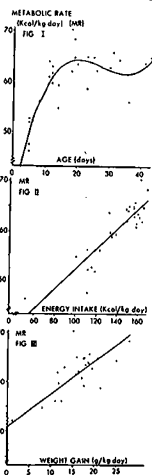


531 INFLUENCE OF AGE, ENERGY INTAKE, AND WEIGHT GAIN ON METABOLIC RATE IN THE VERY LOW BIRTHWEIGHT (VLBW) INFANT.

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We have evaluated the influence of postnatal age, energy intake (EI) and weight gain (Wt.G) on metabolic rate (MR), in 28 studies on 13 appropriate for gestational age formula fed (SMA 20/24) VLBW infants (mean±SE BWT:1155±39g; gest.age: 29.3±0.4 wks). Study age ranged from 5-43d, Wt.G from 0-28g/kg.d, energy intake from 52-169 Kcal/kg.d. MR measured by continuous open circuit indirect calorimetry ranged from 47-70 Kcal/kg.d. MR increased with age in the first 2 weeks (Fig.I; $r=0.85$). A similar relationship was found for energy intake and weight gain with increasing age. Multiple linear regression analysis shows that MR correlates with energy intake (Fig.II; $r=0.88$) and rate of weight gain (Fig.III; $r=0.86$). The interdependence of these 3 parameters ($MR=38+0.13EI+0.36Wt.G$; $r=0.93$) should be considered in the evaluation of energy metabolism under different clinical conditions or therapeutic and nutritional regimes. Changes in MR with postnatal age are modulated by increasing EI and Wt.G.



532 PHARMACOKINETICS OF SULFAPYRIDINE (SP) AND ACETYLSULFAPYRIDINE (ASP) IN PEDIATRIC PATIENTS WITH INFLAMMATORY BOWEL DISEASE (IBD).

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Sulfasalazine is widely used in treating IBD in children; the side effects are thought to be related to the serum concentration of SP and ASP. The pharmacokinetics of SP and ASP were studied in 15 prepubertal children with IBD. The patients were on maintenance sulfasalazine therapy, 30 to 70 mg/kg divided to 2 doses/day. Five patients were studied both during active disease and in remission. Serum samples were drawn at 0, 3, 6, 9 hours after the a.m. dose. SP and ASP concentrations were measured by high pressure liquid chromatography. Acetylator status was determined by % of metabolites in acetylated form (>40%=rapid, <40%=slow). Slow acetylators had higher steady state concentrations (Cp^{ss}) of SP metabolites ($p<.05$), area under curve (AUC)(total) ($p<.025$), AUC(SP) ($p<.001$). AUC(total) & AUC(SP) were significantly higher in patients in remission than those in active phase ($p<.05$). Disease activity and acetylator status had no effect on AUC(ASP). Patients studied during both relapse and remission had a significantly higher Cp^{ss} of SP during remission. No correlation was found between SP concentrations and side effects. **Conclusion:** prepubertal children with active IBD have lower Cp^{ss} of SP metabolites compared to quiescent disease, independent of acetylator status. (Supported in part by UB Research Foundation #1502082C).

534 HEPATIC BINDING PROTEIN (HBP)-ONTOGENY OF THE ASIALOGLYCOPROTEIN RECEPTOR IN MICE.

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Serum asialoglycoproteins bind to the hepatocyte receptor HBP, are internalized and catabolized in lysosomes. Human fetal liver is said to lack receptor, while adult liver contains 10 ug/gm determined as cpm I125-asialoorosomucoid (ASOR) substrate bound.

Female CD-1 mice produced eight timed litters of 10-14 pups, which were killed between birth and 23 days of age. Seven females were killed during pregnancy, immediately after delivery and during suckling. Virgin female and male mice were studied at 6 weeks and 4 months of age. After cervical dislocation, liver was removed, frozen at -70°C and assayed within 30 days. Aliquots of liver were homogenized, incubated with I125-ASOR (prepared from pooled human sera) in 0.1M Tris pH7.9 containing 0.1% Triton X-100 at 30° for 10 minutes. HBP-I125 ASOR complex was precipitated with 10% polyethyleneglycol, filtered, washed and precipitate cpm determined. Binding could be inhibited by specific anti-rat HBP antibody.

Fetal HBP activity was detectable one day prior to birth and rose to a plateau value at 5 days of age with no further increase to 6 weeks. In the last five days of pregnancy, maternal liver HBP activity was increased 150%. These data suggest fetal liver HBP is not involved in fetal glycoprotein metabolism, but HBP is inducible in maternal liver during pregnancy and in pups in the perinatal period. (NIAMDD Grant AM 17702).

535 GROWTH OF PRETERM INFANTS FED EITHER THEIR OWN MOTHER'S MILK (MHM) OR EXPERIMENTAL FORMULA (EF).

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Can intrauterine growth rates be attained in premature infants with birthweights of less than 1500 grams? We compared growth of infants fed an EF designed for low birthweight infants with that of infants fed MHM. Since MHM may promote better growth of her premature infant than bank human milk, it was chosen for study. EF is a whey predominant formula which contains 50% lactose and 50% polycose with 50% of fats as MCT. It provides 1200 mg/l of Ca and 600 mg/l of P. Eleven infants were fed MHM; 13 were fed EF. Mean birthweights were similar: MHM 1189g (940-1440) vs. EF 1270g (980-1490). Mean gestational ages were 30 weeks. No differences in kcal/kg/day or fluids/kg/day were noted. Serum Ca, Alk P'tase, protein and PTH were similar. Growth was as follows: * $p<.001$

GROWTH	WEIGHT	LENGTH	OF
MHM	11g/day	.10cm/day	.07cm/day
EF	22g/day*	.16cm/day*	.12cm/day*

In spite of a growth rate of almost twice MHM, the EF group's bony mineralization appeared slightly improved. EF fed infants reached discharge weight 2 weeks earlier than MHM infants. Intrauterine growth rates were achieved in the EF fed infants.

533 EFFECT OF INTRALIPID ON OXIDATIVE METABOLIC & FUNCTIONAL ACTIVITIES OF POLYMORPHONUCLEAR LEUKOCYTES (PMNL).

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Intralipid (I) is a soy bean emulsion commonly used in parenteral alimentation. We evaluated the oxidative metabolic and functional activities of PMNL exposed to various conc. of this substance. Intralipid produced a dose dependent stimulation of resting PMNL as measured by hexose monophosphate (HMPS) activity, nitroblue tetrazolium dye reduction (NBT), and oxygen consumption (O_2):

	No I	I (5-10mg/ml)	I (25-40mg/ml)
HMPS (CPM/ 10^6 PMNL)	376±85	1380±660	2091±836
NBT (OD $515nm/2.5 \times 10^6$ PMNL)	0.04±0.009	0.06±0.02	0.09±0.03
O_2 Consumption ($1/5 \times 10^6$ PMNL)	2.5±0.4	4.2±0.4	7.2±1.5

There was no difference between control PMNL stimulated with zymosan (Z) and cells stimulated with Z plus I. When PMNL were preincubated with I for 30 min. at conc. of 100 mg/ml or greater, Z stimulated HMPS activity (2199 ± 502) and NBT dye reduction (0.04 ± 0.005) were reduced ($P < 0.01$) when compared to non preincub. PMNL stimulated with Z: HMPS 7402 ± 752 and NBT 0.61 ± 0.06 . No effect was noted after preincubation with smaller conc. of I. Analysis of the kinetics of phagocytosis using 3H methyl thymidine labeled *S. aureus* show that preincubation of PMNL with I (100 mg/ml) caused a significant decrease in internalization of bacteria. Low conc. of I cause immediate stimulation of PMNL oxidative metabolism while higher conc. produce a significant inhibition in activity. This may be important in administration of I.

536 EFFECT OF CHRONIC CIMETIDINE (C) ADMINISTRATION ON GROWTH IN CYSTIC FIBROSIS (CF) PATIENTS.

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Twenty-five CF patients (7.5-17.9 years) participated in a one-year double-blind cross-over study to assess the influence of chronic cimetidine administration on growth parameters. Assessment of the group prior to entering the studies revealed coefficient of fat absorption while receiving pancreatic enzyme supplements from 35-95% (mean 64%), pulmonary function (FVC) from 32-100% predicted (mean 71%), and growth retardation with weight < 5% in 19, height < 5% in 14, and delayed bone age in 16 patients. Growth was assessed by height, weight, K^{40} lean body mass (LBM), muscle mass estimated by 24 hour urine creatinine, fat content by skin fold thickness, and bone age. RESULTS: 1) Average increases for any parameter were no greater on cimetidine than placebo. 2) Growth parameters did not show parallel changes. 3) No toxic effects were observed. 4) Two patients had no height increase and one patient had weight loss but 8 had decreases in LBM during the year. 5) Cimetidine reduced basal and pentagastrin stimulated gastric acid secretion at least by 50% in 19 cases. CONCLUSION: 1) Chronic cimetidine administration should not be a routine part of CF care. 2) Height and weight may be misleading parameters to follow in assessment of nutrition status.