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CHOLESTYRAMINE INHIBITION OF JEJUNAL Na^+ SECRETION IN SMALL BOWEL BACTERIAL COLONIZATION. M. Berant, M. A. Bayne, R. A. Wapnir and F. Lifshitz. Dept. of Peds., North Shore Univ. Hosp., Manhasset, NY 11030 and Dept. of Peds., Cornell Univ. Med. Coll., NY, NY 10021.

The effect of cholestyramine (Ch) on cAMP-mediated jejunal Na^+ secretion associated with increased luminal free fatty acids (FFA) and deconjugated bile salts (DBS) during bacterial colonization of the small bowel was studied. Male Wistar rats (70-90 gm) were injected with mecamlamine HCl for 3 days, to induce bacterial proliferation in the upper small bowel (MC); controls received NaCl (C). Half of each group were fed Ch 4% in the diet. Supernatants of small intestinal contents were assayed for FFA and DBS; jejunal mucosa was examined for cAMP and ATPases; jejunal Na^+ transport was measured by *in vivo* perfusion with Krebs-Henseleit buffer. MC rats not fed Ch had higher luminal concentrations of FFA and DBS as compared to C (FFA: 21 ± 0.3 vs. 13 ± 0.03 mEq/l; $p < 0.02$; DBS: 1.11 vs. 0.23 mM). They also had elevated cAMP (34.14 ± 6.17 vs. 10.72 ± 7.73 pmol/mg protein; $p < 0.01$) and a marked Na^+ efflux into the jejunum (secretion of 101.07 ± 33.5 vs. absorption of 90.15 ± 14.9 $\mu\text{Eq}/\text{min}/\text{cm}$; $p < 0.001$). Feeding Ch to MC rats inhibited these effects: FFA and DBS were lower than in C and cAMP did not differ from C. While Na^+ efflux was noted in MC rats, Na^+ absorption did occur in MC rats fed Ch (38.14 ± 21.6 $\mu\text{Eq}/\text{min}/\text{cm}$; $p < 0.001$). The ATPases were similar in all groups. The data indicate that the cAMP-mediated jejunal Na^+ secretion and the increased luminal FFA and DBS during small intestinal bacterial colonization is inhibited by oral Ch, which binds these products and renders them innocuous.

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THE GREATER NITROGEN SPARING EFFICIENCY OF CHINESE THAN CAUCASIANS REINTERPRETED ACCORDING TO THE JUSTIFICATION THEORY. Samuel P. Bessman and Po-Chao

Huang, USC Med. Sch., Dept. Pharmacol. & Nutrition, Los Angeles. Chinese (CH) lose approximately 2/3 as much nitrogen (N) as Caucasians (CA) on a high fat, high carbohydrate, zero protein intake. The justification theory of hereditary non-essential amino acid (NEA) deficiency states that deficiencies in NEA can occur on marginal protein intake by heterozygotes for any of the 32 enzymes required to synthesize all the NEA. N loss during starvation depends on the ability to resynthesize protein which is turning over. If an essential amino acid is lost then all of the NEA lost by that molecule of protein cannot be reutilized. Efficiency of reutilization depends upon the available constellation of NEA. The low protein intake of the CH, who have subsisted for 50 centuries on cereals, would make it difficult for a heterozygote for NEA synthesis to survive. The CA, a hunter and fisherman, would not lack for protein. Though total caloric nutrition might be poor there would be no selection against mutations in the NEA pathways. This interpretation is supported by data which show that the essential amino acids in the plasma of CH and CA after a 2-week fast are equal whereas the plasma of CH contains 50% more of the NEA. This is associated with a mean loss of 1.8 mgs. of nitrogen per basal calorie in the CA and 1.2 mgs. per basal calorie in the CH. The justification hypothesis may not only supply an explanation of differential response to the same diet, but might even provide dietary suggestions for prevention of many problems of protein synthesis.

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POSTURE AND GASTRIC EMPTYING IN THE NEWBORN. I. Blumenthal, A. Ebel, R.S. Pildes. Cook County Children's Hosp. Univ. of Ill. Dept. of Ped. Chicago, Illinois.

The effect of posture on the pattern of gastric emptying was studied in 14 healthy neonates, ages 5-27 days. Mean ($\pm$ S.E.) birth wt. was 2.3 ± 0.04 g (range 1740-2520g) and gestation, 36.7 ± 0.2 (range 32-42wk). Fifty-six test meals of 10% dextrose with phenol red ($0.03\text{mg}/\text{dl}$) as a marker were given over 2-3 min. The infants were studied in each of four positions: right, left, prone and supine and the postural sequence was varied. The emptying pattern was determined by the double sampling dye dilution technique. Samples were taken at 20 min intervals for 2 hours. The time in min for 1/4, 1/2 and 3/4 of the original volume to leave the stomach is shown in the table:

Position	1/4	1/2	3/4
Right	12.0 ± 1.3 min	27.2 ± 3.7	80.6 ± 18.7
Left	11.4 ± 0.7	31.9 ± 4.7	88.8 ± 11.4
Prone	12.6 ± 0.8	34.6 ± 4.4	87.5 ± 5.9
Supine	13.4 ± 2.2	31.3 ± 5.6	86.8 ± 10.7

The percentage of the test meal remaining in the stomach at 20 min was similar in each of the 4 positions and ranged from 51.4% to 57.6%. There was no correlation with gestational age.

The pattern of emptying was characterized by an initial rapid phase of 2.3% per min for the first 20 min followed by an exponential pattern at a rate of 0.2-0.6% per min for the remaining time. The results indicate that posture does not appear to influence the rate or pattern of neonatal gastric emptying.

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A NON-PARALLEL DECREASE IN PANCREATIC ENZYMES IN REO VIRUS TYPE 3 INFECTION OF SUCKLING MICE. D. Branski, H.S. Faden, T. Hatch, J. Krasner, E. Lebenthal. Division of Gastroenterology, The Buffalo Children's Hospital, SUNY at Buffalo, New York.

A critical phase in the structural and functional maturation of the pancreas takes place in the neonatal period. The response to Reo Virus type 3 in suckling mice was studied after injecting 1×10^7 PFM into 9 day old animals intraperitoneally. Both control and test animals were sacrificed either 3 or 6 days following viral injection. Histology of the pancreas, after 3 and 6 days of viral infection, revealed a normal architecture, normal acinar cells with only a mild mononuclear infiltration observed in mice after 6 days of infection (F).

	A	L	T	CT	CPA	CPB
Control	96 ± 13	4.8 ± 1.9	5.6 ± 1.4	0.5 ± 0.01	0.7 ± 0.5	1.0 ± 1.0
F (6 days)	32 ± 11	2.8 ± 0.9	6.6 ± 1.6	1.1 ± 0.2	0.6 ± 0.1	0.8 ± 0.4

In the 6 day infected mice, amylase (A), is significantly reduced ($p < 0.001$) as compared to the control of the same age. Lipase (L) is decreased as well, but to a lesser degree. In contrast, trypsin (T), increased significantly ($p < 0.02$) in 3 day infected, but no difference in activity is seen in 6 day infected mice. Chymotrypsin (CT), was increased significantly ($p < 0.02$) over the similar control group. Carboxypeptidase A (CPA) & B (CPB) activities were not altered in either the 3 or 6 day infected animals. The enzyme activities can be divided into three groups; the amylase and lipase activities are diminished, the endopeptidases, trypsin and chymotrypsin are increased and the exopeptidases, carboxypeptidase A & B are without change. The effect of the viral infection on the enzyme activities indicate a non-parallel change on the developmental pattern of the pancreatic enzymes. (*units = S.D. = $\mu\text{moles}/\text{mg protein}/\text{min}$.)

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TRANSPLENTALLY INDUCED LIVER INJURY DUE TO LITHOCHOLATE: CANALICULAR Na^+ , K-ACTIVATED ATPase AND MEMBRANE MICROVISCOSITY IN NEWBORNS. Yoram Bujanover,

Barbara Sharp, Joanne O. Whitney and M. Michael Thaler, University of California, Department of Pediatrics, San Francisco

Administration of lithocholate (L) to pregnant rats has been shown to induce inflammatory lesions in liver of newborn offspring, progressing to intrahepatic biliary hypoplasia and chronic cholestasis (Gastroenterology 73:1214, 1977). The site of action and mechanism of L toxicity *in utero* was investigated in newborn rats delivered by dams maintained on standard chow (S) or S+2.5% L throughout pregnancy. Newborns were sacrificed at 1, 2, and 5 days after birth, plasma collected, and liver removed for histological examination and isolation of canalicular-enriched plasma membranes. Membrane Na^+ , K-activated ATPase activity was assayed, and surface microviscosity (mV) determined by fluorescence polarization. Plasma L concentration was 2-3 $\mu\text{mol}/\text{L}$ in treated, and was undetectable in untreated newborns. Hepatic changes in L-treated newborns were periportal inflammation, formation of pseudoducts and giant cells. ATPase activity in membranes from L-treated newborns was reduced by 40% compared with normals (2.58 ± 0.30 vs. 4.14 ± 0.45 $\mu\text{mol P/hr}/\text{mg protein}$). Canalicular mV (in poises) was increased by 30% in L-treated newborns compared with normals (5.65 ± 0.30 vs. 3.99 ± 0.18 ; $p < 0.01$). Conclusion: Exposure to L *in utero* is associated with changes in canalicular membrane function and physical properties known to interfere with bile secretion, and may thus initiate a self-perpetuating cholestatic disorder after birth.

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ANTHROPOMETRIC GROWTH STUDY OF INFANTS LESS THAN 1750 GRAMS USING THREE DIFFERENT FORMULAS. Sergio A. Bustamante and Sheila Schreck (Spon. by Richard A.

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We studied the differences in anthropometric factors of infants taking 1 of 3 commercial formulas by measuring weight, length, crown-rump, head circumference, and dynamic skin fold thickness from 2 sites. This was done 3 times weekly until discharge, or up to 30 days. Formulas were assigned using envelope randomization. Infants with fetal malnutrition or obvious congenital anomalies were not included; infants weighing less than 1000 grams were too few to study statistically. Formula A) Soybean based 20 cal/oz, B) Cows milk based 24 cal/oz, and C) Cows milk based 24 cal/oz with lower osmolality. We studied 105 infants of less than 1750 gms which were subdivided into 4 groups at increments of 250 gms. The 12th day after starting on formula only, i.e., no IV supplementation, infants taking formula A in the 1001-1250 and 1501-1750 gm weight groups weighed less than those taking B and C ($p = 0.01$). No difference in dynamic skin fold thickness was found between the formula groups. Approximately the same number of infants from the 3 formula groups developed complications with impaired oral intake and were excluded. Infants taking formulas B and C achieved the discharge weight (2100 gms) sooner than those on formula A; however, those on B and C had more digestive system complications (6 vs. 0) including necrotizing enterocolitis.

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