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GROWTH OF VERY LOW BIRTH WEIGHT (VLBW) INFANTS FED THEIR OWN MATERNAL BREAST MILK. Houchang D. Modanlou, Shirley P. Misa, and Angeline Yatar, UC, Irvine, Miller Children's Hospital, Long Beach, California.

Twenty-one VLBW infants (Group I) were fed their own mothers milk. Randomly selected similar VLBW infants were fed a standard 24 cal/oz formula (Group II, 19 pts) and a whey protein based 24 cal/oz formula (Group III, 20 pts). Birth weights (gm) and GA (wk) were 1230 ± 200, 30.6 ± 2.1; 1275 ± 222, 31.1 ± 1.7 & 1357 ± 170, 31.3 ± 1.7, respectively. Feedings, of equal volume per kg of body weight, were started at 4.9 ± 2.5, 5.4 ± 4.3 and 5.9 ± 4.5 day of life. Total serum protein, albumin, Ca, P, BUN and pH in each group were in normal physiologic range before, during and after the study period of achieving 1800 gm body weight and not different between the groups. Days of feedings to regain birth weight was statistically different only between I and II (18.5 ± 9.2 vs. 12.4 ± 5.3; $p < .025$). Daily weight gain was different between I and II (16.3 ± 3.1 vs 21.5 ± 4.3 gm; $p < .001$), I and III (16.3 ± 3.1 vs 20.9 ± 4.7 gm; $p < .001$). Days required to achieve 1800 gm were different between I and II (42.3 ± 14.0 vs 29.3 ± 9.7; $p < .005$) and I and III (42.3 ± 14.0 vs 28.4 ± 8.5; $p < .001$). Contrary to weight gain, changes in head growth were higher in breast milk fed infants than II and III from birth to achieving 1800 gm body weight (4.0 ± 2.1, 2.2 ± 1.7 and 3.2 ± 1.8 cm) with significant difference between I and II ($p < .01$). This study reveals that despite lower calorie and protein intake, slower weight gain and longer hospitalization, relative to their weight gain, VLBW infants fed their own maternal milk have more rapid brain growth.

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COLCHICINE INHIBITION OF SUCRASE-ISOMALTASE LABELLING IN INTESTINAL BRUSH BORDERS R.K. Montgomery, A. Forcier, M. Sybicki, and R.J. Grand. Harvard Med. School, Children's Hosp. Med. Ctr., Div. of Gastroenterology, Boston, MA

We have investigated the mechanism by which colchicine reduces disaccharidase activities in intestinal mucosa. Male CDF strain rats were injected with colchicine (0.5mg/kg body weight) freshly dissolved in physiological saline. Two hours after colchicine injection, each animal was injected with 200 μ Ci of 3 H-leucine. Two hours later, animals were sacrificed, and homogenates and brush borders were prepared from mucosal scrapings. Aliquots were precipitated with TCA and counted for label incorporation into total protein. Sucrase was immunoprecipitated from solubilized brush borders at the experimentally determined equivalence point for each sample, using specific rabbit anti-sucrase antibody. Results (x±SEM) showed significant reductions of radiolabelling of brush border protein and immunoprecipitated sucrase, but not of total homogenate protein, and brush border sucrase activity was not significantly reduced:

	control (6)	colchicine (5)	P
Homogenate (DPM/mg prot)	22,864±804	22,151±979	N.S.
Brush border (DPM/mg prot)	38,914±1884	31,855±1383	<0.01
Sucrase (DPM/mg prot)	13,766±1271	10,621±737	<0.05
(μ mole glucose/mg/min)	1.28±0.10	1.12±0.05	N.S.

A significant increase in metaphase arrests in crypts of treated mucosa independently verified the effectiveness of the colchicine dose. CONCLUSIONS: These data suggest that at a dose of 0.5mg/kg body weight, colchicine significantly inhibits the movement of newly synthesized sucrase-isomaltase to the brush border of the intestinal epithelial cell.

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RESPONSE OF BODY LEUCINE METABOLISM TO DIET SUPPLEMENTATION IN CHILDREN WITH CROHN'S DISEASE/GROWTH FAILURE. K.J. Motil, R.J. Grand, V.R. Young, D.E. Matthews, and D.M. Bier. Harvard Med. Sch., Children's Hosp. Med. Ctr., Div. of Gastro., Boston, MA; and Washington Univ., St. Louis, MO.

Responses of body leucine metabolism in children (mean age, 15.0 yr) with Crohn's disease and growth failure ($\bar{X}$ ht and wt, 143.9cm and 37.4kg) were studied before and during dietary supplementation. All children ingested a constant diet (2.35g pro/kg/d and 68Kcal/kg/d), comparable to usual oral intakes, followed by continuous nasogastric feedings of 30% protein and 40% energy increases. At the end of each 7-day period, the children received a primed constant infusion of [3 H]leucine for 4 hrs. Plasma 13 C-leucine and breath 13 CO₂ were measured at 15-min intervals during isotopic plateau. Results (μ mole/kg/hr, $\bar{X}$ ±SEM) for leucine flux (Q), oxidation (Ox) and incorporation (INC) and endogenous release (REL) were:

Functions	Dietary Interval		Normal*
	Basal	Supplemented	
Q	187.4±10.4	201.9± 9.5	168.5±19.7
Ox	90.8± 7.6	41.7± 8.6	---
INC	96.6± 6.1	162.0±17.3	140.7±19.3
REL	70.5±18.7	84.9±10.8	141.2±18.9

*Adapted from Clin. Sci. 79:13, 1980.

Thus there was net leucine conservation with supplementation via increased incorporation, and reduced oxidation and endogenous release. These findings suggest that growth failure in Crohn's is accompanied by abnormalities in leucine metabolism which may be reversed with dietary protein and energy supplementation.

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CLOTTED TRANSPYLORIC FEEDING TUBES USING A PREMATURE FORMULA. Laurie Moyer and Gary Chan. (Spon. by Lowell Glasgow), University of Utah, Department of Pediatrics, Salt Lake City, Utah.

Transpyloric (TP) feeding has been used as a method of feeding the smaller (<1200 gms) infant. We have noticed that many of our infants being fed with a "reformulated" premature infant formula (Enfamil) by TP route have developed clotted feeding tubes in spite of regular irrigation with sterile water. During a 9 month period, we studied all infants fed either the premature formula or their own mothers milk via TP route. An in vitro study was also conducted comparing the premature infant formula and a standard formula with human milk placed in TP tubes at the milk's own original pH and alkaline pH. Fifty-nine infants received TP feedings; 39 were fed premature formula and 20 received human milk. Birth weight and gestational ages between the two groups were similar. No infant receiving human milk developed a clotted feeding tube. However, 15 of the 39 (38%) infants receiving premature formula developed at least one clotted tube. The clotted group was smaller in birth weight (mean of 1044 vs. 1418 gms, $p < .001$), and younger in gestational age (29 vs. 32 weeks, $p < .05$) than the non-clotted group. There were no differences between the two groups in age of initial feeding or rate of infusion. The in vitro study showed that the tubes were completely occluded, consistently, after 24 hours for the premature formula at both original pH (6.70) and alkaline pH (8.0). Neither the human milk nor the standard formula developed any clot. We feel that premature infant formulas should be used with caution when used via the transpyloric route.

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LOSS OF CALCIUM (CA) FROM THE INTESTINE AND SERUM CA IN INFANT AND ADOLESCENT RATS. L.R.J. Nathan, Keith Komnick and M. Kabir Younoszai, College of Medicine, University of Iowa Hospitals and Clinics, Department of Pediatrics, Iowa City, Iowa.

Because in infant rats the intestine is more permeable to Ca than in adults, the infant may be predisposed to hypocalcemia. In suckling (S, 14-15 day old) and adolescent (A, 8 week old) rats we measured net secretion of Ca into the lumen during in situ perfusion of the upper (J) and lower (I) half of the small intestine with a Ca-free solution containing/1: 120 mM NaCl, 5 mM KCl, 25 mM NaHCO₃, and 30 mg of phenol red. Serum Ca was measured before and at the end of the 2 hr perfusion period. The perfusion solution was recirculated through the segments at a rate of 1 ml/min from reservoirs containing 7 or 25 ml of the perfusion solution, respectively, for the S and A rats. As expected, calcium was secreted into the lumen in all rats. Total net Ca secreted (μ moles/2 hrs) was about the same in S and A rats (mean±S.E., S: J 3.9±0.5, I 4.7±0.5; A: J 3.3±0.5, I 4.9±1.1). However, normalized for differences in size of the segments, net secretion rates (μ moles/hr/g wet wt) were much greater in S than A rats (S: J 8.8±0.9, I 8.5±0.6; A: J 1.1±0.1, I 1.4±0.3). Assuming a serum volume of 5% body weight, Ca loss during perfusion, as % of total serum Ca, was 30% in A and 150% in the S rats. At the end of the perfusion serum Ca (μ moles/100 dl) was reduced in S rats only ($p < 0.05$), but was not in the hypocalcemic range (S: 2.0±0.1; A: 2.2±0.3). Thus, like adults, infant rats can maintain relatively normal serum Ca levels, even in the face of large acute losses from the body.

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EFFECT OF INCREASED ZINC INTAKE ON SERUM ZINC LEVELS IN PREMATURE INFANTS. M. Ormazabal, B. Kirkpatrick, D. Mueller and W. Chan. (Spon. by H. Maurer) Medical College of Virginia, Departments of Pediatrics and Biochemistry, Richmond, Virginia.

Previous studies have shown that zinc levels decrease during the first month of life in premature infants. An attempt was made to raise zinc levels by increasing the oral intake in one of two groups of premature infants who were matched for birth wt $\bar{X}$ = 1440 gm, gestational age $\bar{X}$ = 31.3 wks, onset and volume (cc/kg/day) of feedings and caloric intake (k cal/kg/day). Group I received 1.00 mg Zn/dl of formula while Group II received 0.40 mg Zn/dl of formula. Zinc levels were obtained on study day 0 and every 7th day for 4 weeks and determined by atomic absorption spectrophotometry.

Week	Group I (n)		Group II (n)		P
	ug/dl ± SEM		ug/dl ± SEM		
1	68.50	3.58 (10)	66.67	5.07 (9)	NS
2	68.00	3.82 (10)	62.00	5.07 (10)	NS
3	67.50	2.50 (4)	63.89	4.98 (9)	NS
4	75.00	7.36 (4)	48.75	3.75 (4)	NS

Serum albumin, total protein and alkaline phosphatase levels were similar for each feeding group at each time period as was growth, as measured by weight, length and head circumference gains. Despite a 2.5 fold increase in zinc intake in Group I, serum levels were similar to that of control infants. Factors other than zinc intake must govern the level of zinc in the growing premature infant.