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Insulinemia and intraduodenal milk infusion.

Knowledge of intestinal stimulus to insulin release increase rapidly. However, the physiological role of the intestinein the glycoenergetic regulation is yet poorly understood. The consequences of intraduodenal milk infusion were therefore investigated. We performed duodenal intubation for diagnosis purpose in 11 children (1 to 10 years) without pancreatic insufficiency, abnormal jejunal histology or diabetes. Under gastric aspiration, milk (Nutramigen)was infused in the 1st part of duodenum at a constant rate of 4 ml/min/m2. Simultaneously the serum concentration of glucose, insulin, FFA and gastrin were determined. During the first 30 minutes we observed a sharp increase in insulinemia (from 5.5 µU/ml + 2.4 ($\bar{x}$ + SEM) at time 0 to 24.3 + 9.8 at 30 min) a small increase in glucose concentration (from 73.4mg/100 ml + 6.9 to 95.7 + 13.3), a small decrease in FFA (from 594 + 104 µeq/liter to 492 + 157). After 30 minutes a plateau was achieved for glucose (95 mg/100 ml) and for insulin (25 µU/ml). In contrast FFA concentration was low (230 µeq/l at 240 min). No change in gastrin concentration was observed. These results suggest that intraduodenal milk infusion stimulates insulin release. It appears that duodenum plays an important role in energetic homeostasis in normal children.

113 N. van der MEULEN, K. LIPP and R. SCHMIEDEBERG (Intr. by W. Schröter). Specific ^{3}(H) dexamethasone binding to a thymus cytosol receptor and inhibition of RNAsynthesis by glucocorticoids during postnatal development.

The thymus, a target organ for glucocorticoids, responds to the administration of these steroids with decreased utilization of glucose and cytolysis. The presence of specific receptor proteins within the cytoplasm and nucleus of isolated rat thymocytes is instrumental in accumulating and transporting the hormone into the nucleus as well as in the inhibition of RNAsynthesis. The ageassociated thymus involution is caused by catabolic effects of steroidhormones interferring directly or indirectly with nucleic acid synthesis. We considered the possibility that the amount of cytoplasmic glucocorticosteroid binding proteins represents a rate limiting factor for inducible inhibition of RNAsynthesis and thymus involution during postnatal development. Between 50-60 g body weight the relative and absolute weight of rat thymus shows maximal values. Thereafter a continuous ageassociated involution could be observed. The steroidhormone receptorcapacity changes in a striking similarity to the described organ weight curve. At certain developmental stages a very good correlation exists between thymus weight and both the concentration of specific receptorprotein and the ability of dexamethasone to inhibit transcription of thymus nuclei during postnatal development.

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Stimulation of thymocytes by T4 or T3 in vitro.

The stimulatory effect of thyroid hormone (T3, T4) on growth is clinically well known. Although a stimulation of tumor cell growth by T3 was found by Samuels, in vitro experiments of Whitefield and other authors failed to demonstrate an effect of T3 or T4 on cell proliferation. T4 has been shown by us to stimulate thymocytes (TC) proliferation in vivo.

Material and methods:TC and lymphocytes, isolated from male Wistar rats, were incubated 2-32 h; 10^{-4}-10^{-7}M T4 or 10^{-8}-10^{-10}M T3 were added. Proliferation was measured by H3-thymidine uptake (pulse labelling).

Results:T4 (10^{-7}M) as well as T3 at physiol. conc. ($5x10^{-9}$M) increase the proliferation of TC in cell culture ($p<0,01$). With T4, two peaks of H3-thymidine uptake were seen after 8 rsp. 24 h. These peaks were not abolished by preincubation of TC before adding T4. Cell proliferation rose with increasing conc. of T4 and fell off at high T4 conc. In contrast, max. T3 effect was seen already after 4 h incubation.

Conclusion:This in vitro effect of T4 and T3 on cell proliferation is interesting in view of the known stimulatory effect of T4 on growth.

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Galactose-1-phosphate uridyltransferase and galactokinase activity in fetus organs of different gestation ages.

The enzyme activities of uridyltransferase and galactokinase in liver, kidney, brain, lung, heart, muscle and spleen of human fetuses with gestation ages from 7 to 28 weeks have been examined. The activity was determined by the carbon C-14 method. The uridyltransferase activities expressed as nmoles UDP-galactose produced/min./mg protein increased with the increasing gestation ages in liver (7.9-33.0), lung (0.7-6.7), heart (0.6-6.0) and spleen (0.4-5.5), while relatively constant values are obtained during this period from brain (ca. 1.2), kidney (ca. 3.6) and muscle (ca. 2.0). The transferase activity in liver reached a maximum level just before birth, and then decreased slowly to plateau, i.e., the value of a 4 month old baby was 14.5, and that of a one and half year old baby was 8.5. The developmental characteristics of galactokinase in fetal organs were similar to uridyltransferase. In liver (0.17-3.80), lung (0.06-2.3) and heart (0.16-3.2) the activity increased with the increasing gestation ages. K_m of uridyltransferase for galactose-1-phosphate was 0.3 mM in fetus liver, and that of galactokinase for galactose, 0.27 mM.

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Comparative turnover rates of free fatty acids /FFA/ and free glycerol in blood of newborn rabbits.

Determination of tracer kinetic patterns of circulating FFA and free glycerol might be of value in the elucidation of their metabolic pathways and their quantitative contribution to the calorigenesis in the main heat producing organs and tissues of the newly born.

After i.v. injection of ^{14}C-1-palmitate or ^{14}C-1,3-glycerol /2o.10^{6} d.p.m./loo g bwt/ the turnover rate of plasma FFA and free glycerol was found 1o,2 and o,85 µmol/min/loo g bwt respectively in a thermoneutral environment of 35°C /T_a 35°C/. Exposure to cold /T_a 2o°C/ caused a decrease in FFA turnover /5,84 µmol/min/ and an increase in free glycerol turnover /2,45 µmol/min/. The plasma concentrations of both metabolites increased significantly during cold exposure /FFA from o,4o3+ o,o41 to o,518+o,o57; free glycerol from o,251+o,o21 to o,5o5+ o,o57/. These findings indicate that during cold-induced calorigenesis in the newborn rabbit substantial part of stored fatty acids are oxidized in brown adipose tissue itself, while the bulk of the concomitantly produced glycerol is being released into the circulation resulting in a ratio of FFA: glycerol turnover of 2,4 : 1. It practically means that in the cold during hydrolysis of adipose tissue triglycerides instead pf 3 only 2,4 moles of FFA entered and left the blood for each mole of glycerol.

117 J.Mestyán, K.Schultz, Gy. Soltész/ Department of Paediatrics,University Medical School,Pécs, Hungary/: Changes in blood glucose and plasma amino acids in response to glucagon infusion in small for gestational age infants.

The study was designed to show the changes in plasma amino acids in relation the response of blood glucose to glucagon infusion / 0.2 ug/kg/min for 4 hrs / in 7 non-hypoglycaemic /NHSGA/ and 7 hypoglycaemic /HSGA/ newborn infants. In the NHSGA group blood glucose increased from 66±12 mg% to 136±16 mg % by 120 minutes, and thereafter it declined rapidly. In HSGA infants it rose from 22-2 mg% to 69±13 mg % which was maintained during the test period. The unopposed glycaemic action of glucagon suggested a difference in the interaction of hormones between the two groups of neonates.This difference was also indicated by the observation, that while in the NHSGA group a progressive and significant decline occured in the majority of amino acids, in the HSGA infants neither the glucogenic, nor the branched chain amino acids were significantly affected by glucagon.This unresponsiveness raises the question whether the sustained rise in blood glucose entirely resulted from glycogenolysis, or it could also attributed to stimulated gluconeogenesis, It is not unrealistic to suppose that under the metabolic conditions associated with hypoglycaemia hepatic uptake and intrahepatic disposition of accumulated amino acids, for a period of few hrs,may not necessarily respond simultaneously to glucagon.