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MACRO- AND MICRONUTRIENT INTAKES IN CHILDREN WITH CROHN'S DISEASE (CD). James Markowitz, Nancy Moses, Fred Daum (Spon. by Mervin Silverberg) Cornell Univ Med Coll, North Shore Univ Hosp, Dept of Pediatrics, Manhasset, NY

Poor nutrition has been associated with growth failure in the adolescent with long-standing CD. We have used 24-hour dietary recall to assess macro- and micronutrient intakes in 11 Tanner I-II CD patients (ages 8-14.5 yrs) within 6 months of diagnosis. 5 had linear growth failure (<4cm/yr). 8 had a mean weight deficit for height ($\pm$ SE) of 9.2 ± 2.5 kg. All had minimal disease activity (CDAI). 6 received sulfasalazine and 3 prednisone. Daily nutrient requirements were defined by 2 different methods based on each patient's ideal body weight (IBW) for actual height: Recommended Dietary Allowance (RDA) for chronological age, and caloric requirement for healthy Tanner I-II children (86 kcal/kg IBW). While 7/11 had 90% of expected caloric intake for chronological age, only 2/11 had adequate caloric intake for pubertal status. Mean caloric intake ($\pm$ SE) was $68 \pm 7\%$ of that recommended for healthy Tanner I-II children. Mean protein intake was 2.1 ± 0.2 gm/kg IBW. Carbohydrate constituted $44 \pm 2\%$ of calories, and fat $41 \pm 2\%$. Micronutrient intakes <70% RDA included: Vit D and folacin (11/11) zinc, calcium, magnesium, Vit B6 (6/11); iron (4/11); niacin, Vits C and E (3/11); thiamin (2/11); riboflavin (1/11). Phosphorus and Vit B12 intakes were adequate. Vit A intake >150% RDA was found in 8/11. Similar dietary abnormalities including caloric intake were found in patients with and without growth failure. Disturbances in both caloric and micronutrient intakes are common in Tanner I-II children with CD and require appropriate nutritional intervention.

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FAT LOSS DURING GAVAGE FEEDING: EFFECT OF MCT^R OIL SUPPLEMENTATION. Nitin R. Mehta, Margit Hamosh, Joel Bitman, D. Larry Wood (Spon. by Philip L. Calcagno). Georgetown University Medical Center, Washington, D.C. and USDA Agricultural Research Service, Beltsville, M.D.

Human milk (HM) fed to the premature infant is often fortified with MCT oil to increase its caloric density. The fat loss due to adherence to feeding sets (residual fat) was evaluated for the prevailing methods of MCT supplementation. Forty feeding sets, collected from infants on feeds of 3 different compositions, were analyzed for the amount of residual fat (measured as % of total fat in feed by gravimetry). The fatty acid (FA) composition of the fat residue was analyzed by gas liquid chromatography. The results (mean $\pm$ SD) show:

Group	Feed Composition	MCT Addition Method	N	% Residual Fat
A	HM	- - -	5	1.90 ± 0.69
B	HM and MCT	MCT followed by HM	12	2.63 ± 1.59
C	HM and MCT	MCT and HM mixed	23	$15.77 \pm 10.8^*$

*t-test $p < 0.001$

FA composition of the fat residue from Group C (given in % of total FA) shows: C8:0=59.95; C10:0=29.46 and other FA=10.59. Conclusion: 1) There is no significant difference in lipid adherence to feeding sets between Groups A and B; 2) In Group C up to 20% of lipid may be lost in the feeding set; 3) FA composition of the fat residue from Group C (given in % of total FA) shows that up to 90% of MCT oil added is lost in the feeding set.

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INTRACELLULAR AMINO ACID LEVELS AS PREDICTORS OF PROTEIN SYNTHESIS IN MAN. Jack Metcalf, Carolyn Johnson and James Gable, Department of Pediatrics, University of Oklahoma Health Sciences Center, Okla. City

The relation of protein synthesis (PS), a functional expression of amino acid (AA) balance, to the concurrent intracellular (IC) and plasma (P) concentrations of AA is uncertain. We studied 29 normal young adults & 63 neonates 27-44 weeks post-conceptual age, using the circulating neutrophil (LEUK) as the cell model. PS (3 H-LEU incorp, pmol/hr/mg DNA) in the LEUK, 19 AA's (HPLC, fluorometric detection, nmol/ml) in both LEUK & P, and cell water content (dry weight-"trapped" water by 14 C-inulin) were quantified. In infants, PS was inversely related to birthweight (BWT) & gestational age (GA) ($PS = 9265 - 1.2 \text{ BWTg} - 66.35 \text{ GA wks}$; $p=0.0001$). With the exception of VAL, CIT, GLN all the ICAA were higher than in P. HIS, ASP, TYR & ORN were the only significant (<.05) self-correlations between IC & PAA in infants; THR & ILE, in adults. Stepwise multiple regression (MR) was used to identify the set of AA's accounting for the greatest proportion of the variance (R^2) in PS. The sets of AA selected by the program were: for PAA in infants, ILE & ALA ($R^2=0.11$, $p=0.04$); for adults, ASP, GLY, MET ($R^2=0.32$, $p=0.009$). For ICAA in infants, LEU, MET, HIS, LYS, GLY, ALA ($R^2=0.32$, $p=0.001$); for adults, LEU, MET, HIS, ILE, PHE ($R^2=0.54$, $p=0.002$). By further MR analysis, levels of each ICAA predictor of PS in infants & adults was found to be predicted by a different set of PAA's, (e.g., for adults, IC-LEU predicted by P-THR, CIT, VAL, HIS, $R^2=0.60$, $p=0.0001$). We conclude that: (1) the level of PS can be predicted by a specific set of ICAA's better than by any set of PAA's. (2) The levels of the ICAA predictors of PS can then be predicted by specific, but different, sets of PAA's. These data suggest a possible practical means to modulate cell PS indirectly via ICAA concentrations by altering selected PAA.

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EFFICACY OF GASTRIC ACID SUPPRESSION BY CIMETIDINE IN PEDIATRIC PATIENTS. M. Mobassaleh, J. Biller, M. Maksimak, R. Grand, H. Winter. Divisions of Pediatric Gastroenterology, New England Medical Center and Children's Hospital, Boston, MA

The dose of cimetidine necessary to suppress gastric acid production in pediatric patients is not well established. Consequently, we studied 9 pts (mean age 7 yr, range 8m-10yr) with GI and/or pulmonary symptoms of gastroesophageal reflux, documented by abnormal overnight esophageal pH probe. The probe was then advanced into the stomach and gastric pH recorded before and after oral doses of cimetidine: 4 pts received randomized doses (5, 7.5, and 10 mg/kg) at 8-hr intervals; 5 pts received 5 and 10 mg/kg at 4-hr intervals. Age-appropriate standardized meals were given starting 1 hr following administration of the initial dose. Efficacy of gastric acid suppression was defined as gastric pH >4. RESULTS:

Cimetidine Dose (mg/kg)	Length of time gastric pH >4 (hrs)			
	0-1	1-2	2-3	>3
5.0	6/9	1/9	0/9	2/9
7.5	1/4	1/4	1/4	1/4
10.0	*2/9	2/9	4/9	1/9

*One pt, unresponsive to all 3 doses, was treated with 14.3 mg/kg; gastric pH measured >4 for 1.4 hrs only.

CONCLUSIONS: 1) Currently used cimetidine doses of 5 and 7.5 mg/kg fail to suppress gastric acid for >1 hr in some pts. 2) Two pts were unresponsive to doses as high as 10 mg/kg. 3) Documentation of gastric acid suppression is indicated in the management of pediatric patients with peptic related disease.

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APNEA AND LOWER ESOPHAGEAL SPHINCTER PRESSURE IN PRETERM INFANTS. Frank H. Morris, Marylynn Moore, Norman W. Weisbrodt, and Lenard M. Lichtenberger. Univ. Tx. Med. Sch. at Houston, Depts. of Ped. and of Physiol. and Cell Biol., Houston.

We studied the maturation of the lower esophageal sphincter (LES) function in 17 infants without apnea at 29-41 wk postconceptual age (PCA) using an indwelling manometry system with a response rate of 90 mm Hg/sec. LES pressure was measured as the mean of peak pressures obtained during replicate pull-through maneuvers with a 3-lumen catheter. Initial studies obtained in each infant revealed no change in LES pressure with advancing fetal age. Subsequent studies revealed no change during 3 postnatal wks in LES pressure from the first wk pressure of 55.5 ± 4 ($\bar{x} \pm \text{SE}$) mm Hg. However, LES pressure decreased in the 4th postnatal wk corresponding with 36 wk PCA.

We studied 26 additional infants of similar PCA who had apnea that was treated beginning at 7.7 ± 1.1 d with theophylline (THEO). The mean LES pressure in these infants was 69.2 ± 9.4 mm Hg before therapy and 75 ± 5.6 mm Hg during therapy, both greater values than that of infants without apnea ($p < 0.0002$). LES pressure did not decrease in apneic infants on THEO, but remained elevated for 7 postnatal wks.

Conclusions: (1) LES pressures are greater in preterm infants than in older infants; (2) beginning at 36 wks PCA, LES pressures decrease in normal infants; (3) preterm infants with apnea have greater LES pressures before and during THEO therapy than preterm babies without apnea, suggesting an associated pathophysiologic mechanism that is not interrupted by THEO.

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POSTPRANDIAL PLASMA AMINO ACID PATTERNS IN INFANTS FED CASEIN- AND WHEY-BASED FORMULAS.

Kathleen J. Motil, Cutberto Garza, Buford L. Nichols, Baylor College of Medicine, USDA/ARS Children's Nutrition Research Center, Department of Pediatrics, Houston, TX.

The postprandial plasma amino acid patterns in infants fed a casein- or whey-predominant formula were assessed to determine the order of the limiting amino acids. Healthy fullterm infants were studied at birth, 1, 2, and 3 months of age. All infants were fed solely a casein or whey formula. At the first feed of the day, fasting, 1-, and 2-hr postprandial venous blood samples were obtained for the determination of plasma amino acids by standard automated techniques. The volume of milk consumed was determined by pre- and post-feeding weights of the infants using an electronic balance. Results of whey-fed infants expressed as change over baseline (Mean $\pm$ SD) are shown:

Plasma Amino Acids	PAA (1 mo) (%)		PAA (3 mo) (%)	
	1-hr	2-hr	1-hr	2-hr
TRY	10 ± 35	34 ± 20	-12 ± 60	-12 ± 60
CYS	60 ± 149	33 ± 133	-5 ± 40	-23 ± 47
GLY	18 ± 14	-21 ± 6	-10 ± 15	-15 ± 7

Postprandial plasma tryptophan and cystine levels showed marked depressions at 3 months of age; plasma glycine levels fell persistently at 1 and 3 months of age. Thus, nonessential nitrogen and glycine may be limiting nutrients in infants fed a cow's milk whey-predominant formula.