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ERYTHROCYTE 2,3 DIPHOSPHOGLYCERATE (2,3 DPG), A MARKER FOR EARLY DETECTION OF RICKETS. Keith S. Kanarek, Ted A. Tedesco, John S. Curran, C. Brown and Paul R.

Williams (Spon. by Lewis A. Barnes) Department of Pediatrics, University of South Florida College of Medicine, Tampa, Florida.

The hypothesis that erythrocyte 2,3 DPG content may serve as an important marker for early detection of nutritional hypophosphatemic rickets in the premature infant was examined in 12 AGA infants X B. Wt. 1163±83 grams (S.E.M.) for a X of 64 days (range 33-99). All infants were fed a commercial 18:82 formula containing 510 mgms. calcium and 390 mgms. phosphate per liter. Patients entered the study when 100 k cal/kg/day were tolerated enterally and the following plasma and erythrocyte determinations were performed.

Study	Initial	Mid Point (each pt.)	Discharge
2,3 DPG nmoles/ml RBC (x±SEM)	4.7±0.6	5.0±0.3	6.1±0.6
Calcium mgm/dl (x±SEM)	9.5±0.2	9.2±0.3	9.6±0.2
Phosphate mgm/dl (x±SEM)	6.0±0.4	7.0±0.4	7.0±0.3
Alkaline phosphatase U/L (x±SEM)	392±52	445±39	429±40
Weight (gms.) (x±SEM)	1163±83	1430±65	1975±23

No significant differences in erythrocyte 2,3 DPG content, calcium, nor phosphate were noted. No infants demonstrated biochemical, clinical nor radiologic evidence of nutritional hypophosphatemic rickets.

These data suggest that the phosphorus balance in infants fed a commercially available formula in the absence of nutritional hypophosphatemic rickets prevents reduction in erythrocyte 2,3 DPG.

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OSTEOPENIA, HYPERCALCIURIA, AND RENAL CALCULI IN A PATIENT RECEIVING PROLONGED TOTAL PARENTERAL NUTRITION. Lyndon Key, Constantine Anast, James Sutphen, Pat Queen, and Aubrey Katz. Harvard Medical School, Children's Hospital Medical Center, Boston, Mass.

A 7.1 kg infant, age 7m, was maintained on TPN (Ca 261mg/24h, P 124mg/24h, D 400 IU/24h), and developed renal calculi and osteopenia. The urinary Ca/creatinine (Ca/Cr) was 1.0 (nl<0.3). The serum Ca was 9.6mg/dl; the P, 2.5mg/dl. Because of the hypophosphatemia, the concentration of P in the TPN fluid was increased to deliver 240mg/24h. After a transient increase in urinary Ca and P, the Ca/Cr decreased to 0.1. However, the serum P increased to only 3.1mg/dl. D metabolites were: 1,25(OH)₂D 100pg/ml (nl 40±10); 24,25(OH)₂D <1.0ng/ml (nl 2.4±1.1); 25(OH)D 35ng/ml (nl 21±9.4). Following the increase in P concentration in TPN, the renal calculi resolved.

In contrast to other reports of TPN, the osteopenia cannot be attributed to reduced serum 1,25(OH)₂D in this patient. The role of the reduced serum 24,25(OH)₂D in the bone abnormality deserves consideration since there is evidence that this metabolite exerts an effect on bone formation. The elevated serum 1,25(OH)₂D and the depressed serum 24,25(OH)₂D may be secondary to the hypophosphatemia. This suggests the possibility that the abnormal levels of circulating vitamin D metabolites, rather than causing the bone disease, are a physiologic response to the macronutrient status produced by prolonged TPN. These results suggest that modification of macronutrient composition of TPN fluid may be necessary to prevent metabolic bone disease.

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DEFECTIVE ENTEROCYTE GLUCOSE TRANSPORT IN ACUTE VIRAL ENTERITIS. David Keljo, Mary Perdue, Joseph Telch, Ross Shepherd, Dan Butler, Grant Gall, and Richard

Hamilton, Dept. Pediatrics, University of Toronto, Research Institute, Hospital for Sick Children, Toronto, Ont., Canada.

We measured glucose transport in piglets with acute (40 h), experimentally induced transmissible gastroenteritis (TGE), an invasive viral enteritis closely resembling human rotavirus enteritis. Marker perfusion studies, *in vivo*, showed rising net glucose absorption as perfusate glucose increased in controls; in TGE, net glucose flux was reduced and didn't respond to increasing perfusate glucose. Net 3-0-methyl glucose flux was similarly reduced in TGE tissue compared with controls in Ussing chambers.

net flux - glucose (nmol cm ⁻² hr ⁻¹)	Marker Perfusion		Ussing Chamber (2h ⁻¹)	
	30 mmol/l*	60 mmol/l*	basal	phlorizin
control	0.04±0.01	0.11±0.2	0.18±0.02	0.45±0.08
TGE-40h	0.01±0.01**	0.01±0.01**	0.01±0.01**	0.03±0.02**
				-0.05±0.01

(* perfusate glucose; ** p < 0.001 compared with controls)

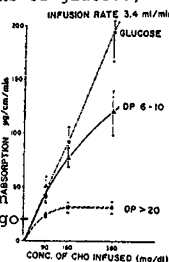
In brush border membrane (BBM) vesicles, isolated from control piglet jejunum, we found sodium gradient-driven, active D-glucose uptake and Na⁺ dependent, glucose inhibitable, high affinity phlorizin binding. In BBM vesicles from TGE pigs we found little or no active glucose uptake and no specific phlorizin binding. We conclude that loss or dysfunction of the BBM Na-dependent glucose carrier contributes to defective glucose absorption in this viral enteritis.

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JEJUNAL ABSORPTION OF SHORT AND LONG CHAIN GLUCOSE OLIGOMERS (GO) IN THE ABSENCE OF PANCREATIC AMYLASE (PA). Benny Kerzner, Howard R. Sloan, H. Juhling

McClung, Christopher C. Chidi. Ohio St. Univ. Coll. of Med., Cols. Children's Hosp., Dept. Peds., Cols., OH.

GO digestion in the newborn and patients with pancreatic insufficiency may depend on brush border glucoamylase, whose efficacy against long chain substrates has not been fully defined. We therefore characterized the oligomeric profile of a starch hydrolyzate (Polycose®) and isolated from it two major fractions (Fns):FnI, a theoretically optimal substrate for glucoamylase, contains GO with degrees of polymerization (DP) 6-10;FnII represents the long chain GO with DP>20 (DP_{AVG}=23 by mol. wt. analysis). Canine Thiry-Vella fistulae, proven to be free of pancreatic and bacterial amylase, were perfused at 3.7, 1.9, and 0.4 ml/min with 90, 180, and 360 mg/dl isotonic solutions of glucose, FnI and FnII. Glucose absorption exceeds FnI at 180 and 360 mg/dl (p<.05, p<.01);FnI absorption markedly exceeds that of FnII (p<.001). Decreasing the perfusion rate increases the absorption of all Fns but significant differences persist between them. Glucose absorption is linear with concentration at all perfusion rates;FnI is linear at 1.9 and 0.4 ml/min;FnII is never linear and reaches a "V_{MAX}" of 31 µg/cm/min at 3.7 ml/min. Conclusion: Limited absorption of long chain GO by PA-free jejunum suggests that an ideal oligomeric substrate should contain GO with DP<10.



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PLASMA SUBSTRATE PROFILE OF VERY LOW BIRTH WEIGHT (VLBW) INFANTS DURING DIFFERENT NUTRIENT REGIMENS. Katherine C. King, Kou-Yi Tseng, Eeva-Liisa Miettinen, & Satish

G. Kalhan. Case Western Res. Univ. at Cleve. Metro. Gen. Hosp., Cleveland, Ohio.

Plasma substrate response and lipid tolerance were examined in VLBW infants during parenteral alimentation (PA) with or without intralipid (IL) infusions. 21 infants, age 2-12 days (d) received PA only; 30 infants, age 9-92 d received PA + IL and 25 infants, age 8-111 d received formula or breast milk (F/B). Plasma concentrations of glucose (G) triglyceride (TG), free fatty acids (FFA), and FFA composition were determined at weekly intervals during PA infusion; within or 4 hrs of cessation of IL; or immediately before next oral feed. Calorie intake ranged from 60-120 Kcal/Kg.d.

	G mg/dl	TG mg/dl	FFA µm/L	C18:2+C18:3 (%)
PA	108±11*	47± 7	279± 23	3.9±0.9
PA+IL<4hr	77± 5	98±14	1160±228	40.5±3.2
PA+IL>4hr	81± 5	35± 3	413± 50	20.1±2.4
F/B*(Mean±SEM)	8± 3	71± 5	450± 30	44.4±1.8

PA alone resulted in significantly lower TG, FFA, & essential fatty acids C18:2 & C18:3. 10/21 infants had no detectable C18:2 & C18:3, youngest being age 4 d. TG and FFA were higher in IL infusion group, and assimilation of both TG & FFA were rapid with significant drop occurring 4 hrs post IL infusion. Concl. (1) Provision of essential FA may be necessary within one week for VLBW infants receiving PA alone. (2) Assimilation of IL is enhanced by concurrent PA infusion. This enhancement may be the result of endogenous insulin response to glucose-amino acid infusions.

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LIPID INTAKE DURING THE SUCKLING PERIOD REGULATES GLUCOSE METABOLISM IN THE DEVELOPING RAT INTESTINE. Robert E. Kimura, Gunilla Thulin, Joseph B. Warshaw,

Yale Univ. Sch. of Med., Dept. of Ped., New Haven, CT.

The oxidation of glucose in developing rat intestine changes dramatically during suckling and weaning. The rate of glucose oxidation to CO₂ by intestinal slices increased 3-fold after weaning and subsequently declined by half to an adult level. The steady state concentration of pyruvate decreased from 45 µM in suckling animals to 20 µM after weaning suggesting a change in pyruvate metabolism during this period without a change in lactate production. Studies with 1-¹⁴C-pyruvate suggested increased pyruvate dehydrogenase (PDH) activity after weaning; production of ¹⁴CO₂ increased from 3.52 to 4.48 n moles/mg/hr after weaning. High levels of fatty acid esters present during the suckling period may be responsible for PDH inhibition. To investigate this possibility, palmitoylcarnitine (400 µM) was added to assays using intestinal slices from post-weaning animals. This resulted in a decrease in glucose oxidation from 2.23 to 1.35 n moles/mg/hr while steady state levels of pyruvate increased from 28 to 42 µM. Oxidation of 1-¹⁴C-pyruvate to ¹⁴CO₂ decreased from 4.32 to 2.38 n moles/mg/hr, again indicating decreased PDH activity. These results mimic those found in tissues from suckling animals. These studies demonstrate an influence of fatty acids on intestinal glucose metabolism during development probably at the level of PDH and emphasize the importance of diet in regulating intestinal metabolism.