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QUANTIFICATION OF GLUCOSE POLYMER (GP) DIGESTION BY PREMATURE SALIVARY AMYLASE (SA). Robert D. Murray, Benny Kerzner, Howard R. Sloan, H. Juhling McClung, Merry Gilbert, Anton H. Ailabouni. Ohio State Univ. Coll. of Medicine, Dept. Peds., Columbus Children's Hospital, Columbus OH.

Since pancreatic amylase (PA) is absent in prematures, GP hydrolysis depends on alternate enzymes. To assess premature SA as a PA surrogate we evaluated its production, acid resistance and hydrolytic potency in a simulated oro-pharyngeal (OP), gastric (G) and intestinal (I) environment. SA was added to yield 1.1 u/ml of a "modular" formula containing 7 gm/dl of ^{14}C GP with degrees of polymerization (DP) 18-29 glucose units. After an 0.25 min. OP phase the G-phase was initiated by dilution with pepsin/HCl to yield pHs of 2, 3, 4, or 5. After 5 min. or 3 hours pH was adjusted to 7.0 with cholate/trypsin chymotrypsin/Na_2CO_3 (I phase). After the OP- and G- phases and 15, 60 and 180 min. ^{3}of I-phase, oligosaccharides were separated by thin layer chromatography. % breakdown=cpm in DP 1-9 / cpm in total sample x 100. Results: In 11 prematures SA activity was 1-33 u/ml; isozyme profile and acid resistance were identical to adult SA. Substantial G-phase breakdown only occurred with 3 hour exposure at pH 4 (12%) and 5 (32%). In the I-phase SA activity resumed. Prior G-phase pH affects ultimate I-phase breakdown, p<.001. (After 5 min. G-phase at pH 2&5, I-phase breakdown = 8&25%; after 3 hour G-phase at pH 2&5, I-phase = 17&55%.) Conclusion: The limited SA of premature saliva can produce significant GP digestion in both the stomach and small intestine.

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ADMINISTRATION OF LIPID IMPROVES NITROGEN RETENTION IN CHILDREN RECEIVING ISOCALORIC TPN. Osamu Nose, James R. Tipton, and Marvin E. Ament. Osaka University, Osaka University Hospital, Department of Pediatrics, Osaka, Japan and University of California, UCLA Hospital and Clinics, Department of Pediatrics, Los Angeles, California, USA.

In 6 infants and children, ages 2 months to 9 years, with congenital gastrointestinal anomalies (4 patients) or with prior history of malignant disease admitted in remission for bone marrow transplantation (2 patients), the effects of three isocaloric intravenous nutritional regimens on energy metabolism and substrate utilization were studied to determine the effect of different levels of carbohydrate and fat on nitrogen retention. All three regimens were designed to deliver adequate calories for growth and fluid volumes normal for age. Solution A provided 8% of energy as amino acids, 87% as carbohydrate and 5% as fat. Solution B provided 8% of energy as amino acids, 60% as carbohydrate and 32% as fat. Solution C provided 8% of energy as amino acids, 34% as carbohydrate and 58% as fat. Each regimen was given through a random crossover design for 3-5 consecutive days to each subject. Oxygen consumption and respiratory quotient (RQ) were measured by indirect calorimetry for 20-30 minutes in the morning on the final day of each study period. Energy metabolism and carbohydrate, fat and protein utilization were calculated from the urinary nitrogen excretion and the nonprotein RQ. Administration of solution A (high carbohydrate, low fat) was associated with moderately increased BMR and RQ and with low nitrogen retention (19.1±12.7%, 1.06±0.14, and 98±28mg N/kg/D). Both the BMR and the RQ decreased when less carbohydrate and more lipid were given (BMR 4.3±11.6% [p<0.05], RQ 0.92±0.09 [p<0.01] for solution B; BMR 3.94±10.6% [p<0.01], RQ 0.86±0.09 [p<0.01] for solution C). Among the solutions tested, optimal nitrogen retention (163±60mg N/kg/D [p<0.05]) was noted with solution B. Our data supports the conclusion that a physiologic balance of fat and carbohydrate administration results in optimal nitrogen retention.

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DEVELOPMENT OF GLUTAMINASE ALONG THE VILLUS-CRYPT AXIS OF RAT JEJUNUM. L.E. Nagy and N. Kretchmer. University of California, Berkeley, Department of Nutritional Sciences.

During the development of intestinal cells, energy is required for a number of functions: maintenance of cellular proliferation, differentiation and migration, and absorption and packaging of absorbed material. While glutamine is the major fuel source for the entire small intestine, the utilization of glutamine during the development of the intestinal cell has not been described. We have examined the regulation of glutaminase (EC 3.5.1.2), the entry enzyme for glutamine metabolism, in enterocytes isolated along the villus-crypt axis from jejunum. Specific activity of glutaminase, μmol/mg protein/h, was 5.07±0.60 in villus cells (0-75% of the total protein removed from jejunum), 6.07±1.29 in the villus crypt junction cells (75-95% of total protein) and 3.91±0.80 in crypt cells (95-100% of total protein). Quantity of glutaminase protein was determined by a dot immunobinding assay using an antibody to purified glutaminase. Immunoreactive glutaminase-protein relative to total cell protein was 6.03±1.99 cpm/μg homogenate protein in villus cells, 3.34±1.16 cpm/μg at the villus-crypt junction and 4.47±1.94 in crypt cells. Thus activity of glutaminase relative to immunoreactive protein was 2.2-fold higher at the villus crypt junction than in villus. All cell types in jejunum have significant activity of glutaminase. The highest activity, present in the area of rapid cellular differentiation, is due to an increased activation of enzyme protein.

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GASTROINTESTINAL (GI) BLOOD FLOW AND O_2 UPTAKE IN PIGLETS: RECOVERY FROM HYPOXEMIA. Philip Nowicki, Randy Miller, Nancy Hansen, John Hayes (Spon. by Grant Morrow), Dept. Peds., Ohio State University and Children's Hospital, Columbus, Ohio.

The inability of the neonatal GI tract to restore circulatory homeostasis after acute hypoxemia may contribute to hypoxic tissue damage, and is relevant to the etiology of necrotizing enterocolitis. Measurements of cardiac output (CO) and GI blood flow (QGI; microspheres), arterio-venous O_2 content gradient ($\Delta A\text{-}VO_2$), and O_2 uptake (VO_2GI) were made in 8 2-day old piglets before (baseline) and during hypoxemia, and then 5 and 60 minutes after restoration of normoxia (recovery). Hypoxemia (pO_2 20 mmHg for 15") was induced by lowering FiO_2. Results:

	Baseline	Hypoxemia	Recovery 5"	Recovery 60"
CO ($ml \cdot min^{-1} \cdot kg^{-1}$)	350±22	216±29*	362±41	361±4
QGI ($ml \cdot min^{-1} \cdot 100gm^{-1}$)	116±11	33±8*	138±27	79±6*
$\Delta A\text{-}VO_2$ (ml $O_2 \cdot 100$ ml^{-1})	1.9±0.1	1.1±0.1*	4.3±0.4*	1.2±0.2*
VO_2GI ($ml\ O_2 \cdot min^{-1} \cdot 100gm^{-1}$)	2.1±0.1	0.4±0.1*	5.4±1.0*	0.9±0.1*

m±SEM * = p<0.05 vs. baseline, by ANOVA

5" into recovery VO_2GI increased above baseline; presumably, this effected repayment of the O_2 debt incurred during hypoxemia. 60" into recovery QGI fell while CO was unchanged, indicating an increase in GI vascular resistance. $\Delta A\text{-}VO_2$ also fell and VO_2GI decreased well below baseline. Thus, the initial phase of recovery is appropriate, but the later phase involves a reduction in GI O_2 transport and VO_2GI. This response could contribute to GI tissue damage subsequent to hypoxemia.

● 705

PROSTAGLANDINS (PG) STIMULATE DISACCHARIDASE ACTIVITY IN ADRENALECTOMIZED SUCKLING RATS. Josef Neu, Wendell N. Crim, Nancy C. Hodge. (Spon. by Donald V. Eitzman) U. of Fla. College of Medicine, Dept. of Pediatrics, Gainesville.

Previous studies have demonstrated that parenteral administration of prostacyclin (PGI_2) and enteral administration of 16,16-dimethylPGE$_2$ (PGE) stimulate the activity of small intestinal disaccharidases in suckling rats. To determine whether this is a glucocorticoid-mediated effect, 10 day old rats were adrenalectomized (ADX) or sham (SH) operated. On days 11-14 these groups received PGE (100 μg/kg. b.i.d.) by gavage. Triacetin (TRI), the PGE vehicle, was administered to ADX and sham control littermates. Sacrifice was on day 15. Corticosterone RIA and autopsy were used to confirm completeness of ADX. Sucrase and maltase (glucoamylase) activity (U=μmole/min/total small intestine) and specific activity (S.A. = U/mg protein) were elevated in adrenal-intact and ADX-PGE treated animals as follows:

	Lac (U)	Lac (SA)	Suc (U)	Suc (SA)	Mal (U)	Mal (SA)
SH-PGE	17.7±5.9	.10±.03	7.5±3.1*	.05±.03*	20.4±10.1*	.13±.09+
SH-TRI	12.7±6.4	.08±.04	2.1±1.2	.02±.01	5.2± 3.4	.04±.02
ADX-PG	16.6±3.8	.10±.04	2.4±1.4*	.014±.01+	7.3± 4.0+	.05±.02#
ADX-TRI	12.5±4.3	.08±.05	0.5±0.2	.004±.002	3.8± 1.1	.03±.01

* p<.02; + p=.05; # p=.09

Prostaglandin stimulation of disaccharidase activites in both control and ADX animals indicates that the adrenal axis is not necessary for this effect. The precocious elevation of disaccharidase activities suggests either a direct effect on the intestine or mediation through another "messenger" system.

● 708

BILE SALTS FUNCTION AS CALCIUM IONOPHORES. David G. Oelberg, Jeffrey W. Sackman, Eugene W. Adcock, Roger Lester and William P. Dubinsky. University of Texas Medical School. Departments of Pediatrics, Physiology, and Internal Medicine. Houston, Texas.

It has been previously demonstrated that bile salts (BS) induce calcium (Ca) dependent hemolysis which is directly proportional to Ca influx into red blood cells. To examine how BS's might facilitate Ca movement across membranes, we investigated Ca uptake by unilamellar phospholipid vesicles (PV) prepared from soy phosphatidates. PV's were incubated with 5.0 mM ^{45}Ca either alone (control) or with added 750 μM BS - taurodeoxycholate (TDC) or taurocholate (TC) (Hepes buffer, pH 7.0, 37°C). After 10 min, Ca uptake was 1.88 ± .08 nmoles by the control PV's, 3.28 ± .04 nmoles by the PV's with TC, and 5.06 ± .03 nmoles by the PV's with TDC. ^{45}Ca uptake was also compared to sodium-22 (^{22}Na) or rubidium-86 (^{86}Rb) uptake in the presence of TDC. After 5 min of incubation, TDC-associated Ca uptake was nearly 2 times greater than those of either Na or Rb. Finally, PV's were exposed either to 25 nM valinomycin (V) which induced an electrochemical gradient across PV membranes, or to 25 nM nigericin (N) which induced a pH gradient. TDC without V or N increased Ca uptake 1.67 ± .08 times over controls. Exposure to V increased TDC-associated Ca uptake 1.45 ± .05 times, and exposure to N increased it 1.90 ± .03 times. Conclusions: (1) BS's induce cation uptake by PV's that is more specific for Ca than for Na or Rb; (2) as was true of red blood cells, TDC induces greater Ca uptake than TC; (3) pH gradients promote greater BS-induced Ca uptake than electrochemical gradients. Hypothesis: The mechanism of BS-induced Ca uptake may be similar to that of Ca ionophore A23187 whose activity is similarly promoted by pH gradients.