† 572 HEPATIC CYSTS IN CPK MICE; A MORPHOLOGIC STUDY AND TECHNIQUE FOR VISUALIZATION OF INTRAHEPATIC BILE DUCTS. Paul C. Grimm, John F. Crocker, Malcolm R. Ogborn, Dickran A. Malatjalian, Dalhousie Univ., Izaak Walton Killam Hospital for Children and Victoria General Hosp., Depts. of Peds. and Path., Halifax, Nova Scotia.

The pathogenesis of hepatic cysts in human polycystic kidney disease (PKD) is unknown. The homozygous mutant, cpk/cpk mouse is characterized by PKD and early uremic death. We have recently found an age related increase of macroscopic liver cysts in 119 heterozygotes (cpk/+); 10%, 22% and 65% at 5, 10 and 15 months of age respectively. To determine if these cysts arise from functioning biliary elements or from non-communicat-ing embryonic remnants, we developed a technique to produce casts of intrahepatic bile ducts. After sacrifice, the common bile ducts in 19 cpk/+ adult mice were isolated and cannulated in situ. MICROFIL silicone rubber was injected under 30 cm H_2O pressure until the distal duct radicals on the surface of the liver were filled. The livers were removed, fixed and cleared with formalin, ethanol, and methyl salicylate. Cysts were also studied by histologic and EM techniques. We demonstrated a continuum from small fusiform dilatations to large saccular structures in communication with the biliary system. These changes were not observed in control C57BL/6J animals. We believe this is the first time that hepatic cysts associated with experimental PKD have been demonstrated to arise from bile ducts and in a manner analogous to cyst development within the nephron.

573 BILIARY ATRESIA AND THE POLYSPLENIA SYNDROME Roberta J Hall, Stephen K Greenholz, Juan M Vasquez-Estevez, John R Lilly (Spon by Frederick C Battaglia) University of Colorado School of Medicine, Department of Surgery, Denver

Currently, there is a widely held but unsubstantiated perception that infants with biliary atresia and co-existing polysplenia uniformly fail Kasai operations; referral for transplantation should be done immediately and without attempted corrective surgery.

14 of 118 consecutive patients (12%) having Kasai hepatic portoenterostomy for biliary atresia at our institution had one or more of the additional components of the polysplenia syndrome: intestinal malrotation (6), preduodenal portal vein (3), aberrant hepatic artery (3), situs inversus (2), absent inferior vena cava (2).

Although technically more demanding, 11 of the 14 polysplenia patients achieved biliary drainage after operation. Three patients had subsequent liver transplantation; 2 of whom died. Long-term survival (mean follow-up, 4 years) was achieved in 5 (45%), again not significantly different from that in the larger group (40.2%).

The Kasai operation is the initial operation of choice in patients with coexisiting biliary atresia and the polysplenia syndrome.

574 ELEVATED SERUM MANGANESE (Mn) IN TOTAL PARENTERAL NUTRITION (TPN) CHOLESTATIC LIVER DISEASE. K. Michael Hambidge, Clare E. Casey, Ronald J. Sokol, Sara J. Fidanza. University of Colorado School of Medicine, University Hospital, Dept. of Pediatrics, Denver CO 80262.

Mn, like copper, is an essential trace element that is excreted almost entirely via the bilary tract. Copper accumulation occurs when bile flow is compromised but similar data for Mn are not available. The objective of this study was to determine serum Mn concentrations in a patient with cholestatic liver disease who was receiving TPN. The subject was a 16 month old female, weighing 10.5 kg, with intractable diarrhea who had been receiving TPN since 1 week of age. Despite repeated attempts at partial enteral feeding, she developed progressive cholestatic liver disease. At the time of this study, liver function tests included: bilirubin 10.4 mg/dl, AST 424 IU/l, alk phos 264 IU/l. She had been receiving 2.4 mcgMn/day added to her TPN infusate. The table gives serum Mn levels determined by flameless atomic absorption spectrophotometry before and at progressive intervals after Mn was withheld from the infusate:

Days off I.V. Mn	0	12	26	35	Adult Controls
Serum Mn(ng/ml)	4.4	3.2	2.0	1.4	0.89±0.18 (X±SD,n=10)

Two additional patients, aged 9 and 15 months, who had also been receiving Mn in long-term TPN but had not developed significant cholestasis both had serum Mn of 1.0 ng/ml. It is suggested that intravenous Mn, as a component of TPN, be administered in reduced quantities to children with cholestatic liver disease.

† 575 INTESTINAL ADAPTATION DURING LACTATION IN THE MOUSE: ENHANCED UPTAKE OF DIETARY PROTEIN ANTIGEN. Paul R. Harmatz, Donald Hanson, Marc Brown, Ronald E. Kleinman, Kurt J. Bloch, W.Allan Walker. Harvard Medical School, Massachusetts General Hospital and Children's Hospital, Department of Pediatrics and Medicine, Boston.

During lactation, dietary protein ingested by the mother may be transferred to the nursing infant. In this study, we sought to determine whether the uptake of protein was enhanced in lactating vs control mice. Lactating BDF1 mice (6-9 days postpartum) and age-matched nulliparous controls were gavaged with ovalbumin (OVA 10 mg in 0.5 ml saline). Blood was taken 15,30,60, and 120 min later and the serum concentration of OVA was measured by enzyme immunoassay (EIA).

	15 min	30 min**	60 min***	120 min	*M ± SEM (ng/ml)
Lactator	20 ± 6*	77 ± 21	55 ± 11	27 ± 5	** P<.02
Control	31 ± 1	34 ± 10	15 ± 2	16 ± 6	***P<.007

The concentration of OVA in the serum of lactators was significantly greater at 30 and 60 min. There was no difference in serum (iOVA) on day 1 of lactation, but the differences noted at mid-lactation were maintained through day 18. Plasma volume and clearance of OVA from the circulation may influence serum OVA concentration. The plasma volume of lactators was found to be 2-fold greater than that of controls. The rate of clearance of unlabelled OVA injected intravenously was not different in the 2 groups. We conclude that the difference in serum concentration of OVA was associated with greater protein uptake from gut in lactating versus control mice.

● 576 AUGMENTATION OF POST-RESECTION MUCOSAL HYPERPLASIA BY LINOLEIC ACID FEEDING by Michael H. Hart, Jung H.Y. Park, Carter J. Grandjean, Steve H. Erdman, and Jon A. Vanderhoof, University of Nebraska College of Medicine, Department of Pediatrics, Omaha, Nebraska.

Previous studies indicate long chain fats have trophic effects on post-resection intestinal mucosal adaptation. We have previously shown that short term essential fatty acid (EFA) deficiency impairs normal post-resection mucosal hyperplasia, while 16, 16 dimethyl-PGE2 administration stimulates hyperplasia. The effect of increased dietary linoleic acid (LA) on promoting mucosal adaptation in resected, non-EFA deficient animals was evaluated. Ten Sprague-Dawley rats (5 w/o males) were pair-fed isocaloric diets containing 1% or 5% (LA) (w/w). After 2 weeks all animals underwent 70% proximal jejunoileal resection. Animals were then pair-fed for 14 days. Following sacrifice mucosal weight, protein, DNA, and sucrase activity were determined:

	MUCOSAL PROTEIN (MG/CM BOWEL, MEAN ± S.D.)		
	Duo/Jej	Mid Ileum	Dist Ileum
1% LA	13.25±3.84	9.39±2.11	6.67±1.59
5% LA	22.69±2.70*	16.58±2.94*	9.52±1.38*

Protein levels were increased in all segments in 5% LA animals ($p<.05$). Similar results were seen for mucosal weight, DNA, and sucrase activity. Our results indicate 5% LA has "trophic" properties on post-resection mucosal adaptation similar to the effect of 16,16 dimethyl-PGE2. In view of reports of increased dietary LA stimulating intestinal PGE2 synthesis, LA supplementation may exhibit its stimulatory effect via this mechanism.

† 577 DEVELOPMENTAL DIFFERENCES IN CALCIUM SOURCES UTILIZED FOR CONTRACTION IN THE CAT'S UPPER GASTROINTESTINAL TRACT. Craig Hillemeier, Dominique Bereiter, and Peter Biancani (Spon. by A. S. Brem). Brown University and Rhode Island Hospital, Depts. of Pediatrics and Internal Medicine, Providence, Rhode Island.

Isolated circular smooth muscle cells from the esophagus, fundus, and antrum in the adult cat and newborn kitten were compared. Dose response curves were obtained to Acetylcholine (Ach) in the presence of normal Calcium (Ca^{+2}), zero Ca^{+2}, and the Ca^{+2} channel blocker D600. Cells from the esophagus of the adult and kitten contracted similarly in Ca^{+2}, but both failed to show any contraction in zero Ca^{+2} or D600. Fundic cells in both the adult and kitten contracted the same in Ca^{+2}, and also showed maximal contraction in zero Ca^{+2} or D600. The contractile response of antral cells was no different between adult and kitten in normal Ca^{+2}. However, the adult antrum contracted 53.3% and 47.6% of maximum in zero Ca^{+2} and D600 respectively, while the kitten's antrum contracted significantly less ($p<.001$) at 6.4% and 3.2% of maximum. Antral cells were then saponified (permeabilized) and exposed to inositol tri-phosphate (IP_3), which releases intracellular Ca^{+2} stores. With IP_3 adult antral cells contracted 52.1%, while the kitten antral cells failed to contract. We conclude that in response to Ach the esophagus and fundus of the adult cat and kitten utilize similar Ca^{+2} sources, with the esophagus using extracellular Ca^{+2} and the fundus depending upon intracellular Ca^{+2} stores. The adult antrum is able to utilize intracellular Ca^{+2} stores for part of it's contraction while the kitten is not. We speculate this may be due to a lack of intracellular Ca^{+2} stores in the newborn kitten antrum.