612 SODIUM DEPLETION IN INFANTS AND CHILDREN WITH BILIARY DRAINAGE PROCEDURES (BDP). Mina Gurevitz, Richard K. Danis, Thomas V. Cradock, Thomas R. Weber and Kathleen B. Schwarz (spon. by Thomas Aceto, Jr.). St. Louis University School of Medicine, Cardinal Glennon Memorial Hospital for Children, Dept. of Peds., St. Louis, MO.

Sodium excretion from the biliary tract has not been well studied in children but could lead to hyponatremia and growth impairment in children with BDP. Salt homeostasis was studied in 17 infant outpatients with BDP for biliary atresia (BA). Bile(B) serum (S), and urine (U) were collected simultaneously and analyzed for Na+ and CL^- at 1-3 month intervals during the post operative period for 1-12 months; 49 analyses of B,S, and U each were performed. Mean B Na+ remained high (122±15 mEq/l) even though mean S Na+ was low (132±7). Since bile volume was sometimes as high as 1000 ml/day, biliary NA+ losses could easily lead to depletion in spite of maximal renal Na+ conservation. (U Na+ fell to 0 when S Na+ was 132 mEq/l). B Cl^- (115±21 mEq/l) consistently exceeded that of S Cl^- (95±12).

Data from 3 additional patients with BDP who became ill from Na depletion presented below. Symptoms resolved in all with saline supplements and bile refeeding.

Diagnosis	Age	Symptoms	S Na+ mEq/l	U Na+ mEq/l	B Na+ mEq/l	B Vol ml/day
BA	4mo	Shock	109	0	111	30-50
BA	1yr	Seizures	108	0	97	800
Choledochal cyst	2yr	Anorexia	128	1	159	300

Conclusion: Children with BDP are at risk for NA+ depletion and should be monitored and treated accordingly.

† 613 ZINC BALANCE IN VERY LOW BIRTH WEIGHT PRETERM INFANTS FED OWN MOTHER'S MILK. K. Michael Hambidge, Margaret A. Jacobs, Roberta L. Barth, Kimberly O. Kuether, University of Colorado Health Sciences Center, Department of Pediatrics, Denver.

Results of zinc balance studies in VLBW preterm infants have been variable, but very negative balance has been reported in infants fed pooled pasteurized human milk (Dauncey et al, Peds Res 11:991-7, 1977). The aim of this study was to determine zinc balance during feeding of own mother's fresh or fresh-frozen milk (MM) and to compare this with balance during formula feeding (FF). Serial 3-day balance studies were conducted in 6 premature infants, 3 males, 3 females (gestational age 27.9 ± 1.9 wks, BW 942 ± 187 g, mean ± SD) on 21 occasions (13 MM, 8 FF) between 2-8 weeks postpartum age (MM 30 ± 13 days, FF 38 ± 14 days). Mean intake, fecal Zn, net absorption (milk Zn - fecal Zn) and balance (milk Zn - [fecal Zn + urine Zn]) are expressed as µg Zn/kg body wt/day in the table.

		Intake	Fecal Zn	Net Absorption	Balance
MM	13	514 ± 303	575 ± 322	-48 ± 103	-88 ± 93
FF	8	1215 ± 243	1357 ± 306	-146 ± 160	-178 ± 163
		($p<0.005$)	($p<0.005$)	($p<0.05$)	($0.10>p>0.05$)

Fecal Zn was + correlated with Zn intake ($r=0.97$, $p<0.001$). Intake was higher in FF, but net Zn absorption and Zn balance were more negative especially prior to 28 days of age (MM (6) -69 ± 105, FF (2) -313 ± 93, $p<0.05$). Individual balance results ranged from -239 to +93 for MM and from -374 to +79 for FF. It is concluded that MM is advantageous with respect to Zn status in VLBW infants, especially prior to 1 month of postnatal age.

† 614 EFFECT OF MATERNAL ANTIBODIES AND ENTERIC CHALLENGE WITH ANTIGEN ON THE UPTAKE OF BYSTANDER PROTEIN IN NEONATAL RABBITS. Paul R. Harmatz, Ronald E. Kleinman, Bruce W. Bunnell, MaryAnne Cristello, W. Allan Walker, Kurt J. Bloch, Harvard Medical School, MGH/CHMC, Depts. of Pediatrics and Medicine, Boston, MA.

Antibodies, passively acquired from the mother, were previously shown to limit the circulation of antigen given enterically to newborn rabbits. In the present experiments, we tested the effect of passive immunization and enteric antigen challenge on uptake of bystander protein. Female rabbits were immunized with bovine gamma globulin (BGG). Litters from these animals were tested prior to suckling by the intragastric administration of bovine serum albumin (BSA) plus BGG (test) or ovalbumin (OVA) (control animals). Three hrs. later, serum from both groups was tested for immunoreactive BSA by radioimmunoassay. Test animals had significantly less serum iBSA than controls. In other experiments the same protocol was followed except for the addition of ^{14}C-polyethylene glycol (^{14}C-PEG). Three hrs. after challenge, the concentration of radioactivity in various segments of intestine was determined. There was less ^{14}C-PEG in the small intestine of test compared to control animals. These findings suggest enteric antigen-antibody interaction in intact animals may limit contact of protein with the absorptive surface on the enterocyte and thus diminish uptake of bystander protein in the neonate.

615 MACROPHAGE FUNCTION IN NEONATAL PROTEIN CALORIE MALNUTRITION IN RATS. M.C. Harris, J.S. Gerdes, R.A. Polin, M.M. Ziegler and S.D. Douglas, Univ. of Pa. Sch. of Med., Children's Hosp. Phila., Dept. Ped., Phila., PA.

Abnormalities in phagocyte function occur in protein calorie malnutrition (PCM). We have compared *in vitro* random motility (RM), chemotactic response (CR) to the synthetic oligopeptide N-formyl methionyl leucyl phenylalanine (fMLP), and bactericidal activity (BA) *in vitro* using type Ic group B streptococcus (GBS) in a rat model of PCM. Pregnant Wistar rats at 14 days gestation received either a normal (23.4% protein), or low (2.5% protein) diet until delivery (day 21) and during the subsequent 21 day period of lactation. Control and low protein pups were studied on day 21. Alveolar Macrophages (AM) were prepared by lavage and peritoneal macrophages (PM) were harvested 48 hours following installation of 1% glycogen in 0.85% NaCL (5cc/100 gms). RM and CR were measured in nucleopore chambers. BA was studied using GBS and 10% human serum at 60 and 120 minutes. RM of human neutrophils (87%) was greater than adult rat (47%) or pup (19%) PM, and adult rat (15%) or pup (8%) AM. Malnutrition did not affect RM. Similarly, CR for PM was not affected by malnutrition. AM demonstrated little CR to fMLP. BA of rat pups was similar for AM and PM at 120 min. (42% killing), whereas in PCM, AM and PM showed diminished BA (22-27%) at 120 min. Impaired host defense in neonatal PCM may be related to blunted microbicidal activity.

616 BARRETT'S ESOPHAGUS IN CHILDHOOD. Eric Hassall, Wilfred Weinstein, Marvin Ament, UCLA School of Medicine, Depts. of Peds. and Medicine, LA, Ca.

Barrett's esophagus or columnar-lined esophagus (CLE) is a metaplasia state associated with gastroesophageal (GE) reflux and increased risk of esophageal adenocarcinoma in adults; in CLE, columnar epithelium replaces normal squamous lining. Previous reports of CLE in children are rare and usually based on single biopsy diagnosis. We report a detailed clinical and histologic study in 10 children with CLE, ages 6 1/2-13 yr., diagnosed between 1976-1983. All were males. 8 had mid or upper esophageal strictures, comprising 25% of all childhood strictures at UCLA during that period. All 10 had low lower esophageal sphincter pressures and or abnormal esophageal pH studies. Vomiting, dysphagia, substernal pain or cough were present for average 8 yr; 9 had severe vomiting or pneumonias below 1 yr. age. A single esophageal biopsy containing columnar epithelium may represent normality or hiatus hernia; in order to avoid this potential confusion, esophageal biopsies were taken under direct vision at endoscopy from several levels above the GE junction. The diagnostic criterion for CLE was the presence of columnar epithelium in 2 or more biopsies taken 2 cm or more above the anatomic GE junction. A total of 40 esophageal biopsies in 10 patients contained columnar epithelium; surface epithelium was gastric-type alone in 6 patients and gastric with intestinal-type in 4. These studies show that a) CLE should be considered in all children with mid or upper esophageal stricture and in those with longstanding symptoms of GE reflux; b) intestinal metaplasia appears to be less common in childhood CLE than in adults.

617 PANCREATIC BIOTINIDASE ACTIVITY: THE POTENTIAL FOR INTESTINAL PROCESSING OF DIETARY PROTEIN-BOUND BIOTIN. Gregory S.Heard, Barry Wolf and *Janardan K. Reddy. Depts. of Human Genetics and Pediatrics, Medical College of Virginia, Richmond, VA, and *Department of Pathology, Northwestern University Medical School, Chicago, Illinois.

Biotinidase cleaves biotin from biocytin (ε-N-biotinyl-lysine) and biotinyl-peptides resulting from the degradation of carboxylases and, consequently, is important in recycling the vitamin. Some of the variability in the clinical features and age of onset of biotinidase deficiency (ranges from 3 weeks to several years) may be a consequence of differences not only in the total dietary biotin consumption but also in the form of the vitamin in the diet. Biotinidase-deficient children whose diets contain foods rich in free (as opposed to protein-bound) biotin may take longer to become biotin-deficient and, hence, to manifest the signs and symptoms of biotinidase deficiency. We have shown that biotinidase activity in rats is not enriched in intestinal brush border membranes but it is present in mucosa from all sections of the small intestine, and it is also in pancreatic homogenates (24.0±5.5 pmol/min/mg protein), isolated secretory granules (6.3 pmol/min/mg) and pancreatic juice from cannulated ducts (316±189 pmol/min/ml or 7.0±4.2 pmol/min/mg). The apparent K_m of N-biotinyl-p-aminobenzoate (an artificial substrate) for biotinidase was 10 µM and broad pH optima of 4 to 8 were observed for enzymes from both rat serum and pancreatic juice. These findings suggest that biotinidase has an important role in increasing the bioavailability of dietary biotin.