† 570 SERIAL UV RADIATION EFFECTS ON VITAMIN D METABOLITES IN BLACKS & WHITES

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Factors affecting 25-hydroxyvitamin D (25OHD) response to chronic sub-erythemal ultraviolet (UV) exposure have not been studied in adults or infants. This study was a pilot one in adults before infant studies: we hypothesized that serum parathyroid hormone (PTH), vit. D binding protein (DBP), 25OHD & race affect the 25OHD response to chronic sub-erythemal UV. Sequential sub-erythemal doses of UV-B (280-315 nm) were given to blacks & whites, ages 20-35 yrs. PTH was measured by radioimmunassay RIA, C-terminal; calcitonin (CT) by RIA, 1-32 CT; 25OHD, protein binding assay; DBP, immuno-diffusion. Initial UV-B dose was below minimal erythemal dose for most sensitive white skin with 10% increase per exposure for 4 wks. Total body UV was given bi-wkly for 6 wks. Blood was drawn wkly; baseline & final blood were obtained 1 wk pre- & post- UV; 8/subject. Serum 25OHD rose from 21 ± SE 2.2 ng/dl, to 27 ± 1.6 at wk. 1, 36 ± 2.0 at wk. 4, & 47 ± 2.7 at end. Δ 25OHD was post- minus pre- 25OHD. No significant correlations were found between Δ 25OHD & basal serum PTH, DBP & 25OHD. White 25OHD was higher than blacks throughout the study (p<.001). White 25OHD was 27 ± 1.6 pre- & 52 ± 2.7 post-; black 25OHD was 11 ± 2.4 & 41 ± 4.3 respectively. The 25OHD response vs. time was similar for whites & blacks, with slope 4.12 for whites, r=.979 (p <.001) & 4.49 for blacks, r=.993 (p<.001). No correlations were found between Δ 25OHD & sex, Ca, Mg, P, CT & ionized Ca. Thus 25OHD response to chronic sub-erythemal UV radiation is independent of basal PTH, D binding protein, 25OHD, & race. We speculate that infant response might be similar.

● 573 AMINO ACIDS ARE POTENT INHIBITORS OF BILE ACID UPTAKE BY LIVER PLASMA MEMBRANE VESICLES ISOLATED FROM SUCKLING RATS.

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Elevated serum bile acid concentrations in the developing animal are due, in part, to impaired hepatic uptake. Since amino acids (AA) which undergo Na^+-dependent transport interfere with taurocholate (TC) uptake by basolateral liver plasma membrane vesicles (LPMV) from adult rats (Gastroenterology 84:1364, 1983), we studied their effects on TC uptake from suckling (14d) rats. The initial velocity of Na^+-dependent TC uptake was markedly inhibited by the Na^+-dependent AA L-alanine (ALA) or L-glutamine (GLN) which employ separate transport systems (A and N) but not by the Na^+-independent AA 2-aminobicyclo (2,2,1)-heptane-2-carboxylic acid (BCH):

	2.5mM ALA	5.0mM ALA	1.0mM GLN	5.0mM GLN	5.0mM BCH
TC Uptake (% Control)	63±19 p<0.001	37±29 p<0.001	62±22 p<0.001	33±20 p<0.001	102±11 NS

Summary: Physiologic concentrations of Na^+-dependent AA (measured total portal vein conc. ~ 3.5 mM) markedly inhibited TC uptake in the developing rat probably by dissipation of the transmembrane Na^+ gradient. Since TC uptake velocities by vesicles from 14d rats are only 30% of adult values (Gastroenterology 84:1399, 1983), further impairment by Na-dependent AA profoundly reduced uptake to less than 10% of normal adult rates. Therefore, inhibition of bile acid uptake by AA may contribute to physiologic cholestasis of infancy and hyperalimentation-induced cholestasis.

571 SUPPLEMENTED HUMAN MILK (SPHM) EFFECT ON GROWTH & SERUM BIOCHEMISTRIES IN PRE-TERM (PT) INFANTS.

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Optimal PT infant feeding is unclear. Human milk (HM) may not support rapid extrauterine PT infant growth. We studied growth rate & serum chemistries in PT infants fed HM supplemented with protein, minerals, & trace elements in 49 well PT infants (BW 1000-1500 gm, GA 28-34 wks), assigned to 5 feeding groups: Preterm HM (PHM), PHM with supplement (SPHM), banked HM (BHM); or premature formula (PF), from full enteral intake to 1800 gm wt Significant differences were as follows:

	cc/kg	cal/kg	Wt. gm/d	Length cm/d	Hc cm/d	AMC mm/d	n
PMM	173	125	21.5	.12	.03	.04	11
SPHM	161	125	24.8	.13	.04	.05	11
BHM	182	128	17.7	.11	.03	.04	10
SBHM	166	132	22.7	.15	.05	.05	10
PF	167	129	33.4	.22	.07	.08	7
P	<.005	<.3	<.001	<.001	<.004	<.002	

Formula fed infants grew fastest; slowest on BHM. Those on SPHM grew faster than non-supplemented controls. There were no differences among feeding tolerance or NEC. Serum BUN, Ca & alk. phos. levels among groups were different.

Human milk (both PT & banked) supplemented with protein, minerals & trace elements gave PT infants 1000-1500 gm wt. gain. Formula fed infants grew significantly greater than either non-supplemented or supplemented breast fed infants, suggesting that the supplement may need further adjustment.

574 RESPONSE OF MICROVILLAR α-AND β-SACCHARIDASES TO STARCH, SUCROSE AND GLUCOSE POLYMERS IN SOLID AND LIQUID DIETS.

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Glucose polymers (GP) were found to influence activity of microvillar sucrase (S), maltase (M), glucoamylase (G) and lactase (L), when fed to adult rats in solid diets at 70 cal% level (Ped. Res. 17:1983). Because of the clinical importance of GP in infant formulas we determined the effect of GP, sucrose and starch at levels provided in infant formulas, i.e., at 40 cal%. Sugars were included either in solid diets (SD) or in liquid soy protein based formulas (LF) and fed for 48 hours to rats that were previously fed a diet containing 5 cal% of carbohydrate for 7 days. Activities of S,M,G, and L were measured in the upper (UJ) and lower jejunum (LJ). Each sugar caused a significant increase in the activity (expressed per protein or per segment) of all enzymes tested. Although in the UJ the activity of S was increased equally by SD and LF, in the LJ the increase was greater with LF, making the UJ to LJ gradient disappear. All SD evoked an equal response of L activity in UJ and LJ, whereas LF evoked a comparable increase in LJ but considerably less in UJ. The effect of LF on M and G was comparable to SD at both levels of jejunum. CONCLUSIONS: Our studies show further evidence of the responsiveness of α-and β-saccharidases to GP and sucrose. The physical properties of the diet, i.e., SD vs. LF influence the locus of maximal response. Studies of the changes of digestive capability of the small intestine should include both levels of the jejunum.

572 PROXIMAL MUSCLE WEAKNESS RESPONDING TO SELENIUM THERAPY: A CASE REPORT.

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Van Rij reported proximal muscle weakness in a case of selenium (Se) deficiency in New Zealand in 1979. Since then cardiomyopathy, but not proximal muscle weakness, has been reported in the USA. A 33 y.o. white female was on home parenteral nutrition for 4½ years due to a duodenocecostomy secondary to a traumatic jejunoilectomy. During the first year she noted proximal muscle weakness which did not improve over the next 3½ years. She noted weakness with arm lifting and inability to rise from a squatting position. During a study of Se function, glutathione peroxidase levels were found to be profoundly low in plasma and blood cells.

Glutathione Peroxidase Levels (GSHPx)

	Plasma (u/ml)	Platelet (u/mg prot)	Granulocyte (u/10^8 cells)	Mononuclear (u/10^8 cells)	(u/gmHgb)
Normal	.19-.33	8.13-201	.92-3.3	7.5-25.0	20.3-25.9
Before	.02	10.8	.35	.94	1.06
After 4 wks	.24	273.3	1.84	17.9	5.96
After 12 wks	.21	237.5	3.05	7.68	25.1

Plasma Se level initially was 32.5 ng.ml (nl=60-120). After 3-4 weeks of treatment with Se (400 μg/d IV) as selenous acid, the patient's muscle strength testing markedly improved as the GSHPx activities in plasma, platelets, granulocytes, & mononuclear cells became normal. Red cell GSHPx activity approached normal levels much more slowly, and may not be a sensitive reflection of whole body selenium status.

575 METOCLOPRAMIDE INCREASES LOWER ESOPHAGEAL SPHINCTER PRESSURE (LESP) AND REDUCES THE NUMBER OF EPISODES AND DURATION OF REFLUX IN INFANTS WITH GASTROESOPHAGEAL REFLUX (GER),

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Thirteen infants (age range 1-24 mo; x-6.8 mo) with GER were studied with esophageal manometry (8) and 24 hr pH probe monitoring (13) to determine what effect metaclopramide has on LESP and reflux. Esophageal manometry was performed before and after intravenous metoclopramide (0.1 mg/kg). Twenty-four hr pH monitoring was divided into 2, 12 hr periods: A, no drug, and B, drug, metoclopramide 0.1 mg/kg/dose orally, given 30 min before every other 3 hr feeding. Mean LESP before metoclopramide, 17.9 mmHg; after, 26.3 mmHg (p<0.01). Statistical significance between A and B was achieved for the $\bar{x}$ number of episodes of reflux (39.0 +23.0 vs 22.2 + 11.6) (p<0.025) and the $\bar{x}$ duration of reflux (134.9 +73.5 min vs 74.9 + 42.1 min) (p<0.01), but not for the $\bar{x}$ duration of each reflux episode (3.46 min vs 3.37 min) (p>0.5). Mean differences in the number of episodes of reflux and the duration without and with drug 1, 2, and 3 hrs postprandially were: 10.9 ± 8.58 vs 3.62 ± 5.53 (p<0.01) and 26.8 ± 22.3 min vs 7.69 ± 11.4 min (p<0.01); 12.8 ± 11.0 vs 3.31 ± 3.68 (p<0.005) and 48.5 ± 42.7 vs 15.9 ± 11.2 min (p<0.01); 9.31 ± 6.65 vs 5.00 ± 3.70 (p<0.05) and 36.8 ± 24.8 min vs 18.6 ± 16.0 min (p<0.025). We conclude that in infants with GER metoclopramide: 1) increases LESP, 2) decreases the total number of episodes of reflux, and 3) reduces the total time esophageal pH is <4.0. These later effects occur primarily during the first and second postprandial hours.