● 554 FETAL ALCOHOL SYNDROME: A MECHANISM FOR GROWTH RETARDATION.

Fayez K. Ghishan, Rashmi Patwardhan and Harry L. Greene, Department of Pediatrics and Medicine, Vanderbilt Medical School, Nashville, Tennessee 37232.

Growth retardation (GR) is a principle feature of Fetal Alcohol Syndrome (FAS). GR is of prenatal onset and generally there is no postnatal catch-up growth. Since Zinc (Zn) is an essential element for protein synthesis and growth during the critical prenatal and postnatal periods, we postulated that ethanol (E) may interefere with placental Zn transport. Therefore, we studied the effect of both acute and chronic (E) ingestion during pregnancy on in vivo placental Zn transport in pregnant rats. Two groups of pregnant rats were studied: 1. Acute E group (rats were given a single dose of E, 4 g/kg as a 25% V/V solution and control received isocaloric dextrose; 2 hour prior to study). 2. Chronic E group (rats were fed Leiber-DeCarli 5% ethanol diet from the 4th to 20th day of pregnancy and controls were isocalorically pair fed). On the 20th day of pregnancy, 2 µci of ^{65}Zn was injected intravenously and tissue samples were obtained 10 min. thereafter by caesarean section. Transport of Zn was expressed as percent uptake of injected ^{65}Zn per gm of placental or fetal tissue. Acute E did not affect fetal and placental weights, however, chronic E feeding resulted in 30% decrease in fetal weight ($p < 0.05$) and 18% increase in placental weight ($p < 0.05$). Both acute and chronic E caused a significant decrease, 40% and 30% ($p < 0.05$) in placental and fetal ^{65}Zn uptake respectively. Our results indicate that maternal to fetal Zn transport is depressed by E because of a defect in placental transport of zinc. These results may explain partly the GR seen in FAS.

555 EFFICACY OF DIET AND DRUGS ON DERMATITIS HERPETIFORMIS (D.H.) OF CHILDHOOD.

Giunta A.*, Prampolini L.*, Marzorati D.*, Giannetti A.** (sponsored by F. Sereni). * Dept. of Pediatrics II°, Milano, ** Dept. of Dermatology, Pavia.

40 children (27 F and 13 M) of 3-13 years of age with DH were studied from genetic, immunological and gastrointestinal points of view. Genetic analysis of HLA haplotypes showed and incidence of HLA B8 of 50% and DW3 of 59.3%. Immunopathology of the skin demonstrated the presence of granular IgA deposits alongD.E.J. Immune complexes were present in all cases. Subtotal mucosal atrophy was present in 32% and partial atrophy in 50% of the children. 35 patients were put on a strict dietary regimen for at least 12 months. The results were as follows: jejunal mucosa became normal in all patients indipendently of HLA haplotype. Gluten challenge reinduced mucosal atrophy and mild skin lesions. Diet alone induced complete regression of the skin lesionsin 43%; in the other 57% diet did not permit complete control of the skin lesions but allowed a 50% or more reduction in the mean dose of dapsone . 5 patients treated only with dapsone showed a satisfactory response of the skin lesion with no effect on the jejunal mucosa.

● 556 INTESTINAL CALCIUM-BINDING PROTEIN IN THE DEVELOPING RAT DUODENUM.

W.A. Gleason and G.L. Lankford, Department of Pediatrics, University of Texas Health Science Center at San Antonio (Spon. By M.K. Park).

To monitor the maturation of the Vitamin D dependent active calcium transport mechanism in neonatal rat intestine, calcium-binding protein (CaBP) was measured in duodena of suckling and weanling rats. Duodena from pylorus to ligament of Treitz were removed from pups from 1 to 7 weeks old, weighed, rinsed and homogenized. The 30,000 xg supernate of each homogenate was assayed for calcium-binding activity using the Chelex-100 competitive radiocalcium binding assay, for CaBP by radial immunodiffusion using an antibody raised in chickens against purified rat intestinal CaBP, and for total protein.

Age (Wk)	Ca Bound (umol)	CaBP (ug)	Total Protein (mg)
1	475 ± 38	500 ± 90	7.1 ± 0.3
2	517 ± 58	287 ± 41	7.3 ± 0.5
3	1249 ± 46	2280 ± 258	10.1 ± 0.4
4	1173 ± 44	2316 ± 170	10.1 ± 0.3
5	1159 ± 35	1543 ± 35	9.1 ± 1.0
6	700 ± 19	747 ± 21	10.4 ± 0.2
7	756 ± 15	899 ± 45	9.8 ± 0.2

Mean Values Per Gram Wet Weight of Duodenal Segment ± SE

The marked enhancement of calcium binding activity and immunoreactive CaBP noted at weaning (Wk 3), corresponds with 1) enhancement and localization of active calcium transport characterizing functional maturation of the neonatal rat intestine and 2) maturation of the vitamin D endocrine system.

● 557 DECREASED BONE MINERAL CONTENT (BMC) IN BREAST-FED INFANTS WITHOUT SUPPLEMENTAL VITAMIN D (D): "CATCH UP" MINERALIZATION AT 6 MONTHS AND ONE YEAR; POSSIBLE EFFECTS ON LENGTH

F.R. Greer, J.E. Searcy, R.S. Levin, J.J. Steichen, R.C. Tsang, U Cincinnati

We previously reported a double blind randomized prospective study of 9 exclusively breast-fed infants without supplemental D (placebo, plac) and 9 exclusively breast-fed infants on supplement of 400 IU D/d (suppl.); 12 infants were fed exclusively Similac 20 cal/oz (form). As reported, at 3 mos plac had significantly lower BMC (modified photon absorptiometry) and serum 25-OH D. By 6 mos, serum 25-OHD had decreased and was still lower in plac vs suppl. (12.9±4.0 SE vs 32.7±4.1ng/ml. $p<.01$). BMC at 6 mos was 70±6, 75±5, and 94±7mg/cm for plac suppl. and form; plac and suppl. did not differ; plac was less than form ($p<.05$). After 6 mos, the study was unblinded, infants received solids and breast-fed infants received 400 IU D/d. 10 form and 13 breast-fed infants were seen at 12 mos, when serum 25-OHD, parathyroid hormone, calcitonin, Ca, P, Mg and alkaline phosphatase did not differ between plac and suppl. BMC at 12 mos was 120±19, 108±20, and 132±8mg/cm for plac, suppl. and form and did not differ; grps did not differ in wt, but plac was shorter (73.3±.38cm) than suppl. (75.8± .89cm, $p<.1$) or form (75.8±0.32cm, $p<.02$); one way analysis of variance for 3 grps, $p<.05$. Thus, vitamin D supplements may be necessary for optimal bone mineralization in breast-fed infants but "catch-up" mineralization may occur in infants not given D initially. We speculate that D supplements may also be necessary for optimal body length in breast-fed infants.

558 GASTROINTESTINAL CLEARANCE OF ALPHA-1-ANTITRYPSIN: A METHOD FOR DIAGNOSING PROTEIN-LOSING ENTEROPATHY.

B. Grill, T.Tinghitella, C.Hillemeier, J.Gryboski. Yale Univ. School of Med., Dept. of Pediatrics, New Haven, CT.

Because of the unavailability of alternate methods to determine protein-loss, we measured the gastrointestinal clearance of alpha-1-antitrypsin (AAT), by a modification of a European technique. We studied eight patients, three with hypoalbuminemia and disorders suggestive of protein-losing enteropathy, and five with or without hypoalbuminemia and gastrointestinal disorders not necessarily associated with protein loss. Clearance of AAT was calculated from the following equation: $C_{AAT} = (FxV)/P$ where C is in cc/days; F = fecal concentration of AAT in cc/l; V = fecal volume in cc/day; P = serum AAT in mg/l. Stools were collected over a three day period and serum AAT obtained on each of three days and averaged in some patients, and from two or one specimen in other patients. The stool was homogenized, and a one gram aliquot resuspended in distilled water. AAT was measured by radioimmunodiffusion using standard techniques. Serum AAT was measured by standard techniques.

Pts	$\bar{x}$ ALB	$\bar{x}$P	$\bar{x}C_{AAT}$	$R(C_{AAT})$
1-3	3.0 mg%	336 mg%	149.0 cc/d	70.9-277.2
4-8	3.7 mg%	341 mg%	4.1 cc/d	1.0- 12.5

We conclude that a three day stool collection with determination of C_{AAT} may be useful in identifying children with protein losing enteropathy.

● 559 GROWTH AND METABOLIC RESPONSE OF PRETERM INFANTS FED PRETERM AND MATURE BREAST MILK

Steven J. Gross (Spon. by G.W. Brumley) Duke Univ. Med. Ctr. Dept. Ped., Durham.

25 appropriately grown infants with birthweight <1600 gm were assigned randomly to one of 3 sources of nutrition from birth: 1. Cow's milk formula (67 kcal/dl, 2.0 gm/dl protein). 2. Preterm breast milk, obtained from donors 1-8 wk following preterm delivery, collected and pooled separately by postpartum week. Each week infants received milk corresponding to their postnatal age. Protein concentration decreased from 2.3 gm/dl (wk 1) to 1.2 gm/dl (wk 8). 3. Mature breast milk, obtained from donors >3 months following term delivery, containing 1.0 gm/dl protein. All infants were fed 180 ml/kg/day until achieving a wt of 1800 gm. Growth and biochemical measurements were made weekly. Data indicate mean ± SD.

	Cow's milk formula (n=9)	Preterm breast milk (n=8)	Mature breast milk (n=8)
Birthweight (gm)	1373 ± 261	1278 ± 299	1263 ± 311
Gestational Age (wk)	31.2 ± 1.5	31.0 ± 2.1	30.4 ± 2.3
Days to regain BW	9.3 ± 4.5	13.1 ± 2.2	23.3 ± 9.0*
Daily weight gain from BW to 1800 gm	27.5 ± 4.3	22.5 ± 8.0	16.6 ± 4.9**
Length (cm/wk)	0.80 ± 0.17	0.70 ± 0.12	0.51 ± 0.18*
Head circ. (cm/wk)	0.80 ± 0.31	0.82 ± 0.10	0.70 ± 0.08

Mean values for BUN, serum sodium, protein, albumin, and blood pH were similar in the 3 groups. Infants fed preterm breast milk demonstrated similar growth and metabolic response as infants fed cow's milk formula.

*$p<.01$ vs cow's milk and preterm breast milk;**$p<.01$ vs cow's milk.