tissue varies over a rather wide range—from 5 to 47 mg % (an average of 19.0 ± 3.33 mg %). We were not able to detect any direct relationship between the degree of glucose absorption and its suppression of lipolytic activity, although in a number of observations (see Nos. 6, 9, 13, and 14 in table), a considerably more pronounced suppression of lipolysis was noted upon increased glucose absorption than in those cases in which this absorption was less pronounced. As is well known [2, 4], the addition of adrenalin directly to isolated rat adipose tissue activates its lipolysis and is accompanied by an increase in the NEFA yield in the incubation medium. The same is observed upon the addition of adrenalin in a dose of $0.2 \mu g$ per ml of medium to adipose tissue of children (see table): here its lipolytic activity is increased by an average of 36%.

Thus, isolated children's adipose tissue possesses lipolytic activity, i.e., the ability to discharge NEFA into the medium during incubation; the lipolytic activity of children's adipose tissue is inhibited by glucose and activated by adrenalin.

LITERATURE CITED

- 1. N. K. Davtyan, Byull. Éksper. Biol., No. 11 (1962), p. 63.
- 2. N. K. Davtyan, Probl. Éndokrinol., No. 6 (1963), p. 33.
- 3. S. M. Leites, Ter. Arkh., No. 6 (1963), p. 3.
- 4. S. M. Leites and Chou Hsu, Probl. Éndokrinol., No. 5 (1963), p. 30.
- 5. G. Angervall, Acta physiol. scand., 48 (1960), p. 71.
- 6. V. P. Dole, J. clin. Invest., 35 (1956), p. 150.
- 7. H. Engelberg, J. Lipid Res., 2 (1961), p. 169.
- 8. R. S. Gordon, A. Cherkes, et al., J. clin. Invest., 36 (1957), p. 810.
- 9. P. J. Nestel and R. J. Hevel, Proc. Soc. exp. Biol., 109, New York (1962), p. 985.
- 10. D. Steinberg and M. Von, Transactions of the Fifth International Biochemical Congress. Symposium 7. Lipid Biosynthesis [in Russian], Moscow (1962), p. 157.