

89 LINDOLEIC ACID METABOLISM IN PARENTERALLY FED INFANTS
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The major essential fatty acid in Intralipid is linoleic acid (LIN; 18:2w6). The hepatic metabolism of LIN supplied with intravenous lipid emulsions is reflected by the composition of very low density lipoproteins (VLDL). We studied the essential fatty acid content of VLDL triglycerides (TG), cholesterol esters (CE) and phospholipids (PL) in 17 fullterm, appropriate for gestational age infants (birthweight 3126 ± 70g, gest. age 39.4 ± 0.3 weeks; MSE) during i.v. alimentation with glucose/amino acid solutions only (GL/A; age 6.5 ± 0.5 days) and after addition of Intralipid (GL/A/IL; age 13.2 ± 0.7 days). Results: Total lipid content of VLDL was not altered by transition from GL/A to GL/A/IL (39.4 ± 6.2 vs. 35.6 ± 5.7 mg/dl). The administration of Intralipid led to a pronounced increase of LIN in all lipid classes (cf. Table). Arachidonic acid (AA; 20:4w6), the major functional metabolite of LIN, was not changed in TG, but decreased markedly in CE and PL.

LINDOLEIC AND ARACHIDONIC ACID (% WT/WT, MESE) IN VLDL-LIPIDS
(x p < 0.05) TG-LIN TG-AA CE-LIN CE-AA PL-LIN PL-AA
GL/A 2.8 ± 0.5 1.6 ± 0.3 7.2 ± 0.9 6.5 ± 1.0 4.8 ± 0.8 9.9 ± 1.3
GL/A/IL 31.8 ± 2.8* 1.5 ± 0.2 22.1 ± 3.2* 3.6 ± 0.7* 14.1 ± 1.9* 5.3 ± 0.8*

Conclusions: 1. Linoleic acid administered with Intralipid is avidly incorporated into all lipid classes of VLDL. 2. The high linoleic acid load of the lipid emulsion results in a reduced content of LIN-metabolites, such as arachidonic acid, in VLDL-CE and -PL. 3. Both a competition of linoleic acid with its metabolites for incorporation into CE and PL as well as an inhibition of hepatic chain elongation and desaturation by the high LIN intake may be responsible for the reduction of AA.

90 CARNITINE SUPPLEMENTATION IN PREMATURE INFANTS
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The possible effects of carnitine supplementation on nitrogen metabolism were studied on ACA preterm infants (birthweight 980-1750g) maintained on mixed nutrition (50% pooled milk 50% formula daily). Started by various postnatal ages (mean 25.5 days) 15 infants received L-carnitine supplemented formula (600 nmol/ml over endogenous content) during 7 days, another 10 served as controls. Plasma carnitines increased whereas alanine (0.21 ± 0.02, 0.19 ± 0.03, 0.25 ± 0.02 mmol/l; day 0, 7, and 14, means ± SEM, p < 0.05) and glutamine (0.37 ± 0.06, 0.31 ± 0.05, 0.41 ± 0.05 mmol/l p < 0.05) decreased with a fall of urea level. Urinary urea (2.6 ± 0.23, 2.25 ± 0.18, 2.31 ± 0.21 mmol/kg/day, p < 0.05) and ammonia (1.07 ± 0.1, 0.87 ± 0.1, 1.4 ± 0.1 mmol/kg/day, p < 0.05) decreased suggesting lowered amino acid degradation. Surprisingly, 7 days after the supplementation, excretion of acylcarnitines remained high (5.9 ± 1.5, 13.1 ± 2.5, 10.3 ± 1.3 umol/day, p < 0.05) which was not seen for the free fraction.

91 BRANCHED CHAIN AMINO ACIDS AND PERINATAL NUTRITIVE STATE.
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A comparative study of plasma amino acids in the umbilical cord (aa)(totals and ratios: malnutrition indices) and clinical nutritive state by Rohrer's ratio (Rr) between well nourished premature (GI) and undernourished neonates born at term (GII). Patients groups: GI(n:30), GA:33.9(.3) (+/-SEM)wk.Wt:2084(.06)g and Rr:25.1(3.6)p; GII(n:14), GA:38.2(.3)wk.Wt:2328(.05)g,Rr:8.14(4.2)p. Blood sampling: .5 ml UVB was mixed with EDDA and centrifuged immediately. Sulfosalicylic acid was then used to separated proteins from plasma. The supernatant was either used immediately or stored at -20°C. Technical analysis: HPIECH and fluorimetric assay (Chromaspeck J 180). Statistical study: Student's and Welch's tests. (aa) concentrations are expressed in micromol/dl. (versus-; \$; p .05). RESULTS.- GII-GI: S:460(28)-436(21); E:136(7)-145(7); NE:323(24)-291(18); NE/E:2.4(.1)-2.0(.1); BCA:37(1.9)-46(2.4)\$; ALA/THR:1.9(.1)-1.3(.1)\$; GLY/VAL:2.2(.4)-1.2(.1)\$; ALA/LEU:5.0(.5)-2.9(.1)\$; ALA/BCA:1.6(.1)-0.9(.1)\$; and, PHE/TYR:1.2(.1)-1.3(.1). There are a close correlation between ALA:LEU (r:.72,p<.01) in GII but not in GI. We conclude that undernourished neonates born at term has strongly activated the alanine-glucose cycle. The BCA catabolized in muscle provide a major nitrogen source for biosynthesis of alanine which play and important role in energy metabolism.

92 THE METABOLIC TOLERANCE OF INTRAVENOUS AMINO ACIDS IN PRETERM INFANTS IN THE FIRST WEEK OF LIFE.
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42 infants gestation <32 weeks (28.6+2.16.M+SD) received supplementary parenteral nutrition. Birthweight 1099+388g (M+SD) (19 <1kg). 37 infants were ventilated on day 1 and 29 on day 6. Dextrose, electrolytes, amino acids and lipids were given to supplement expressed breast milk or formula. The total nitrogen intake and proportion provided by the amino acids were 0.09 (range 0.01-0.38)g/kg/d (65%) on day 1 and 0.3 (range 0.07-0.48)g/kg/d (66%) on day 6. The total calorie: nitrogen ratio on day 1 was 847 and this decreased to 264 on day 6. The reduction of body weight during the 6 days was 6%. Plasma albumin increased 11% from 22.4+6.4 (M+SD)g/L to 25.0+5.0g/L (p<0.05). Daily plasma, electrolytes, glucose, urea were all normal but the mean plasma phosphate fell to less than 1mmol/L on days 4-6. The mean plasma alkaline phosphatase of 209+87 (M+SD)IU/L on day 1 rose to 270+115IU/L by day 5 (p<0.01). The total bilirubin rose from 99.7+52 (M+SD)umoles/L on day 1 to peak at 130+49umoles on day 4 to fall to 104+55umoles/L on day 6. This was thought to represent 'normal' preterm hyperbilirubinaemia. Overall the metabolic tolerance of the preparation in these immature and sick infants was excellent.

93 THE PLASMA AMINOGRAMS OF VERY LOW BIRTHWEIGHT INFANTS ON SUPPLEMENTARY INTRAVENOUS AMINO ACIDS IN THE FIRST WEEK OF LIFE.
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MB233G: is an amino acid (aa) preparation formulated with reference to cord blood aa of the mid trimester foetus i.e. it has been designed specifically for newborn infants. In 1 year, 52 infants with birthweight <1500g were partially or wholly i.v. fed as full enteral feeds were not tolerated. 42 infants were evaluated (exclusions: 6 infants dying <6 days, 4 who received aa's for <5 days). 19 infants were <1000g. Parenteral nutrition was provided as dextrose, electrolytes, MB233G, and Intralipid by peripheral infusion. The i.v. aa load increased from 0.37 (range 0.06-2.1)g/Kg/d on day 1 to 1.31 (0.01-2.4)g/Kg/d on day 6 (Nitrogen intake 0.058 (0.01-0.32)g/Kg/d on day 1 to 0.20 (0.01-0.37)g/Kg/d on day 6). The enteral intake of expressed breast milk or formula contributed a further 0.03 (0-0.23)g nitrogen/Kg/d on day 1 to 0.10 (0-0.47)g/Kg/d on day 6. Plasma aminograms (L.K.B. 4400 aminoanalyser) on the 5th day (day 6) of the aa infusion showed cystine levels lower than the foetal cord range and aspartate levels higher. All other aa's fell within the normal reference range.

94 HIGH DENSITY LIPOPROTEIN AND ITS SUBFRACTIONS IN CHILDHOOD OBESITY.
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Fasting levels of plasma high density lipoprotein cholesterol (HDL-C) and subfractions and their relation to the degree of adiposity, plasma insulin (IRI) and age were studied in 79 control (C) and 93 obese (O) children. Mean (+SE) age and body fat content of C and O children were 11.3+0.3 vs 11.4+0.2 yr and 19.4+0.6 vs 37.8+0.5% respectively. Plasma IRI was measured by RIA, HDL-C and subfractions enzymatically using precipitation techniques. O children had high IRI (0:155+16, C:56+6 pmol/l), low HDL-C (0:1.03+0.03 C:1.31+0.04 mmol/l), HDL₂-C (0:0.24+0.02, C:0.57+0.03 mmol/l) levels and low HDL₂/HDL₃-C ratio (0:0.29+0.03 C:0.78+0.05, p<0.001) compared to C. HDL₂-C levels were not different in the two groups. Multiple regression analysis showed that the effect of age and IRI on HDL-C and subfractions was negligible, while body fat content was responsible for 34, 59 and 37% of the variation in HDL-C, HDL₂-C and HDL₂/HDL₃-C ratio respectively.