847 EFFECT OF VARIOUS CARBOHYDRATE (CHO) DIETS ON FASTING TRIGLYCERIDE (F TRIG) AND 24 HOUR TRIGLYCERIDE CONCENTRATIONS (TRIG IC). Mark M. Danney, John T. Hayford, Robert G. Thompson, University of Iowa College of Medicine, Department of Pediatrics, Iowa City, Iowa

The conclusion that a high CHO diet results in hypertriglyceridemia has been based entirely on F Trig values. This study was designed to evaluate the effects of variations in CHO intake on Trig IC as well as F Trig. Four diets were studied: 45% sucrose (A), 65% sucrose (B), 45% corn syrup (C), and 65% corn syrup (D). All diets included 15% protein and 300 mg. of cholesterol. The eight subjects consumed the constant liquid diets in random order (Latin square design) for 10 days prior to the 24 hour constant blood withdrawal study which provided the mean 24 hour triglyceride concentration or Trig IC.

	A	B	C	D
Mean F Trig (mg/dl)	61	92	56	88
Mean Trig IC (mg/dl)	88	101	72	74

Statistics by Analysis of Variance

	A+B vs C+D	A+C vs B+D	A-B	A-C	A-D	B-C	B-D	C-D
F Trig	NS	*	*	NS	*	*	NS	*
Trig IC	*	NS	NS	*	NS	*	*	NS

* = p<.05, NS = Not Significant

Conclusions: 1. The F Trig does not accurately reflect the Trig IC. 2. F Trig levels correlate with amount of carbohydrate. 3. Trig IC levels correlate with type of carbohydrate with higher Trig IC occurring during sucrose ingestion. 4. A high carbohydrate diet per se does not result in hypertriglyceridemia.

848 FETAL GROWTH RETARDATION (FGR) AND HYPERINSULINISM: EVIDENCE FOR AN ABERRANT INTRACELLULAR RESPONSE TO INSULIN. A. Joseph D'Ercole, Louis E. Underwood, John Groelke, and Arlane Plet, Dept. of Ped., Univ. of North Carolina, Chapel Hill.

A term newborn girl with FGR (1616 gm) and phenotypic features of Leprechaunism had hyperglycemia and marked persistent hyperinsulinism (232-3000 uU/ml). After sudden death at 7 weeks, postmortem exam revealed findings nearly identical to those described by Donahue and Uchida (J. Pediatr. 45:505, 1954). Studies were undertaken to distinguish between an abnormal insulin and an abnormal cellular response to insulin. Evidence that the patient's immunoreactive insulin (IRI) behaved chemically and biologically like normal insulin is as follows:(1)By gel chromatography, 92% of the IRI migrated as insulin; 8% as proinsulin.(2)Serum IRI reacted normally with insulin receptors in human placental cell membranes.(3)Serum IRI was degraded at a normal rate by a crude preparation of insulin glucagon protease.(4)Serum insulin-like activity in a rat fat pad bioassay was proportional to serum IRI. The patient apparently had normal insulin receptors, since her skin fibroblasts could specifically bind ^{125}I insulin as well as those of newborn controls (2-7%/1x10^6 cells). On the other hand, preliminary studies indicate that the patient's fibroblasts had a significantly reduced capacity to incorporate ^{3}H-thymidine in response to either insulin or serum. These results infer that the explanation for this patient's hyperinsulinism, insulin resistance, and presumably FGR rests with a defective intracellular response to insulin and, possibly to other growth factors.

849 EFFECTS OF P LOADING AND INCREASED ENDOGENOUS PARATHYROID (PTH) DURING "EXCHANGE" TRANSFUSIONS ON URINARY P AND CYCLIC AMP (CAMP), Edward F. Donovan, David R. Brown, I-Wen Chen, J. Robert Johnson, C. Bobik and Reginald C. Tsang, Univ. of Cincinnati and Pittsburgh & Good Samaritan Hospital, Cincinnati.

Citrate complexing of Ca during neonatal "exchange" blood transfusions is associated with stimulation of PTH; theoretically P loading occurs from partially hemolyzed or P buffered blood. During exchange transfusions in 31 neonates (gestation 27-41 wks, postnatal ages 0.1-8d), serum ionized Ca and Mg decreased (paired t,p<.05), P increased from 5.23±.26 (mean+SEM) to 6.64±.29 mg/dl (paired t,p<.01) and PTH (radioimmunoassay, N terminal) increased from 17.5±4.6 to 113.0±21.5 ul-Eq/ml (paired t, p<.05). No significant change in serum CAMP (Gilman) was noted, 51.56±10.29 and 55.03±8.33 pmol/ml, n=9. Following transfusion, n=24, there was an early (2.6±.3 hrs) increase in urinary CAMP (UCAMP) concentration from 962±207 to 1346±208 pmol/ml (paired t, p<.05), and a later (3.9±6 hrs) increase in urinary P (UP) concentration from 14.6±3.6 to 33.7±6.6 mg/dl and UP excretion from .024±.012 to .05±.02 mg/min (paired t,p<.05). Infants<34 wks gestation (n=10) vs ≥34 wks (n=10) did not differ in Δ(maximum minus prestudy value) UP (19.87±.14 vs 17.5±4.07 mg/dl) or ΔUCAMP (43.19± vs 104.8±39.7 pmol/ml). Infants ≤2 days old (n=10) vs >2d (n=11) did not differ in ΔUP (17.26±6.37 vs 20.65±5.9); ΔUCAMP was lower in older infants (116.3±31.3 vs 26.1±14.6 pmol/ml). Thus, newborn infants, regardless of gestational age or postnatal age, appear to respond to P loading and increased endogenous PTH by increasing UP and UCAMP while serum CAMP remains unchanged.

850 N.M. DRAYER, R. van DAMMEE-LOMBAERTS, C. ROUWE Department of Pediatrics, University Hospital, Groningen (Introduced by A.M. Bongiovanni)
Hypercalciuria in children with diabetes mellitus.

In our own control group of 58 healthy children the urinary calcium excretion (mg/kg bodyweight/24hr) was for 56 children 2.4±1.4 (S.D.) and for 4 children more than 2 S.D. above the mean. In 15 out of the 47 diabetic children who were all receiving insulin therapy, the urinary excretion of calcium was 2 S.D. above the mean of the control group. The urinary calcium excretion (mg/kg/24hr) correlated with the glucosuria: 2.1 + 0.9 urinary glucose (g/kg/24 hr); n=32, r=0.470, p less than 0.01. On the basis of this correlation the hypercalciuria in 10 of the 15 hypercalciuric children could not be attributed to glucosuria. 9 out of the 10 hypercalciuric children, where the hypercalciuria could not be attributed to glucosuria, showed on follow-up a persistent hypercalciuria. In these hypercalciuric children the tubular reabsorption of phosphate and the TmP/GFR were not different from the other diabetic children, but after a calcium load the TmP/GFR was raised significantly in the hypercalciuric group only. The urinary cAMP/creatinine ratio in the hypercalciuric children during a 2 hour fast was below that of healthy children. Plasma PTH was elevated in 3 of 32 diabetic children. Children with diabetes mellitus have an unexplained high frequency of hypercalciuria.

851 PREVENTION OF EARLY NEONATAL HYPOCALCEMIA WITH 25 HYDROXYCHOLECALCIFEROL(25OHD$_3$). Alan R.Fleischman, John F. Rosen, MarleneE.Moses, Albert Einstein Coll.Med., Montefiore Hosp.& Med.Ctr.& Bronx Lebanon Hosp., Dept.Ped., The Bronx, NY

This study utilizes oral 25OHD$_3$ in an attempt to prevent early neonatal hypocalcemia in newborn rats and humans.

Pregnant rats were fed a normal diet or one deficient only in vitamin D. Pups were divided into those litters treated with 25OHD$_3$ orally, 0.5µg/d, for the first five days of life, and those given sodium chloride as controls. Neonatal rat sera were obtained on the sixth day of life with samples from a single litter pooled for analysis. Data are expressed as mean ± S.E.M.:

Maternal Diet:	Normal				Vitamin D Deficient			
	Ca++ mg/dl	Ca_T mg/dl	Mg meq/l	25OHD ng/ml	Ca++ mg/dl	Ca_T mg/dl	Mg meq/l	25OHD ng/ml
Control	3.41 ±0.20	8.09 ±0.44	1.81 ±0.25	12.5 ± 0.7	3.19 ±0.15	5.95 ±0.45	1.49 ±0.05	6.2 ±0.2
25OHD$_3$ treated	3.36 ±0.07	8.19 ±0.49	1.79 ±0.77	38.8 ± 5.8	3.70 ±0.22	8.50 ±0.47	1.89 ±0.20	30.8 ±4.3

The significant decrease in serum levels of ionized calcium, magnesium and 25OHD in the offspring of vitamin D deficient rats was corrected by treatment.

Preliminary experience in human premature neonates treated for the first five days of life with oral 25OHD$_3$, 2µg/kg/d, revealed comparable data. Serum levels of ionized calcium increased over this period and urinary calcium:creatinine ratios remained low (< 0.1).

These data indicate:1-oral 25OHD$_3$ is well absorbed;2-oral 25OHD$_3$ in this dose does not cause hypercalcemia or hypercalciuria; and 3-most significantly, oral 25OHD3 may prevent early hypocalcemia.

852 ESSENTIAL FATTY ACIDS (EFA) IN CORD BLOOD OF THIRD TRIMESTER INFANTS AND MATERNAL PLASMA. Zvi Friedman, Edward L. Lamberth, Abraham Danon, William J. Mann (Spon. by Nicholas M. Nelson). Penn State Univ. Coll. Med., M. S. Hershey Med. Ctr., Dept. Ped., Dept. Ob/Gyn, Hershey, Pa. and Dept. Med., Vanderbilt Med. Ctr., Nashville, Tenn.

EFA are incorporated into brain lipids and serve as prostaglandin (PG) precursors. Increased CNS morbidity in low birthweight infants is well recognized. Plasma levels and EFA were measured in phospholipids (PL), cholesterol esters (CE), triglycerides (TG) and free fatty acids (FFA) by TLC and GLC in 16 postpartum mothers (28-44 weeks) and in the umbilical vein and artery of 32 newborns. Groups of eight 24-33, 34-37, 38-42 and 43-44-week-old infants were studied. Increased PL, CE and TG ($p<0.001$) were noted in maternal plasma compared with cord blood; linoleic acid was lower ($p<0.001$) in cord blood PL, CE and FFA. EFA derivatives-Δ-8,11,14-eicosatrienoic, arachidonic and docosahexaenoic acids were higher in cord blood ($p<0.001$). Total polyenoic EFA increased with advanced gestation, and at term, was close to maternal levels. Δ-5,8,11-eicosatrienoic acid (elevated in EFA deficiency) was elevated in cord blood as compared with maternal values ($p<0.001$); other criteria of EFA deficiency were absent. The study demonstrated that during the third trimester, fetal EFAs are elongated and desaturated. These higher polyenoic acids are incorporated into lipids in the developing CNS and also serve as substrate for PGS biosynthesis. The lower linoleic acid level in the fetus may play an important role in transplacental transport of EFA.