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COMPARATIVE ABSORPTION OF GLUCOSE (G) AND LACTOSE (L) IN PREMATURES ASSESSED WITH ^{13}C -SUBSTRATES. William C. MacLean, Jr., Robert D. Murray, William Wong, Peter D. Klein. Ross Laboratories & Dept of Pediatrics, Ohio State U Coll of Med, Columbus, OH; Johns Hopkins, Baltimore; Baylor Coll Med, Children's Nutr Res Center USDA/ARS, Houston.

Postmortem intestinal lactase assay & postnatal breath H_2 studies suggest suboptimal lactose absorption until 38+ wks post-conception. G and L absorption were compared directly using ^{13}C substrates. 18 paired studies were done in 6 infants (gest age 28-34wk, age 11-60d, wt 770-2050g, intake 94-161 kcal/kg/d.) Breath $^{13}\text{CO}_2$ above baseline by interval samples during 8 hr after ^{13}C -L or ^{13}C -G had been added to 8 am feeding was determined by isotope ratio MS, as was ^{13}C (non- and dialyzable) in 24hr stool collections. ^{13}C was recovered in breath by 0.5hr in only 18% of studies with G, 21% with L. Peak respiratory $^{13}\text{CO}_2$ excretion occurred at 2-4 hr with both G&L. Cumulative %dose ^{13}C (Cum %d) was unexpectedly greater with L than G at 5, 6, 7, 8 hrs pc, the opposite of that found with full terms. ($P < 0.05$ - 0.001 , $\bar{x}$ 8hr Cum %d; $G = 41 \pm 14\%$, $L = 51 \pm 10\%$, assuming CO_2 production rate $300 \text{ mL}/\text{M}^2/\text{hr}$). Despite this, L was excreted in stool more frequently than G (89% vs 56% of studies, $X^2 = 4.98$) & in greater amount ($\bar{x}$: G 3.3% of dose, L 6.1%). The apparent % dose oxidized per % dose absorbed was 22% greater after L than G. In 3 studies multiple breath $^{13}\text{CO}_2$ peaks followed breath H_2 peaks. The small premature assimilates L more rapidly than G. Paradox of increased ^{13}C in breath and stool and altered apparent oxidation of L suggest different routes of absorption of G and L and implicate colonic bacteria in L assimilation. NIH HD 10111, HD 18188, AM 28129, USDA/ARS.

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DECREASED GASTRIC PGE_2 SECRETION BY "STRESSED" RATS, Lucyndia R. Marino (sponsored by William J. Byrne), University of Michigan, C.S. Mott Children's Hospital, Department of Pediatrics, Ann Arbor, Michigan

Rats that are restrained and deprived of food for hours develop gastric ulcers. To evaluate the effects of such "stress" on gastric PGE_2 & H^+ secretion, 2 groups were studied: 9 control (C) and 10 "stressed" (S) rats. Stressed rats were in restraint cages for 24 hours. Fasting samples from both groups for PGE_2 & H^+ were obtained by luminal gastric perfusion with .9% NaCl & 4 mEq/l KCl for 6, 10' periods. At the end of the 60' rats were sacrificed & stomachs removed & examined. No animal had ulcers at post mortem exam. Mean ($\pm$ SD) PGE_2 & H^+ secretion are shown below.

	H^+ uEq/hr	PGE_2 ng/hr
Control	$.060 \pm .043$	14.30 ± 5.81
Stress	$.040 \pm .023$	9.40 ± 4.13
P value	NS	$p < .05$

The rate of PGE_2 & H^+ secretion per min decreased exponentially with time in C and S, $p < .001$. There was a linear correlation between PGE_2 (pg/min) & H^+ (mEq/min) for both groups $p < .001$. In conclusion, "stress" decreases PGE_2 secretion by gastric mucosa prior to the development of ulcerations. Although there is a linear relationship between PGE_2 & H^+ secretion, 24 hr "stress" does not decrease both concomitantly. The decreased PGE_2 secretion, by "stressed animals", in the presence of normal H^+ secretion may identify a mechanism for injury and a defect in cytoprotection.

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THE EFFECTS OF CIMETIDINE (C) ON GASTRIC PGE_2 AND H^+ PRODUCTION Lucyndia R. Marino (sponsored by William J. Byrne), University of Michigan, C.S. Mott Children's Hospital, Dept. of Pediatrics, Ann Arbor, Michigan

For unknown reasons, antacids are more efficacious than C in the prophylaxis of UGI bleeding. To evaluate whether C adversely effects PGE_2 production & therefore cytoprotection, 3 groups of rats were studied: 5, control (Co); 8, Cimetidine (C_1) 1 dose IP 60 mg/kg; 6, Cimetidine (C_{14}) single daily dose IM 200 mg/Kg/day X 14 d. Samples for measurement of PGE_2 & H^+ were obtained in 6, 10' periods by luminal gastric perfusion with 0.9% NaCl & 4 mEq/l KCl. During periods 5 & 6, Co rats were perfused with isotonic NaHCO_3 to evaluate the effects of basic pH on PGE_2 secretion. During periods 1-4, $\bar{x}$ H^+ secretion was 2X greater in Co than C_1 or C_{14} rats. PGE_2 secretion was not significantly different. There was an exponential relationship between total PGE_2 (ng/hr) & time ($p < .001$) & H^+ (mEq/hr) & time ($p < .001$) for the 3 groups. Calculated values for H^+ (mEq/hr) and PGE_2 (ng/hr) for the entire 60' revealed that H^+ and PGE_2 secretion by Co rats were 2X the values for C_1 & C_{14} . After perfusion with NaHCO_3 , PGE_2 (ng/min) increased 2X in Co rats. There was a linear correlation between H^+ & PGE_2 secretion in Co & C_{14} ($p < .001$) but no correlation between H^+ & PGE_2 in C_1 . In summary long term & acute therapy with C decrease luminal secretion of PGE_2 & H^+ . The relationship between H^+ & PGE_2 secretion is abolished in rats given one dose of C. NaHCO_3 buffers H^+ and increases PGE_2 secretion. The reduction in PGE_2 secretion produced by C may adversely effect cytoprotection in the stomach.

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THE RELATIONSHIP BETWEEN GASTRIC PGE_2 & H^+ SECRETION IN THE BASAL (B) & STIMULATED (S) STATE, Lucyndia R. Marino (Sponsored by William J. Byrne), University of Michigan, C.S. Mott Children's Hospital, Department of Pediatrics, Ann Arbor, Michigan

Nine children; $\bar{x}$ age 12.5 yrs; 4F, 5M; had gastric acid analyses performed. All pts had normal gastric mucosa at endoscopy. Gastric secretions were collected during 4, 15' periods in the B state & 6, 15' periods following 6 ug/kg pentagastrin SC. Three pts had increased BAO but their MAO was normal. In B there was no correlation between PGE_2 & H^+ secretion. After pentagastrin $\bar{x}$ H^+ (mEq/hr) increased in all & significantly in 5/9 ($p < .05$). Mean PGE_2 secretion ng/hr increased in 6/9 but was not statistically significant. PGE_2 secretion (ng/min) increased in at least two S time periods and the group x PGE_2 (ng/min) peaked in S period #2 and was 2X basal. Acid production also peaked in S period #2. In 5/9 pts during S periods 1-6, there was an exponential relationship ($p < .05$) between PGE_2 (ng/min) & H^+ (mEq/min). In the other 4 pts there was no correlation between PGE_2 & H^+ . Two of these 4 pts had secretory diarrhea & 2 duodenal Crohn's. The other 5 had duodenal ulcers (DU). In summary, there is no relationship between PGE_2 & H^+ secretion in the B state. Pentagastrin does increase both H^+ & PGE_2 secretion. In pts with DU there is a relationship between pentagastrin stimulated H^+ & PGE_2 secretion; however, this relationship appears to be disrupted with other gastrointestinal diseases.

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EFFECT OF CONTINUOUS INFUSION ON THE CONTENT AND COMPOSITION OF THE LIPIDS IN HUMAN BREAST MILK. Mary C. McKenna, Linda L. Wright, Suzanne Ullsperger. (Sponsored by Allen Schwartz), Univ. Md. Sch. Med., Dept. Peds., Baltimore

Premature newborn infants are often fed breast milk by nasogastric or nasojugal infusion in order to provide a constant supply of nutrients. Many lipophilic compounds have been shown to be taken up by infusion tubing. We examined the effect of infusion tubing on the lipid content and fatty acid (FA) composition of breast milk. Frozen breast milk samples were thawed, loaded into a syringe and pumped at 2 ml/hr through infusion tubing. Samples were taken from the syringe reservoir at time 0, 4, 8, 12 hrs and collected under N_2 from the tubing effluent at 0, 1, 2, 3, 4, 8, and 12 hrs. Milk from the tubing effluent at 4 hrs had a small decrease in the percent of oleic (-1.5%), linoleic (-0.6%) and α -linolenic (-0.13%) acids and an increase in the percent of myristic (+1.09%), palmitic (+0.54%), and stearic (+0.49%) acids. These changes were more pronounced in the 12-hr tubing effluent. The polyunsaturated/saturated FA ratio of the milk decreased from an initial value of 1.13 to 1.03 in the 4 hr effluent. The amount of total lipid decreased by a mean of 18% and 24% after infusion for 4 and 12 hrs respectively. Separation of the lipid classes by TLC suggested that the loss of lipids led to an increase in the free FA and decrease in the triglyceride content of the milk. The data indicates that a substantial amount of the lipids (and therefore calories) were lost from human breast milk delivered by continuous infusion. The relative amount of the individual lipid classes may also be altered by interaction with the tubing.

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Growth and Mineral Metabolism in preterm infants receiving a novel human milk fortifier. Houchang Modanlou, Mary O. Lim, James W. Hansen. Long Beach Mem. Hosp., Long Beach, CA, and Mead Johnson Co., Evansville, IN. Preterm human milk (PHM) initially contains high levels of nutrients to help meet most premature infant needs. However, calcium and phosphorus and, after 2 to 4 weeks, some other nutrient levels in PHM are lower than required for optimal nutrition. A human milk fortifier (HMF) was designed to be added to PHM after 2 weeks post-partum to raise nutrient levels to approximate requirements. HMF increased protein (60:40 whey:casein) by 0.7 g/dl, calories by 4 kcal/oz, and minerals (mg/dl) as follows: Ca 60, P 33, Na 6.9, K 16, Cl 18, Mg 4, Cu 0.04, Zn 0.8. Ten preterm infants received PHM alone, 8 received PHM plus HMF (FPHM), and 12 received Enfamil Premature Formula (EPF).

	Days From	Cal eff	Serum	Serum	BMC
Feed	1.5-1.8 kg	g/100 kcal	Protein	Sodium	Calcium mg/cm
PHM	12.0 $\pm$ 3.2*	13.4 $\pm$ 1.8*	4.2 $\pm$.2*	136 $\pm$ 3	9.54 $\pm$.4*
FPHM	8.9 $\pm$ 1.1	15.2 $\pm$ 1.4	4.6 $\pm$.5	136 $\pm$ 4	9.8 $\pm$.5
EPF	8.3 $\pm$ 2.3	16.3 $\pm$ 1.2	5.0 $\pm$.3	134 $\pm$ 2	9.9 $\pm$.3

* $p < .05$ compared to the other groups. Serum values determined after 2 to 4 weeks on study. BMC = Bone mineral content of mid humerus at study conclusion. Cal eff = caloric efficiency. Those infants receiving HMF responded comparably to those receiving EPF, but grew faster and tended to have better mineral status (not statistically significant) than those receiving PHM alone. No clinically significant differences were observed in metabolic parameters, feeding tolerance, or complications. This data supports the use of HMF in human milk fed preterm infants.