**644** AN IMPROVED MODEL FOR NITROGEN BALANCE IN PREMATURE INFANTS IN THE CLINICAL SETTING. J.L. Sutphen (Spon. by R.J. Grand. Harvard Med. School, Children's Hosp. Med. Ctr., Division of Gastroenterology, Boston, MA

Nitrogen balance in the clinical setting is useful in assessing efficacy of nutritional therapy and evaluating nutrient products. Multiple often uncontrollable variables influence nitrogen balance. Expression of nitrogen balance as a function of nitrogen intake is therefore an oversimplification. Using stepwise linear regression techniques we examined 8 potentially influential variables for effects on nitrogen balance in 20 premature infants receiving intravenous alimentation as sole nutrient source: gest. age,day of life, birth weight, nitrogen intake, total calorie intake, weight for age, length for age and fat intake. Analyses disclosed nitrogen and calorie intake to be the most important independent variables. Their simultaneous consideration yielded a highly significant regression equation: N bal (mg) = 2.13[Kcal/kg] + 0.64 [N intake mg/kg] - 164, $R^2 = 0.86$ ($p < 0.001$). Inclusion of any of the above remaining variables in the equation produced no improvement in estimate. Consideration of 3-methyl-histidine(3MH) output, an index of muscle catabolism improved the estimate of nitrogen balance based on the above two variables yielding another equation: N bal (mg) = 2.08 [Kcal/kg]+ 0.65 [N intake mg/kg] - 13.34 3MH[nmoles/kg]- 131, $R^2 = 0.90$ $p < 0.001$ . The first equation allows one to predict nitrogen balance as a function of two known variables. The second equation permits analysis of nitrogen balance when intake and clinical status vary. The inclusion of 3MH output corrects the regression estimate for catabolism.

**645** MECHANISMS OF SPECIFIC INTERACTION BETWEEN MOUSE ENTEROCYTES AND ROTAVIRUS: IMPLICATIONS IN PATHOGENESIS OF VIRAL ENTERITIS IN HUMAN INFANTS. Marie R. Talty, Ping-C Lee, Patricia J. Carmody, and Pearay L. Ogra. State Univ. of N.Y. at Bflo. and Children's Hospital of Bflo., N.Y.

The nature of virus-intestinal cellular interaction was examined in duodenal and jejunal enterocytes (EC) obtained from groups of adult and suckling mice inoculated orally with mouse rotavirus (MRV). The techniques of immunofluorescence (IF), electron microscopy (EM), and rosetting of MRV-coated sheep erythrocytes (SRBC) were employed for these studies. EM studies of small intestinal homogenates obtained 24 hours after infection revealed the presence of large amounts of MRV in suckling mice, but little or no virus was observed in the adult mice. IF studies demonstrated the presence of viral antigen in the cytoplasm in 15 to 40% of suckling EC and in 0 to 2% of adult EC. Isolated EC from uninfected adult or suckling mice were incubated with purified MRV-coated SRBC's. Specific binding of EC to virus coated SRBC's, as evidenced by formation of rosettes, was observed with 20% of suckling EC and only infrequently with adult EC. These data suggest that the degree of replication of MRV in the intestine may be determined by the availability of virus-specific receptors on EC. The differences in the relative proportion of such receptors between the suckling and adult EC may explain the unique predeliction of infants to rotavirus infection. Although the pathogenesis of MRV closely resembles human rotavirus infection, the identification of similar receptors on human enterocytes remains to be established.

**646** FECAL ALPHA-1-ANTITRYPSIN IN CHILDREN WITH CROHN'S DISEASE. Daniel W. Thomas, Frank R. Sinatra, and Russell J. Merritt (Spon. by Robert M. McAllister), University of Southern California School of Medicine, Children's Hospital of Los Angeles, Department of Pediatrics, Los Angeles.

Random fecal $\alpha$-1-antitrypsin (R-FA1AT) was monitored for 4 to 22 months in 18 children with Crohn's disease aged 7 to 17 years. Excess R-FA1AT indicates abnormal fecal loss of serum protein (Clin. Res. 28 (1):97A, 1980). The relationship between the number of abnormal clinical findings (ACF) and R-FA1AT was evaluated. Eleven parameters were selected to assess disease activity (abdominal pain, weight loss, fever, diarrhea, extra-intestinal symptoms, Hgb, ESR, WBC, albumin, hematochezia, and abdominal mass). Elevated R-FA1AT was associated with high ACF ($\chi^2 p<0.001$). Patients had $5.7\pm1.7$ ACF when R-FA1AT was $\geq 3.4$ (mg/gm dry stool) ($\bar{X}$ of normal + 3SD) compared to $1.4\pm1.0$ when R-FA1AT was <3.4 ($p<0.001$). With treatment ACF fell from $5.9\pm1.3$ to $0.9\pm0.8$ ($p<0.001$), and R-FA1AT decreased from $10.3\pm5.0$ to $1.7\pm0.7$ ($p<0.001$). R-FA1AT became abnormal in 2 patients prior to an increase in ACF upon relapse. Two children still had abnormal R-FA1AT despite declining ACF in early remission. We conclude that R-FA1AT is an objective, sensitive, and noninvasive marker for activity of Crohn's disease.

**647** DELAYED ONTOGENIC DEVELOPMENT IN THE BYPASSED ILEUM OF THE INFANT RAT. K.Kenneth Tsuboi, Linda K.Kwong, W.D. Andrew Ford, Thomas Colby, and Philip Sunshine. Stanford Univ. School of Med., Dept. of Pediatrics, Stanford, CA

Ontogenic development continues after birth in mammalian enteric epithelium as an adaptive mechanism to extrauterine life. Structural, functional and cytokinetic changes of particular prominence occur over a short critical period preparatory to dietary change with weaning. The effect of intraluminal influences on these maturational changes was examined by preparing an experimental surgical bypass in the intestine of the infant rat. Surgical bypass of the terminal one-third length of the small intestine of 12-14 day old suckling rats was performed, maintaining the remaining intestine in continuity (anastomosed to cecum). The bypassed segment, although showing general normal developmental patterns of sucrase and maltase, showed coincident delay in normal reduction of both lactase and enterocyte life-span. The intestine maintained in continuity showed precocious appearance of sucrase and accumulation along with maltase to greater than control levels, accompanied by a normal coincident decline in both lactase and enterocyte life-span. In summary, involvement of intraluminal influences on enteric ontogenic development is expressed by a delay in the excluded segment (bypassed) and by stimulation in the shortened segment in continuity. The data also provide further support of the hypothesis that the life-span of the enterocyte serves as a primary determinant postnatally of enteric lactase levels.

**648** RELATIONSHIP BETWEEN SERUM CONCENTRATION AND URINARY EXCRETION OF ZN AND CU IN PREMATURE INFANTS. E.E. Tyrala, J.I. Manser, N.L. Brodsky. Temple University School of Medicine, St. Christopher's Hospital for Children, and Albert Einstein Medical Center (North), Philadelphia, Pa. (Spons. by V.H. Auerbach)

Serum zinc (Zn), copper (Cu), and 24 hour urine collections for Zn, Cu, and amino acid excretion were measured in 12 primarily intravenously nourished premature infants (mean GA=30.6 wks., mean birth wt=1067gms.). Five infants had two or more serial measurements. All infants were clinically stable, non post-op, and not infected at times of study. Mean age at time of first study = 3.3 wks. Serum Zn and 24 hr. urinary Zn excretion were positively correlated ($r=.74$ $p<.0006$). Infants with hypozincemia (serum Zn less than 70µg/dl) generally had urine Zn losses of <130µg/24 hr. Urine Zn concentrations of <100µg/24hr., with a serum Zn of greater than 70µg/dl was an indication of impending hypozincemia. The premature infant is capable of renal conservation of Zn under conditions of actual and impending hypozincemia.

Serum Cu and 24hr. urinary Cu excretion were not related ($p=.17$). Urinary copper excretion, previously shown to be related to increased urinary excretion of glycine and methionine during therapy with Freamine II was markedly reduced with the use of Freamine III and the concommitant reduction in glycine and methionine excretion. Urine Cu excretion averaged <1µg/24hr. and did not contribute significantly to Cu losses.

**649** Hypozincemia in Growing Premature Singleton and Twin Infants. E.E. Tyrala, J.I. Manser, N.L. Brodsky, N. Tran, (Spons. by V.H. Auerbach), Temple University School of Medicine, St. Christopher's Hospital for Children, Albert Einstein Medical Center (North), Philadelphia, Pa.

Serial serum zinc (Zn) concentrations were measured from birth to a mean age of 16 weeks in 17 well, AGA, singleton, premature infants (mean GA=30 wks., mean B.W.=1253gms.) and compared to serum Zn concentrations in 4 sets of premature twins (mean GA=30 wks., mean individual B.W.=1104gms.) who were followed to a mean age of 18 weeks. All infants were fed standard infant formulas. Average weekly daily weight gain, linear growth, caloric, protein, and Zn intake were not significantly different between the two groups. Serum Zn concentrations measured in the first 72 hrs. of life averaged 170µg/dl in the singleton infants and 144µg/dl in the twins ($p=.3$).

| Week | | 1 | 2 | 3 | 4 | 5 | 6 | 7-9 | 10-12 | 13-18 | 19+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Zn | Singl. | 118 | 86 | 82 | 74 | 65 | 58 | 55 | 62 | 71 | 70 |
| µg/dl | Twins | 132 | 79 | 82 | 65 | 48 | 45 | 49 | 41 | 57 | 61 |
| | | ns | ns | ns | ns | p= <.01 | p= <.05 | ns | p= <.01 | p= <.05 | p= .16 |

Hypozincemia (serum Zn <70µg/dl) is common in the growing premature infant. The premature twin is at particular risk, however, for the development of marked hypozincemia (serum Zn <50µg /dl) during the first 5 months of life. Inadequate Zn content of infant formulas, poor Zn absorption and/or decreased body stores may all be contributory.