ABNORMALITIES OF TISSUE LIPIDS

AORTA FATTY ACIDS IN THE NEWBORN PERIOD

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The phosphoglyceride fatty acids of the aorta were studied in a group of 18 newborn infants with different gestational ages. All the infants had died during the earliest hours of life, before feeding. The total lipids, free cholesterol and lipid phosphorus were quantified in the lipid extract of the homogenized aorta, and no changes in their concentration were found throughout the period studied. As for the fatty acids, the following findings contrasting with those previously reported in brain tissue were observed as the gestational age went up: an increase of the w6 fatty acids, specially of the 20:4w6 with a slowing down of its elongation, and a decrease of the 18:0 in ethanolamine phosphoglycerides. In choline phosphoglycerides the 18:2w6 increased as the gestational age progressed, whereas the 18:0 and 18:1w9 did not vary. The 22:4w6/22:5w6 index increased in both phosphoglycerides but more significantly in lecithin. The w3/w6 ratio only increased clearly in choline phosphoglycerides.

IN VIVO METABOLISM OF C^{14} -LABELLED PALMITIC ACID IN BROWN AND WHITE ADIPOSE TISSUE OF WELL-FED AND STARVED NEWBORN RABBITS. T. Heim, H. Schenk, H. Wagner, L. Winkler, E. Goetze, F. Varga. Dept. Paediat. Univ. Pécs, Hungary and Inst. Pathol. Physiol. Univ. Jena, GDR.

After injection of 20.10 CPM/100 g body weight C^{14} palmitic acid, the primary FFA pool was in 7 days old well-fed rabbits /Group I/ $9.57 \pm 2.12 \mu\text{mole}$, and in rabbits of the same age but previously starved for 48 hrs. at 20 °C /Group II/ 3.17 ± 0.41 and in those starved 72 hours at 35 °C /Group III/ 1.89 ± 0.50 . There was no significant difference in half time $t_{1/2}$ and turnover time, but turnover rate $\mu\text{mole FFA/min}$ was significantly diminished by starvation. /Group I/ 9.38 ± 0.72 , Group II. 3.44 ± 0.49 , Group III. 2.67 ± 0.60 /. Starvation at different ambient temperatures affected FFA phospholipid and glyceride pools $\mu\text{mole/100 g body weight}$ as well as the total flux of labelled fatty acid $\mu\text{mole/min}$ between the intracellular and extracellular lipid compartments of brown and white adipose tissue. In brown adipose tissue FFA flux was in Group I.: $0.315 \pm 0.031 \mu\text{mole/min}$, in Group II.: 0.226 ± 0.039 , in Group III.: 0.047 ± 0.008 . The corresponding figures for white adipose tissue in Group I.: $0.072 \pm 0.011 \mu\text{mole/min}$, in Group II.: 0.009 ± 0.001 , in Group III.: 0.014 ± 0.004 . Functions of the two adipose tissues will be discussed.

CHEMICAL AND CELLULAR DEVELOPMENT OF ADIPOSE TISSUE AFTER BIRTH. Douglas Gairdner, Addenbrooke's Hospital, Cambridge, England.

During the first months after birth, rapid changes are taking place in adipose tissue: 1, multiplication of fat cells; 2, enlargement of fat cells; 3, change in the fatty acid composition of triglyceride, the predominantly saturated fatty acids of the fetus being replaced by unsaturated. Some or all of these changes are influenced by the infant's diet, which may thus be responsible for long-term consequences (obesity, possibly atheroma); hence the importance of the subject.

These changes have been followed in a series of term and preterm infants by examining biopsy and post mortem samples of adipose tissue, using (a) microscopy for measuring cell size and number, (b) chromatography for fatty acid composition.

CHANGES IN ERYTHROCYTE STROMAL LIPIDS IN THE PREMATURE INFANT ACCORDING TO DIFFERENT DIETS.

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Some lipid compounds of the human erythrocyte stroma were studied in three groups of premature newborn infants fed different milk formulae. Two blood samples were obtained: immediately after birth and 20 days of feeding a given formula. Three milk formulae with a widely different linoleate content were used (A=6.08%, B=15.98%, C=0.6% of the total caloric intake). Free cholesterol which decreased after feeding A and B, did not vary after C, because of its low 18:2w6 concentration. In choline phosphoglycerides a clear sign of essential fatty acid deficiency was found in the infants fed C: a striking very significant increase of the triene/tetraene ratio (20:3w9 / 20:4w6) not only in plasma but also in red cell stroma. An increase of 18:1w9 was also found, accompanying a very low percentage of 18:2w6 in the group fed formula C. It is concluded that a content of acid linoleic in the infant diet of only 0.6% of the total caloric intake is essential fatty acid deficient.

PARTIAL LIPODYSTROPHY, R.J. West, A.S. Fosbrooke, J.K. Lloyd. Institute of Child Health, London, U.K.

Six children with partial lipodystrophy have been studied. Oral glucose tolerance was not grossly impaired but fasting serum insulin ($19.4 \pm 6.3 \mu\text{U/ml}$) was higher than normal, and hyperinsulinaemia after oral glucose (maximum $250 \mu\text{U/ml}$) was present in 3/6 children. Plasma growth hormone was normal; serum cortisol was low in 1/6 children. Fasting serum cholesterol, triglyceride, and plasma non-esterified fatty acids were normal. Fatty acid composition of adipose tissue triglyceride from non-dystrophic areas in 3/3 children showed an increased proportion of palmitoleic acid (8.8, 10.0, 11.0%) compared with normal ($5.8 \pm 1.5\%$). Plasma post-heparin lipolytic activity was normal in 3/4 children. Sweat production and sodium concentration did not differ between lipodystrophic and normal areas in 2/2 patients. Treatment with propranolol, in an attempt to inhibit sympathetic mediated lipolysis, had no beneficial effect in 2/2 children over a 4-6 month period. The results suggest that increased insulin resistance is present at an early stage of the disease, but do not provide evidence for sympathetic nervous system overactivity.

PREPARATION OF A URINARY LIPODYSTROPHIC FRACTION. I. Foss and O. Trygstad, Section of Endocrinology Institute of Pediatric Research, Oslo, Norway.

Urine from patients with generalized congenital lipodystrophy has been fractionated by precipitation of the protein with benzoic acid succeeded by Sephadex gel filtration. A fraction with mol.wt. in the range of 1,500 was found to have metabolic activity in intact rabbits and mice. In acute experiments there was observed an increase of serum triglycerides only. Following 20 days of injection of the lipodystrophic fraction (LDF) a lipodystrophic state was induced in the animals, with increase of BMR, and decrease of body weight in spite of a 40% increase of food consumption. The fat depots disappeared, blood glucose increased, and an insulin resistance was developed. Urine obtained from the injected rabbits induced similar changes in mice. Following injection of LDF in infantile rats there was observed a growth of the gonades, and a significant increase of serum somatomedin. In hypophysectomized rats there were observed no growth of the tibial width or of the gonades. It is concluded that the LDF probably contained hypothalamic releasing hormones, which also may be responsible for the development of the disease.