23 IN VIVO AND IN VITRO RESPONSE TO LACTOSE (L) BY THE FLORA OF BREAST-(BR) AND BOTTLE-FED (BO) INFANTS.

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To compare in vivo and in vitro responses of fecal flora to L, breath was collected from 15 BR and 12 BO infants aged 0.1-6 mo and analyzed for H_2 and CH_4. Stools from 5 BR and 8 BO were analyzed for H_2, lactic acid (LA), glucose (GL), and galactose (GA) before and after anaerobic incubation with 1.25% L at pH 6.8 and 5.5. Peak breath H_2 values for BR and BO were 36±38 ppm (range 2-120) and 16±10 ppm (4-25). Breath CH_4 values for BR and BO were 6±3 (0-12) and 8±3 (2-11).

	H_2		LA		L		GL		GA	
			µmol/g feces (mean ± SD)							
	BR	BO	BR	BO	BR	BO	BR	BO	BR	BO
PI*	-	-	16(26)	7(4)	0	0	6(6)	1(2)	5(5)	1(1)
+L†	10(7)	19(5)	151(53)	102(39)	1(1)	2(4)	6(6)	21(17)	8(14)	14(16)
+L‡	4(5)	6(2)	164(54)	86(46)	1(2)	2(2)	5(6)	44(22)	11(20)	36(24)

* Preincubated stool; †Postincubation pH 6.8 and ‡5.5. Preliminary results indicate that stool incubates at pH 6.8 of BO infants produced more H_2, GL, and GA than those of BR who accumulated more LA. At pH 5.5, H_2 production decreased in both groups; GL and GA accumulation increased further in BO. This is the first report of CH_4 production in infants. Fecal flora of BO appeared to have a limited capacity to utilize excessive amounts of GL and GA, particularly at pH 5.5, while BR continued to transform GL and GA into LA. We postulate that these differences may explain the means by which BR and BO metabolized malabsorbed L in vivo.

24 ORAL REHYDRATION THERAPY: A SOLUTION TO THE PROBLEM.

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In previous studies, an oral rehydration solution containing glycine (gly) and a glucose polymer performed better than WHO-ORS in reversing water secretion in an animal model of secretory diarrhoea. We have now assessed the relative contribution of gly and glucose (glu) concentration and osmolality (osm) to the efficacy of oral rehydration solutions (ORS). After exposure to pure cholera toxin, whole rat small intestine was perfused in situ with 7 different ORS. All ORS derived from a basic solution (BORS) containing Na 50, K 25, Cl 75 and glu 50 mM, to which 25 or 50 mM of either gly, glu or mannitol was added. All ORS reversed water secretion to absorption with varying efficacy. Maximum water absorption was obtained with BORS (161±10 µl/min/g dry wt). When osm of BORS was raised to 225 and 250 mOsm/Kg by adding mannitol, water absorption decreased significantly (106±5 and 44±4 respectively, $p<0.01$). At each of these osm, substitution of mannitol by gly or glu resulted in similar increases in water absorption ($p<0.02$), but all modifications compared unfavourably with BORS. Na secretion occurred with all ORS tested, despite net water absorption. These results suggest that (1) osmolality is a key factor influencing the efficacy of ORS, (2) glycine has no advantage over glucose in promoting water absorption and (3) solutions with [glu]:[Na] ratio up to 2:1 are beneficial.

25 SUPPLEMENTATION OF FRESH HUMAN MILK WITH HUMAN MILK PROTEIN FOR FEEDING VERY-LOW-BIRTH-WEIGHT (VLBW) INFANTS.

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18 VLBW-infants (BW = 800-1600 g, AGA) were studied until discharge at a weight of 2000 g. All infants received mother's own milk. 9 received such milk supplemented with 1 g of ultrafiltrated human milk protein. At a milk intake of 180 ml/kg/d the protein intake varied between 2 and 4.5 g/kg/d. Protein fortification of human milk produced a higher weight gain (32.2 g/d vs. 26.4 g/d, $p<0.005$). The best protein utilization (weight gain per g of protein) was obtained at a protein intake of about 3 g/kg/d. Blood urea nitrogen (BUN) concentrations and other indices of protein metabolism correlated to protein intake. The mean BUN concentration was 7.2 mg% in the protein supplemented group. Protein supplementation resulted in an elevation of plasma total amino acid concentrations at the end of the study (2788 vs. 2316 µmoles/l). Individual plasma amino acid concentrations also varied with protein intake. A daily human milk protein intake of about 3 g/kg produced a rate of weight gain corresponding to that found in utero at the corresponding gestational age in VLBW-infants. The plasma responses of protein metabolism observed at this protein intake are similar to those found in term infants, breast-fed. We suggest that these results can be used as a model when evaluating feeding regimens with non-human proteins in VLBW-infants.

26 PROTEIN METABOLISM IN VERY-LOW-BIRTH-WEIGHT (VLBW) INFANTS SMALL FOR GESTATIONAL AGE (SGA) FED PROTEIN FORTIFIED HUMAN MILK (HM).

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In order to demonstrate metabolic differences in response to protein intake we have studied 23 AGA and 19 SGA infants of VLBW at the eight day of life. The infants received HM or HM fortified with HM-lyophilisate at protein intakes ranging from 2.0 to 3.5 g/kg/d as calculated from protein content of the milk and the volumes ingested. α-amino-nitrogen and amino acids in serum as well as total nitrogen and α-amino-nitrogen excretion in the urine was measured. Serum α-amino-nitrogen and amino acid concentrations correlated with protein intake in both groups of infants, but concentrations were significantly higher in the SGA-infants above a protein intake of 2.5 g/kg/d. α-amino-nitrogen and total nitrogen excretions in the urine reflected also protein intake and were significantly increased in the SGA-infants. On the eight day of life a protein intake above 2.5 g/kg/d produces metabolic indices of protein overload in SGA-infants. These findings demonstrate that differences in protein metabolism are present between AGA and SGA infants of VLBW. A decreased rate of protein utilization for some time after birth may be present in the SGA-infants. When the nutritional management of VLBW-infants is planned these differences must be considered and protein intakes should be increased with caution in the SGA-infants.

27 THE MOTOR RESPONSE OF THE SMALL INTESTINE TO MILK FEEDS IN PRETERM INFANTS.

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Small intestinal motor activity is characterised by a cyclical pattern of activity which is disrupted following food and is replaced by a continuous fed pattern. In the preterm infant, however, both patterns are poorly developed.

Using constantly perfused multilumen jejunal catheters we have studied the effect of enteral feeding on small intestinal motor activity in 10 preterm infants studied on 23 occasions between 28 and 44 weeks gestation. When given a bolus feed appropriate to the infants age, the fasting pattern was not disrupted or replaced by a fed pattern in the very preterm infant (28-30 weeks) but disruption and the appearance of the fed pattern increased with increasing gestational age ($r=0.6$, $p=0.005$), increasing fasting motor maturation ($r=0.6$, $p=0.005$), reduced feed frequency ($r=0.66$, $p=0.001$) increasing feed volume ($r=0.8$, $p<0.0001$) and particularly with increased time fed enterally ($r=0.87$, $p<0.0001$).

These data suggest that although gestational age and fasting motor maturation are important, the major determinants in the development of mature postprandial activity in the preterm infant is the exposure of the intestine to enteral nutrition and the introduction of bolus feeds.

The adoption of these feeding practices should facilitate intestinal motor development and enteral feeding tolerance.

28 IS IMMUNOGLOBULIN 'A' (IgA) RELEASE IN THE INTESTINE CONTROLLED BY A CALCIUM DEPENDENT SECRETORY MECHANISM?

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It has previously been shown that cholecystokinin octapeptide (CCK-8)as well as pilocarpine can accelerate the release of IgA in the gastrointestinal tract. The purpose of this study was to ascertain if CCK-8-induced IgA release involves calcium-coupled secretory mechanisms in the intestine. Hooded Lister rats were immunized with ovalbumin and Freund's complete adjuvant and a booster injection given 14 days later. On the 21st day a 10 cm. long segment of intestine was isolated 10 cm. distal to the pylorus. This was perfused with saline 0.5 ml/2.5 minutes. After a flush out period of 30 mins. aliquots were collected every 2.5 mins. After 10 minutes 1 u CCK was given I.V.(group 1). A second group of animals received a calcium channel blocker Verapramil I.V. at a rate of 2 mcg/min. for 20 minutes. After the first 10 mins.20 ng CCk-8 were administered I.V. Group 3 was given Verapramil only as above.
Results: following CCK-8 there was a significant rise of IgA into the intestinal fluid which began at 2.5 minutes and persisted for 10 minutes. Prior administration of Verapramil completely inhibited the CCK-8-induced release of IgA. Conclusion: it is suggested that CCK-8-induced release of IgA into the gastrointestinal tract is related to calcium dependent secretory mechanisms.