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**ANTIGEN ABSORPTION IS ENHANCED DURING VIRAL ENTERITIS**  
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We examined the absorption of bovine serum albumin from the intestines of 2 week old piglets before and during the course of experimentally induced transmissible gastroenteritis virus infection. Blood levels of immunoreactive bovine serum albumin were measured before and 4 hours after administration of 1 gram per kilogram of bovine serum albumin through a surgically implanted jejunal catheter. The apparent permeability to the albumin was elevated during both the early invasive phase 12 hours post inoculation with virus, and the diarrheal phase 84 hours post inoculation. In some animals, the elevation persisted into the recovery phase 336 hours post inoculation. The absorbed immunoreactive bovine serum albumin moved with the 70,000 molecular weight fraction on gel chromatography. Once absorbed, the albumin was cleared very slowly from the infected animals' circulation, with a half life of 8-15 days. We conclude that antigen absorption is enhanced during viral enteritis. We speculate that immune responses to this absorbed antigen might serve to prolong the disease state.

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**MIXING CARBOHYDRATES(CHO) - ADDITIVE AND COMPETITIVE EFFECTS ON ABSORPTION.** Benny Kerzner, Howard R. Sloan, Gary Birken, Anton H. Ailabouni, H. Juhling McClung, Ohio State Univ. Coll. of Medicine, Dept. Peds, Columbus, OH.

When intestinal absorptive function is limited, it may be possible to enhance CHO absorption by combining CHOs having different digestive-absorptive pathways. Employing single pass perfusion studies in partially atrophic amylase-free canine jejunal Thiry-Vella fistulae we found that absorption of glucose(G), sucrose(S), and long chain glucose polymers (LCGP) was saturable with infusion conc. of 882, 378, and 347 mg/dl respectively. Absorption of S and LCGP was additive even from solutions containing 900 mg/dl of each sugar ( $S=159\pm 16$ ,  $LCGP=70\pm 6$ , mixture= $235\pm 22$   $\mu\text{gms/cm}^2/\text{min}$ ). Virtually no G was found in the fistula effluents ( $<0.1$  mg/ml). Adding 900 mg S to 900 mg LCGP did not improve CHO absorption beyond values for 900 mg/dl of G infused alone ( $378\pm 16$   $\mu\text{gms/cm}^2/\text{min}$ ). Addition of the  $V_{\text{max}}$  conc. (400 mg/dl) of LCGP to the  $V_{\text{max}}$  conc. of G (900 mg/dl) causes mutual inhibition of the absorption of both (G,  $346\pm 23$  vs.  $232\pm 25$ ; LCGP,  $63\pm 8$  vs.  $28$ ;  $p<.001$ ). Additive absorption occurs with addition of 400 mg/dl LCGP to 500 mg/dl of G ( $LCGP=56\pm 8$ ,  $G=152\pm 8$ , mixture= $208\pm 10$ ). **Conclusions:** 1) For G polymers and S, hydrolysis, not absorption of the resultant monosaccharides, is rate limiting for jejunal assimilation. 2) G and LCGP either interact in the glycocalyx or compete for absorptive pathways. 3) S does not compete with LCGP for absorption. **Speculation:** In formulating enteral feedings or rehydration solutions mixtures of S and LCGP may enhance absorption, but addition of LCGP to G may be deleterious.

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**INHIBITION OF MALTOPENTAOSE (MP) ABSORPTION BY MEDIUM AND LONG CHAIN ELEMENTS IN CORN STARCH HYDROLYSATE (CSH).** Benny Kerzner, Howard R. Sloan, Anton Ailabouni, Constance Seckel, Gary Birken, H. Juhling McClung, Ohio State Univ. College of Medicine, Dept. Peds., Columbus, OH.

To define optimal carbohydrates for infants with limited pancreatic and intestinal mucosal function we synthesized and evaluated the absorption of  $^{14}\text{C}$ -MP - a glucose oligomer ideally suited to glucoamylase activity. Representative medium (degree of polymerization, DP, 4-9) and long chain (DP $\geq 75$ ) polymers were isolated from a CSH. Using partially atrophic, pancreatic amylase-free, canine, jejunal, Thiry-Vella fistulae, absorption of DP 5 was measured: alone, in the presence of 90, 180, 270, 360 and 450 mg/dl of DP 4-8, with similar concentrations of DP $\geq 75$  and with 450 mg/dl of sucrose. **Results:** Absorption of DP 4-8 was greater than sucrose which exceeds DP $\geq 75$ . Although at 450 mg/dl there was equivalent inhibition of DP 5 absorption by DP 4-8 (46%) and DP $\geq 75$  (45%), at 90 mg/dl, inhibition by DP 4-8 was significantly less than by DP 75 (10 vs. 27%,  $p<.01$ ). Sucrose did not inhibit DP 5 absorption ( $p<.001$ ). **Conclusions:** 1) Although assimilation of glucose polymers is less efficient than shorter oligomers, both inhibit DP 5 absorption. 2) At low concentrations the greater inhibition by DP $\geq 75$  probably reflects a protracted affinity for the glucoamylase enzyme. 3) Sucrose does not compete for oligomer assimilation. **Speculation:** These results suggest that substitution of sucrose for a portion of the polymers in a CSH, especially the long chain elements, will improve carbohydrate absorption.

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**PROSPECTIVE HIGH VS LOW CALCIUM AND PHOSPHORUS WITH FIXED LOW VITAMIN D CONTENT IN PARENTERAL NUTRITION (PN) IN INFANCY.** Winston W.K. Koo, Reginald C. Tsang, Jean Steichen, Michael Farrell, John Noseworthy, Paul Succop, U.C.

Vit D 50 to 1000 IU/d in PN has been suggested for infants but prospective studies are lacking. Effects of high or low Ca and P on Ca metabolism have not been examined. We hypothesized that high Ca and P with low vit D in PN results in normal Ca, P and vit D metabolism in infants. 12 wt appropriate for gestation infants (3M,9F; 11W,1B); 32-41 wks; 1780-3630g at birth with surgical indications for PN received fixed 'low' 25 IU vit D<sub>2</sub>/dl and were randomized to either high Ca 60mg and P 46.5mg/dl, or low Ca 20mg and P 15.5mg/dl content. Measures were made at start, 10d, 3wks and 6wks. There were no differences in serum Ca, Mg, P, alkaline phosphatase, parathyroid hormone, calcitonin, and values were normal. Mean serum 25 hydroxyvit D (25OHD, protein binding) was 16-39 for high and 15-31ng/ml for low Ca,P grp. Mean serum 1,25 dihydroxyvitamin D (1,25(OH)<sub>2</sub>D, HPLC-binding) and tubular reabsorption P (TRP) in high Ca,P grp were stable 72-47pg/ml, and, 88-80% resp; values in low Ca,P grp were higher 78-127pg/ml and 75-99% ( $p<0.05$  covariance, high v low). Urine Ca and Mg/Creatinine were not different between grps. 5 infants on PN for 71-175d with same vit and low Ca,P had normal 25OHD with high TRP and 1,25(OH)<sub>2</sub>D. Thus, PN with 1) low 25IU vit D/dl maintains normal serum 25OHD for up to 6mos 2) high Ca,P similar to that absorbed from human milk results in stable serum 1,25(OH)<sub>2</sub>D and TRP. We speculate that low Ca, P intake results in adaptive increase in 1,25(OH)<sub>2</sub>D and TRP reflecting insufficient Ca and P intake; and high Ca and P may benefit bone mineralization in long term PN.

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**NATURAL HISTORY OF RICKETS AND FRACTURES IN VERY LOW BIRTH WEIGHT (VLBW) INFANTS DURING INFANCY.** Winston W.K. Koo, Alan Oestreich, Reginald C. Tsang, Roberta Sherman and Jean Steichen, University of Cincinnati Medical Center.

Radiographic bone demineralization (BD), rickets (R) and fractures (F) have been noted anecdotally in VLBW infants but there is no prospective study of the course and outcome of the skeletal pathology. We hypothesized that BD, R and F are frequent in VLBW infants, but the lesions are self resolving with time. X-rays from 60 VLBW (birth wt  $<1500$ g) infants were taken prospectively. Birth wts were 580-1500g, gestation 24-34wks; 32B, 28W; 54 AGA and 6 SGA. Single view x-rays were taken of both forearms at 3, 6, 9 and 12mos. Other x-rays taken for clinical reasons also were reviewed. BD, R and F were determined using standard criteria. 'Mild' BD was regarded as normal. From 40 infants who completed the study BD occurred in 22.5%, R in 17.5%, F in 20%. BD was noted in all infants with R or F. All infants with R had F but only one infant had F without R. All abnormal x-rays were noted by 6mos, 75% by 3mos and one infant by 7wks. F occurred predominantly in extremities (8 radius and/or ulna, 3 humerus, 3 femur), 5 ribs; unusual sites included 1 scapula; 5 had multiple F. Prior to radiographic changes, 60% received parenteral nutrition (calcium 20mg, phosphorus 15.5mg/dl, vit D<sub>2</sub> 20IU/kg/d); 70% received own mother's milk or standard 20kcal/oz formula; 30% received high calcium and phosphorus formulas in varying quantities. By 9mos all F and R healed regardless of mode of therapy or diet. By 12mos, 1 infant only still had BD. We conclude 1) BD, R and F are present in 1 in 5 of VLBW infants; 2) infants with rickets are likely to have fractures; 3) if one fracture is noted other fractures are likely. Until definitive preventive measures become available, we suggest care be taken during physical manipulation of these infants.

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**PANCREATIC FLUID SECRETION IN CYSTIC FIBROSIS (CF).** H. Kopelman, M. Corey, K. Gaskin, P. Durie, and G. Forstner, Hospital for Sick Children, GI Div., Dept. Ped. and Kinsmen CF Res. Center, Toronto. (Sponsored by Dr. K. N. Drummond, Montreal Children's Hospital, Dept. Ped., Montreal).

The cause of exocrine duct obstruction in CF is uncertain. To evaluate the role of impaired fluid secretion in the pathogenesis of pancreatic duct obstruction, we examined the relation of fluid secretion to protein output in CF and in control pts. with equivalent pancreatic function (trypsin output). A quantitative marker perfusion technique was used for collection of duodenal contents during pancreatic stimulation with secretin and CCK.

Water secretion in 55 CF pts. ( $3.15\pm 2.58$  ml/kg/hr) was less than in 57 non-CF controls ( $10.48\pm 5.23$  ml/kg/hr,  $p<0.0001$ ). Flow was correlated with trypsin output in CF ( $r=0.66$ ,  $p<0.0001$ ) and non-CF ( $r=0.51$ ,  $p<0.0001$ ) pts. By analysis of covariance, the slopes were identical but the CF adjusted mean flow, correcting for trypsin output, was 41% of the non-CF value. Water secretion correlated with HCO<sub>3</sub> output in CF ( $r=0.77$ ,  $p<0.0001$ ) and non-CF ( $r=0.67$ ,  $p<0.0001$ ) pts. Protein output was independent of flow in 21 non-CF pts. ( $r=0.22$ ,  $p>0.1$ ) but was related to H<sub>2</sub>O secretion ( $r=0.84$ ,  $p<0.001$ ) in 28 CF pts. By multiple linear regression analyses, the effect of flow on protein output was independent of trypsin.

**Conclusions:** 1. Flow is diminished in CF independent of pancreatic acinar impairment. 2. Defective HCO<sub>3</sub> output may account for the defect in fluid secretion. 3. Protein output in CF is dependent on flow. The data suggest that impaired flow in CF leads to protein stasis within ducts.