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EFFECT OF AGE ON ABSORPTION OF MANGANESE FROM HUMAN MILK, COW'S MILK AND INFANT FORMULA. Hassan Raghieb, Wai-Yee Chan and Owen M. Rennert. University of Oklahoma Health Sciences Center, Departments of Pediatrics and Biochemistry and Molecular Biology, Oklahoma City.

Bioavailability of manganese (Mn) in human milk, cow's milk and infant formula (Similac) was found to be different. The effect of intestinal maturation on the absorption of nutrients is well established. Using neonatal rat pups 8 to 11 days old, we have studied the effect of age on the bioavailability of manganese from these three types of milk. Rat pups starved for 14 hours were fed by intubation with Mn-54 labeled milk or formula. Absorption of Mn was monitored 3 hours after feeding by measuring Mn-54 radioactivity in the whole carcass without the digestive tract or the liver alone. Based on total radioactivity recovered in the carcass, absorption of Mn from formula was not affected by the age of the animal. Younger pups, however, absorbed Mn more efficiently from human milk and cow's milk than older pups (human milk: 31.6% fed at 8 days compared with 14.5% at 11 days; cow's milk 27.0% fed at 11 days compared with 13.1% at 11 days). A transition from high absorption to low absorption occurred at 10 days. Even though the liver accumulated comparable amounts of Mn-54 in 8 day old pups when they were fed labeled milk, or formula, significantly higher quantities of Mn-54 were found in the liver of formula fed 11 day old pups. This difference in absorption of Mn from different types of milk might be accounted for by the change with age in the ability of the absorptive system to process the different Mn binding ligands in the milk studied. (Supported by NIH grant HD 16730)

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HYDROXYSTEROL AND BILE ACID METABOLISM: TRANSITION FROM FETAL TO NEONATAL LIFE. Indira Rajagopalan, Harikrishna Shukla, Engeline Kok and Norman B. Javitt (Spon. by Joseph Dancis) Department of Pediatrics, New York University School of Medicine, NY 10016

We have developed a noninvasive approach using stable isotopes that describes quantitatively the transition in hydroxy-sterol and bile acid metabolism. Deuterated (D4)-26-hydroxycholesterol disulfate (50 µg) dissolved in MCT oil is given to normal neonates before initial feeding and diapers collected at approximately 2 hr intervals for 72 hours. Prior to analysis, a radioactive standard is added which permits calculation of total recovery of endogenous and isotopic 26-hydroxycholesterol after quantitation by isotope ratio mass spectrometry. The data from 3 normal neonates indicates rapid excretion of a preformed pool. In one instance, 82% recovery of D4 implied enterohepatic circulation, confirmed by identification of isotope in urine. Our approach (1) defines the normal transition from fetal to neonatal life, (2) gives insight into the role of altered bile acid metabolism in the pathogenesis of neonatal cholestasis (3) is generally applicable to a variety of metabolic pathways.

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PERINATAL FACTORS UNDERLYING NEONATAL CHOLESTASIS. Aarti Raut, Prabha C. Dosi, Edwin G. Brown, Bernard Z. Karmel, Richard W. Krouskop, and Avron Y. Sweet. Division of Neonatology, Mount Sinai School of Medicine, Department of Pediatrics, New York, New York.

We evaluated prolonged use of total parenteral nutrition (TPN) and the effects of perinatal factors in infants who developed neonatal cholestasis (CH). Only infants <1500 gms at birth admitted to our Intensive Care Nursery during the first 3 days of life who survived >2 weeks, had no GI disease and received TPN >14 days were studied; 40 infants met these criteria. 19 infants developed CH (direct bilirubin $\geq 1.5 \text{ mg/dl}$ >1 week) and 21 did not. Infants who developed CH received TPN longer than those without CH ($P < 0.01$). However, the mean duration ($\pm 1 \text{ SD}$) of TPN at the onset of CH was 32 ± 13 days while the mean duration ($\pm 1 \text{ SD}$) of TPN in the unaffected infants was 33 ± 16 days ($P = \text{NS}$).

Infants with CH compared to those without CH had a greater frequency of hypotension ($P < 0.01$) and severe hyaline membrane disease ($P < 0.01$), although no differences were observed in birth weight, gestational age, Apgar scores, and the first blood gas results. These findings indicate that among immature infants, severe systemic and pulmonary hypoperfusion (shock) appears to damage the liver to the degree that during TPN its impaired excretory function is overwhelmed, leading to CH.

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PSYCHOLOGICAL EVALUATION OF CHILDREN WITH ABDOMINAL PAIN: OBJECTIVE TESTING FAILS TO DISCRIMINATE BETWEEN "NON ORGANIC" AND "ORGANIC" CASES. D. Raymer, O. Weinger & R. Hamilton, Dept. Ped., Hosp. Sick Children, & Dept. Applied Psych., University of Toronto, Toronto, Ontario.

To determine prevalence of psychological problems in children with abdominal pain, we compared 60 randomly selected patients, 8-16 yr. with chronic pain caused by Crohn's disease, Cr, (24), ulcerative colitis, UC, (20), or non-organic disorders, RAP, (16) with 30 pain-free matched controls. A self-report battery of 5 tests assessing personal, family and social adjustment showed 3 groups with significant abnormalities compared with controls.

	Psychological Scores (m±SEM)				
	Crohn's	Ulc.Colitis	RAP	Control	p
Self-esteem	40.7±1.7	43.5±1.5	39.8±2.0	46.3±1.0	<0.01
Pers. maladjust.	29.3±1.9	30.4±2.1	30.1±1.8	29.7±1.8	NS
Life events	4.3±0.7	2.5±0.5	2.1±0.5	2.9±0.4	<0.02
Stim. appraisal	7.0±0.9	6.5±0.6	7.1±1.0	5.5±0.8	NS
Depression	8.3±1.3	8.2±1.6	8.3±2.1	4.5±0.8	<0.08

Paired comparisons showed Cr & RAP groups scored lower in self-esteem than controls ($p < .005$); Cr patients reported more stressful prior events than UC; depression was greater in Cr ($p < .02$) and UC cases ($p < .05$) than in controls but organic and non-organic groups did not differ significantly in any of the variables. Severe psychological stress ($2 \text{SD} > \text{mean}$) was equivalent in organic and non-organic groups. We conclude that significant emotional problems accompany abdominal pain in children. Contrary to previous observational reports, psychological measures failed to distinguish "non organic" from "organic" cases.

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PANCREATIC ENZYMES PARTICIPATE IN THE DEGRADATION OF INTESTINAL SUCRASE-ISOMALTASE. Jacques E. Riby and Norman Kretschmer. University of California, Berkeley, Department of Nutritional Sciences.

The pancreatic ducts were by-passed with a catheter placed within the common bile duct in order to prevent the entry of pancreatic enzymes into the duodenum without interrupting bile flow. For eight days, rats were fed a diet (peptones, sucrose, coconut oil, vitamins and minerals) that could be digested without pancreatic enzymes. Control animals were sham operated and pair fed with the same diet. Relative rates of synthesis and degradation were estimated by pulse labelling and double labelling respectively, for sucrase and for total protein, in total intestinal mucosa and along the gradient of cells collected from the tip of the villus to the bottom of the crypt. The rate of degradation of sucrase was 1.7 times higher than that of total protein in controls, whereas in experimental animals it was equal to that of total protein. This change in rate of degradation produced a proportional increase of sucrase activity in experimental animals. The effect of pancreatic enzymes on sucrase was apparent along the entire length of the villus but not in the crypt. These data support the hypothesis that pancreatic proteases release sucrase-isomaltase from the brush-border membrane, resulting in the observed increase of the rate of degradation. Electrophoretic separation of immunoprecipitated sucrase-isomaltase showed that the intact pro-sucrase-isomaltase observed in operated animals is split into two subunits (sucrase and isomaltase) by the action of pancreatic proteases in control animals. Supported in part by grant # 1 T32 HD 07266-01 NIH

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EFFECTS OF MALNUTRITION AND PASSIVELY ACQUIRED MUCOSAL IMMUNITY ON ROTAVIRUS INFECTION DURING INFANCY. M. Riepenhoff-Talty, E. Offor, E. Kowalski, P.J. Carmody, and P.L. Ogra. SUNY, Buffalo, N.Y. 14222.

Groups of severely malnourished and well nourished (control) 6 day old mice were treated with orally administered human immune serum globulin (ISG) containing antibody to rotavirus, or with culture media (placebo). The animals were infected with mouse rotavirus (MRV) 1 to 2 hrs after the last dose of ISG or placebo. The techniques of immunofluorescence and enzyme linked immunoassay were employed to determine the course of MRV replication in isolated villous enterocytes and for shedding of viral antigen in the feces respectively. Moderately severe diarrhea was observed within 48 hrs. in 100% of (118) malnourished or control animals in the placebo groups. However, only 49% of ISG treated malnourished animals and 30% of control animals developed diarrhea. The fecal shedding of MRV antigen in malnourished placebo treated animals was 4 to 8 fold higher than in ISG treated malnourished or control animals. Of particular importance is the observation that the frequency of detection of MRV in the enterocytes was significantly ($P < 0.01$) higher in malnourished placebo group (11%), than in ISG treated malnourished animals (<1%). These observations indicate that enhanced replication of MRV in enterocytes may underlie the development of severe diarrhea in malnourished infected subjects. It is suggested that the availability of immune mechanisms through the intestinal mucosa is particularly effective in limiting the pathogenesis of MRV during malnutrition.