† **630** IMPORTANCE OF PROTEIN QUALITY IN FEEDING THE LOW BIRTH WEIGHT (LBW) INFANT. Sudha Kashyap,Emi Okamoto, Shinobu Kanaya,Christine Zucker and William C. Heird. Columbia Univ. Col. of Phys. & Surgs., Dept. of Peds., New York.

To provide insight into the cause of metabolic acidosis (MA) observed in LBW infants fed high casein formulas, net acid balances (NAB) were preformed in infants (BW 900-1750g) fed formulas with a whey:casein ratio of either 18:82 (A; n=8) or 60:40 (B; n=8). Both provided 3.4g/kg.d of protein and 120 kcal/kg.d. Simultaneously, plasma and urinary amino acid (AA) concentrations including both bound and free cysteine concentrations (Malloy, et al, Anal. Biochem.112:407, 1981) were determined.

Neither growth nor nitrogen retention of the 2 groups differed. No infant in A or B developed MA and NAB did not differ (A=-1.61 mEq/kg.d; B=-1.72 mEq/kg.d). However, urinary organic acid excretion (OA), a component of net acid intake (NAI), was greater in A(1.81±0.3 mEq/kg.d vs 1.45±0.12 mEq/kg.d; $p<0.05$). Plasma {Tyr} but not other {AA} was higher in A(28.6±18.9 μm/dL vs 11.5±2.7 μm/dL; $p<0.05$) suggesting that some of their greater OA was a tyr metabolite. Neither other NAI components nor net acid output components differed. Despite the difference in cys intake, neither plasma nor urinary bound or free {cys} differed. Urinary {tau} of B was higher (5.27±0.5 μm/kg.d vs 4.17±0.7 μm/kg.d; $p<0.05$) but only 1/5 that of infants fed human milk (Rassin, et al, Pediatr. 71:179, 1983).

These data provide new information concerning both the mechanism of MA that may occur in infants fed higher intakes of casein and the importance of cys intake. They do not provide strong support for the superiority of whey proteins when total protein intake is <3.4g/kg.d.

● **631** PROTEIN AND ENERGY REQUIREMENTS OF LOW BIRTH WEIGHT (LBW) INFANTS. Sudha Kashyap,Mary Forsythe,Christine Zucker and William C. Heird. Columbia Univ. Col. of Phys. & Surgs., Dept. of Peds., New York.

Growth and metabolic response of LBW infants (BW, 900-1750g) assigned randomly at birth to formulas providing daily protein and calorie intakes of either 2.25g/kg and 120 kcal/kg(A;n=9), 3.8g/kg and 120 kcal/kg(B;n=9) or 3.8 g/kg and 160 kcal/kg (C;n=9) were determined. Weight gain of infants fed A (23.8±3.0g/d) was less ($p<0.05$) than that of infants fed B (33.6±4.8g/d) or C (40.2±4.9g/d); that of C was greater than that of B ($p<0.05$). Increases in length and head circumference were not different among the 3 groups but triceps and subscapular skinfold thickness (ΔSFT) increased more rapidly in those fed C($p<0.05$). Nitrogen retention of infants fed A(140±44mg/kg.d) was less ($p<0.05$) than infants fed B or C(293±88 and 283±38mg/kg.d). BUN (1.3±0.8 mg/dl, albumin (2.76±0.3g/dl) and prealbumin (6.2±1.9mg/dl) concentrations as well as the plasma concentration of most amino acids were also lower ($p<0.05$) in infants fed A. The lower weight gain and nitrogen retention of infants fed A along with the metabolic evidence of protein inadequacy suggests that a protein intake of 2.25g/kg.d is inadequate.

The higher protein content of B and C was well tolerated with no evidence of better utilization with higher energy intake. The fact that the greater weight gain of infants fed C was accompanby a greater ΔSFT suggests that these infants merely became fatter; thus, the higher energy intake appears to offer no advantage.

● **632** LECTIN-FREE DIET REDUCES INTRALUMINAL HYDROGEN (H_2) PRODUCTION IN PATIENTS WITH BACTERIAL OVERGROWTH SECONDARY TO INTESTINAL PSEUDO-OBSTRUCTION (IP). Mitchell H. Katz, Siv Modler, Susan Yarbrough, Jay A. Perman. Univ of California San Francisco, Department of Pediatrics and GCRC-PCRC.

Symptomatic small-intestinal bacterial overgrowth in patients with IP is associated with elevated fasting breath H_2 (FH_2) concentration (Gastroenterology 84: 1272, 1983). Symptoms and elevated FH_2 may be abolished by oral antibiotic therapy. Since plant lectins induce bacterial overgrowth in experimental animals (Gastroenterology 1983; 84:506), we determined whether lectin-free diets induce responses comparable to the effects of antibiotics. Three symptomatic patients ages 8-16 yrs with IP and confirmed bacterial overgrowth received lectin-free diets containing 21% of calories as carbohydrates for 1 week. All subjects reported symptomatic improvement. Lectin restriction reduced FH_2 from 203.2 ± 32.6 to 26.8 ± 20.8 ppm (mean ± SD; $p< 0.05$). Total breath H_2 excretion following 0.3 g/kg of the nonabsorbable sugar lactulose declined from 170 ± 133 to 61 ± 79 ml/6h. H_2 production/24h from glucose incubated with duodenal fluid obtained before and after lectin restriction declined 4-fold in the 1 subject where aspirate volumes permitted this determination. Adherence to the diet for 4 months in 1 subject has resulted in continued symptomatic improvement and reversal of previous dependence on antibiotics. Conclusion: These results demonstrate that dietary modifications in patients with bacterial overgrowth affect bacterial fermentative activities and associated symptoms.

633 ALLERGIC ESOPHAGITIS (A.E) Aubrey J. Katz, Alex F. Flores, Frank J. Twarog. (Spon. by Harvey Colten), Dept. of Ped. Harvard School of Medicine, Childrens Hosp. Med. Ctr., Boston.

Allergic gastroenteropathy (A.G.) is characterized by inflammation and eosinophilic infiltration of the stomach, small intestine,and/or colon. Esophageal involvement has been noted but not extensively studied. We present 6 patients with esophagitis as part of the spectrum of A.G. Esophagitis alone occurred in 2/6.

Age	Esophagitis	Gastritis	Enteritis	Colitis	pH probe
½ (yrs)	+	±	-	-	neg
6	+	-	-	-	neg
12	+	+	+	-	neg
10	+	+	+	-	neg
12	+	+	+	-	neg
25	+	+	+	-	N/D

Continual intraesophageal monitoring demonstrated no acid reflux. All patients had elevated IgE levels. (750-5,000 units). 5/6 patients had Rast/skin tests positive to all foods and inhalent allergens. Esophageal biopsies revealed severe esophagitis with marked basal cell hyperplasia, inflammation with eosinophilic predominance. Treatment:5 responded to steroids. 1 responded to diet. Conclusion:1. Esophagitis commonly occurs in A.E. and is not due to GE reflux. 2. A.E. occurs alone or in combination with gastritis/enteritis. 3. Symptoms suggestive of esophagitis are often absent.

† **634** FAMILIAL APNEA AND GASTROESOPHAGEAL REFLUX. Dorothy H. Kelly, Aubrey J. Katz, Lucienne A. Cahen, Alex F. Flores, Daniel C. Shannon. Department of Pediatrics, Harvard Medical School, Boston, MA.

We studied 10 children (BW$\bar{x}$3.5kg, 2.4-4.7kg; GA$\bar{x}$39.9, 36-42 wk; 7f,3m) in 4 families with gastroesophageal reflux (GER) and recurrent apnea. All had sleep apnea with cyanosis leading to resuscitation (R) in 5, and vigorous stimulation (VS) in 5. They also had stridor, choking, obstructive apnea and cyanosis while awake leading to R(3) and VS (7). Symptoms of apnea began at a mean of 3.5 wk(2d-7.4 wk) and of GER at 5.2 wk(2d-26wk). Pneumograms documented an increase in periodic breathing ($\bar{x}$9.0±6.8%) and/or prolonged apnea ≥ 16 sec. in 7/9 infants. GER was documented by Ba esophagram (6/9), pH probe (6/8) or endoscopy (3/6). Symptoms of GER resolved at 21 and 25 months in 2 with medical management (MM) including metaclopromide. Two infants (5 and 12 wk) are symptomatic on MM. Because of failure of MM, fundoplication (FP) was performed in 6 at a mean of 2.9 yr (1.0-8.8 yr). After FP, all continued to have sleep apnea requiring VS or R. In one, because of recurrence of awake stridor and obstructive apnea 4 years after FP, FP was repeated and obstructive apnea has not recurred in 10 mo. In one family, a female sibling with history of vomiting, apnea, cyanosis at 3 wk and frequent choking and spitting died of SIDS at 2 mo. In a second family, the father had a FP at 28 yr for severe GER. In summary, we have described familial apnea and GER in 10 children all of whom had awake stridor and obstructive apnea as symptoms of reflux. These symptoms were relieved by FP; however, significant sleep apnea persisted.

● **635** HIGH DIETARY PROTEIN/ENERGY (P/E) RATIO SPARES TISSUE NITROGEN (N) IN ASCITES TUMOR-BEARING MICE TREATED WITH ACINETOBACTER GLUTAMINASE-ASPARAGINASE (AGA). Craig L. Kien, Alfred J. Anderson, John S. Holcenberg, Medical College of Wisconsin, Midwest Children's Cancer Center, Milwaukee Children's Hospital, Departments of Pediatrics, Biochemistry, Medicine, & Pharmacology/Toxicology, Milwaukee, WI

Previously, we showed that a high protein (58g%; HP) diet caused marked N sparing of liver (L), intestine (I), thymus (T), and carcass (C) compared to an isocaloric, normal protein diet (20g%; NP) in normal mice treated with AGA (Pediatr Res 17: 192A). In the present study, 32 mice previously innoculated with Ehrlich Ascites tumor were randomized into 2 treatments: AGA (600 IU/kg/d, 7d) and 0.9% NaCl controls and into 2 diets HP or NP. In both diet groups, AGA resulted in markedly lower ($p < 0.01$) tumor burdens as characterized by ascites volume, cell count, or tumor N content, but HP did not increase tumor burden. In the AGA group: HP resulted in significant N sparing in the following tissues (% increase from NP; P value): L (18%; P=0.05), I (25%; $p<0.05$), T (164%; $p<0.01$); HP completely prevented the N depleting effect of AGA on L and T; HP resulted in significantly ($p< 0.05$) higher hematocrit (42%) and serum total protein (5.5 g/dl) compared to NP (33% and 5.0 respectively) but in lower total food intake and insignificant differences in blood counts. Our conclusion may be relevant to man, to other cancers, and to those forms of chemotherapy which affect N utilization: an increased dietary P/E ratio, even without energy supplementation, may improve N content of T, L, and I without antagonizing anti-cancer therapy. (Support: NIH Grant CA20061)