• 691

DEMONSTRATION OF DIRECT GLUCOSE ABSORPTION THROUGH INFANT RECTAL MUCOSA

Carlos H. Lifschitz, Charles S. Irving, Lisa M. Marks, Peter D. Klein, Buford L. Nichols. Baylor College of Medicine, USDA/ARS Children Nutrition Research Center, Department of Pediatrics, Houston, TX. Klein, and

Previous studies of the fate of malabsorbed glucose in the colon provided kinetic evidence that glucose can be transported through the rectal mucosa of the infant without being broken down to volatile fatty rectal mucosa of the infant without being broken down to volatile fatty acids. To obtain evidence for the direct absorption of glucose across rectal mucosa <u>in vivo</u>, UL-¹³C₆-glucose was administered rectally through a small catheter to 3 healthy newborn infants within the first hour of life and to 3 infants recovering from diarchea. Three samples of peripheral blood were drawn between 5 and 30 min following the administration of the isotope. Glucose was isolated from deproteinized plasma and converted to its aldonitrile penta-acetate derivative with hydroxylamine hydrochloride, and analyzed by gas chromatography/mass spectrometry for the presence of di-, tri-, tetra- and hexa-¹³C-labeled glucose molecules. Maximum ratios of hexa-¹³C-glucose to endogenous plasma glucose in the range of .3-.5% were detected in 2/3 of newborns and 2/3 of infants with diarchea. These levels exceeded three times the standard deviation of the isotopic ratiometric measurement. Statistically standard deviation of the isotopic ratiometric measurement. Statistically significant levels of the di-, tri-, and tetra-1³C-labeled glucose were not found when glucose was administered rectally. However, maximum mole ratios of 1.7, 0.8, and 0.3% were observed for hexa-, tetra-, and tri- ^{13}C -labeled glucose, respectively, after IV administration. Conclusion: These data provide the first direct evidence for the systemic availability of intact glucose molecules absorbed from the human rectum.

692 GLIADIN ANTIBODIES (GA) IN PEDIATRIC GAS-TROINTESTINAL DISORDERS. John D. Lloyd-still, Carlos M. Arroyave, Jan Jacobitz, Dept. of Peds., Children's Memorial Hospital, Chicago Measurement of gliadin antibodies (IgA and IgG) have been advocated for the screening and follow up of Celiac disease (J Pediatr 1983, 102:655, J Pediatr Gastro Nutr 1984; 3:205). We measured IgA and IgG gliadin antibody by ELISA in 26 age matched non atopic, non allergic controls, and disease controls including Crohn's, ulcerative colitis and cystic fi-brosis (CF) patients. Children undergoing D-xylose absorption tests (5 gm. p.o.) for investigation of malabsorption were prospectively studied for IgA and IgG gliadin antibodies during a 12 month period. The data was analyzed for IgA and IgG gliadin antibody levels, diagnosis, age and D-xylose values. Celiac Enteritis U. Colitis Crohn's Mise. CF Controls

		<u>Celiac</u>	Enteritis	U. Colitis	Crohn'	s Misc.	<u>CF</u>	Contro1	s
	(N)	6	28	8	14	13	26	25	
IgA		100	25	0	0	7.7	15.4	0	
IgG	(%)	100	78.6	12.5	28.6	69.2	65.4	8	
Conclusions:			l) IgA	and IgG-G	GA are	raise	1 in	100%	of

f active Celiacs. 2) IgA-GA are more specific than IgG-GA. 3) These elevations appear non-specific. 4) Xylose absorption correlated poorly with IgA and IgG-GA.

HEXOSAMINIDASE: A MARKER FOR HEALING AFTER ISCHEMIC • 693 GUT INJURY. Thom E Lobe, Eric D. Dobkin, David K. Rassin, William K. Gourley, Keith T. Oldham, Jatinder Bhatia. University of Texas Medical Branch, Departments of

Surgery, Pediatrics, and Pathology, Galveston, TX. Serum hexosaminidase activity (HEX) is elevated with ischemic gut injury. To determine if subsequent decreases in HEX correlate with gut healing, 97 weanling rats were subjected to laparotomy at which alternate vascular bundles were ligated along the base at which alternate vascular bundles were ligated along the base of the entire anterior mesenteric artery arcade. Fifteen rats served as pre-operative controls. After recovery, rats were allowed ad lib food and water. Groups were then killed at inter-vals, blood was drawn for HEX determination, and samples of small bowel were taken for histological evaluation. DATA: TIME (hrs) Control 6 12 24 36 48 72 15 9 9 9 HEX Mean 1192 940* 1258 1362* 1363* 1776* 1421* (SD) (158) (130) (192)(154) (102) (511) (194)

TIME	(days	s) <u>4</u>	5	6	8	10	12	15
N =		9	9	5	5	5	3	4
HEX	Mean	1392*	1399*	1503*	1275	996*	1305	1200
	(SD)	(120)	(139)	(96)	(65)	(111)	(18)	(137)
HEX	= total	activity	nmol/hr.m	1; *p<	0.01 V	s contro	1.	

Microscopically, focal ischemic necrosis began at 6 hours. Be-tween 12 and 48 hrs, cellular changes were consistent with pro-gressive ischemic injury. Evidence of healing was apparent beginning at 5 days and these histological changes correlated with changes in HEX. Thus HEX proves useful as a marker for bealing wit in this model. healing gut in this model.

PELVIC FLOOR DYSFUNCTION IN CHILDREN WITH CHRONIC CONSTIPATION. Vera A. Loening-Baucke (Sponsored by Robert Thompson). University of Iowa, Department of 694

Pediatrics, Iowa City, IA. We have previously shown that some children with chronic con-We have previously shown that some children with chronic con-stipation and encopresis (P) have impairment of rectal sensation. 23 consecutive P (constipation \geq 1 year) and 8 healthy controls (H), 4-13 yr old, were asked to defecate water-filled balloons containing 30, 50 and 100 ml water, while sitting on a toilet chair. 12 P (Pdef+) and all H defecated all 3 balloons, whereas 11 P (Pdef-) defecated 1 or no balloons. We evaluated EMG activi-ty of the external anal sphincter/pelvic floor during defecation while lying down. The mean anal multiproved pressure (mm Hg) durwhile lying down. The mean anal pullthrough pressure (mm Hg) dur-ing rest (APr) and during squeeze (APsq) were obtained using intraluminal transducers. Threshold of rectal sensation (Tsens), threshold of the rectosphincteric reflex (TRSR), and the rectal volume (ml of air) which produced an urge to defecate (CV) were measured by distending a rectal balloon. All H had decreased EMG activity during trials to defecate. The pelvic floor showed in-creased EMG activity in 17% Pdef+ compared to 82% Pdef- (P<0.03). APr APsq Tsens TRSR CV $\begin{array}{r} \text{APsq} \\ 228 + 54 \\ 227 + 42 \\ 222 + 35 \end{array}$ TRSR CV H 118 + 19 228 + 54 12 + 6 9 + 7 90 + 16 Pdef+ 108 + 15 227 + 42 21 + 10 15 + 5 185 + 107 Pdef- 109 + 20 222 + 35 28 + 13 15 + 6 207 + 87 APr and APsq were not different in the 3 groups. Tsens, TRSR and CV were significantly increased in both constipated groups when compared to H (p(0.05)). This study indicates that 48% of P have a $\begin{array}{r}
118 + 19 \\
108 + 15 \\
109 + 20
\end{array}$ during attempted defecation.

Serial whole blood B₆ vitamer levels in breast, for-mula and parenterally fed premature infants. Ernest **695**

OYD mila and parenterallý fed premature infants. Ernest E. McCoy, Ken Strynadka, David Schiff & Ann Cornet. Univ. of Alberta School of Med., Dept. Pediatrics, Edmonton, Alta. To assess adequacy of vitamin B₆ intake in premature infants, serial blood samples were obtained for vit B₆ determination. A 0.5ml sample was frozen, B₆ vitamers extracted, separated and quantitated by a HPLC-fluorometric technique. The method measur-ed pyridoxal phosphate (PLP), pyridoxamine phosphate (PMP), pyri-doxal (PL), pyridoxine (PN) and pyridoxamine (PM). Multiple blood samples were obtained over a 2-8wk period. Most infants receiving breast milk had low levels of PLP but bich PL resulted receiving breast milk had low levels of PLP but high PL resulted in total normal B levels but some had low total vitamin B. Several infants receiving formula had marked elevations of ⁶PL and PLP -- PL 256ng/ml (Normal=2-8) and PLP 120ng/ml (Normal 6-15). The values decreased to normal with continued feeding. Five infants received total parenteral nutrition (TPN) using commerciinfants received total parenteral nutrition (TPN) using commercial solutions for varying causes for 2-6wk period. PL and PLP values increased. Infant I -- PL 941ng/ml whole blood; PLP 293ng/ml. Infant II PL 765ng/ml, PLP 85ng/ml. Infant III PL & PLP beyond measurement. PL & PLP remained high until TPN was terminated. Conclusion: 1) Breastfed infants may have low whole blood PLP levels. 2) Some premature infants receiving proprietary formula have very high blood levels of PL and PLP suggesting an immaturity of B, degradation. 3) Premature infants receiving TNP develop very high levels of FL and PLP. Commonly used multivitamin solutions used in TPN have excessive amounts of B, for premature infants. for premature infants.

ESTABLISHMENT OF AN ANIMAL MODEL OF OVALBUMIN - SENSITIZED MOUSE TO STUDY PROTEIN INTOLERANCE IN VITRO . Christiane Malo t 696 Université de Montréal, Montréal, Canada.

Université de Montréal, Montréal, Canada. Protein intolerance, such as cow's milk allergy, represents a common cause of enteropathy in infancy. However, no reliable laboratory test for the diagnosis is presently available. An antmal model of ovalbumin (OVA) - sen-sitized mouse has been established to study the response of the small intesti-nal mucosa in organ culture. A decrease of Y-glutamyltranspeptidase (Y-GT), alkaline phosphatase (AlPase), sucrase (S), lactase (L) and glucoamylase (GA) activities was observed in the explants (T) cultured during 24 hours in presence of OVA. In contrast, a large increase of these enzymatic activities was noted in the culture media (M), the overall effect observed beeing a net stimulation of the total enzymatic activities of the culture system. During the same period, a 20% decrease in ³H-thymldine incorporation into DNA was noted (P < .005). noted (P < .005).

 γ -GT AlPase
 Tissue
 98.1 ± 7.3
 97.9 ± 10.6
 98.1 ± 10.6
 82.4 ± 5.6
 87.7 ± 10.7

 Medium
 147.0 ± 8.4
 144.5 ± 7.6
 124.7 ± 5.9
 168.7 ± 27.2
 119.9 ± 5.8

 T + M
 132.2 ± 4.9
 133.0 ± 4.4
 114.9 ± 3.0
 135.7 ± 14.1
 115.3 ± 4.7
 Results (X ± S.E.M.) from 12 experiments are expressed as % of activity with respect to autocontrols cultured in presence of β -lactoglobulin. The enzymes accumulated mainly in the particulate fraction of the culture

medium (brush border membrane fraction) suggesting an increased turn-over of some membrane components by a process of shedding or microvesiculation. This animal model represents a useful tool to evaluate the modifications of the small bowel mucosa induced by an allergen and to establish the criteria for an in vitro diagnosis of protein intolerance.

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