1261 RETENTION AND ELIMINATION OF ^{65}Zn IN ACRODERMATITIS ENTEROPATHICA: A STUDY WITH WHOLE BODY COUNTING. Olli Simell, Matti Suomela, Tua Rahola, Tuomas Westermarck (Spon. by David Rosenblatt). Childrens Hospital, University of Helsinki, and Institute for Radiation Protection, Helsinki, Finland.

Hypozincemia is a constant finding in patients with acrodermatitis enteropathica (AE). Oral supplementation with large doses of zinc corrects both hypozincemia and clinical manifestations. We measured changes in serum zinc values in 3 healthy subjects and 4 patients with AE after an oral dose of zinc sulphate (1 mg/kg); increments were identical in patients and controls. We then studied zinc retention and elimination after a tracer dose of 37 kBq (1 μCi) of carrier-free $ZnCl_2$ in 4 patients (aged 5-40 years, 1 male) and four controls (32-41 years, 2 males) using whole body counting technique. The radiation dose was accepted by the Institute for Radiation Protection and remained clearly below the background radiation dose. Body distribution of the label was similar in patients and controls. Zinc retention and biological half-life were calculated for counts between 7 and 120 days of the study by fitting single exponential function to the data. The patients showed a 23±1% retention (mean±SE) and 109±11 days' biological half-life. In female controls the values were 81±1% and 159±16 days, and in male controls 45±1% days, respectively. We conclude that tracer amounts of zinc are poorly absorbed in AE, and that the turnover of zinc appears to be faster in the patients than in controls also while patients are on zinc supplementation.

1262 EEG ABNORMALITIES IN CHILDREN IN IDDM. Carol Singer-Granick, Patricia Crumrine, Allan Drash, Dorothy Becker, Department of Pediatrics, University of Pittsburgh, School of Medicine, Pittsburgh, PA

We obtained EEGs on 74 children (39 F, 35 M) with IDDM ranging in age from 10.4-19.8 yrs (mean 15.3) and duration of IDDM from 1.2-17.1 yrs (mean 8.8). Abnormal EEGs were found in 14 (18.9%), one of whom had poorly controlled seizures. Abnormalities were paroxysmal, either focal 7 (50%) or generalized 2 (14%), while 5 (36%) exhibited background slowing. There was no difference between those with abnormal and normal EEGs comparing glycosylated hemoglobin (HbA1) at the time of the study or the prior 2.1±1 yr (12.5% vs 12.9%). Nor were there differences in mean blood glucose (measured hourly over 48 hrs) or hypoglycemia (< 60 mg%) during this period, MAGE, or labile HbA1. We found no difference in mean duration of illness or age of onset in the 2 groups. However, those children developing IDDM before age 5 yrs were more likely to have abnormal EEGs (p=.01). Matching those with abnormal and normal EEGs by duration, age, and sex, there again were no differences in the above variables. Only 25% with abnormal EEGs also had abnormal fluorescein angiograms, compared to 60% in the normal EEG group. Thus, despite a lower prevalence of total EEG abnormalities than in prior reports in IDDM (25-60%) we find a similar increase in paroxysmal features (11%) compared to the 1-2% reported for normal children. These are more likely to occur in children with age of onset <5 yrs and appear to be unrelated to any available measure of glycemic control or microvascular complications. Prospective studies will be required to elucidate the etiology and clinical significance of these findings.

●1263 LACTIC ACID TOLERANCE IN THE NON-HYPOXIC FETAL LAMB. John W. Sparks, William W. Hay, Jr., Giacomo Meschia, Frederick C. Battaglia. Division of Perinatal Medicine, University of Colorado School of Medicine, Denver.

Lactic acid is considered in contradictory ways: as a normal fetal nutrient or as an indicator of fetal distress. Fetal lactic acidemia has been induced experimentally by hypoxia, combining effects of hypoxia and lactic acidemia. We chose to separate these effects by infusing lactic acid into 14 late gestation fetal lambs via a continuous infusion technique, and into 5 lambs using a lactate clamp technique. Animals (Fetal Weight=3.5±0.2 kg) had catheters in the umbilical vein, fetal pedal artery, pedal vein and brachial vein, and, in 6 animals, the fetal hepatic vein. Animals were well oxygenated and well nourished after recovery for at least 7 days. Lactic acid infusion into the fetal brachial vein at an average of 20 mg/min for 90-180 min rapidly established a new metabolic steady state, increasing arterial lactate by 2.8±0.36 mM*, and decreasing arterial pH from 7.36±0.006 to 7.29±0.02*. Umbilical lactate uptake determined by the Fick Principle fell from 7.3±1.2 to 3.1±1.7 mg/min* during infusion, and fetal arterial glucose increased from 1.02±0.06 mM to 1.13±0.05 mM*, decreasing both umbilical glucose uptake from 18.2±1.4 to 14.9±2.7 mg/min* and umbilical glucose/oxygen quotient (GOQ) from .51±.05 to .39±.02*. During infusion, hepatic lactate/oxygen quotient rose from 0.31±0.08 to .77±.12*, and hepatic GOQ reversed from +0.33±.05 (consumption) to -0.56±0.27* (production). [*=p <0.05, paired].

Rapid continuous fetal lactic acid infusion is well tolerated by the non-hypoxic fetal lamb, and alters fetal metabolism of glucose and lactic acid.

●1264 CYCLICAL SERUM 25-HYDROXYVITAMIN D PARALLELLING SUNSHINE EXPOSURE IN EXCLUSIVELY BREAST-FED INFANTS: MIRROR IMAGE IN SUMMER VS WINTER BORN. B. Specker, D. Buckley, J. Searcy, R. Levin, R.C. Tsang. U. of Cincinnati.

Previously we reported low serum 25-hydroxyvitamin D (25OHD) in breast fed infants without vitamin D supplements. However prospective serum 25-OHD vs sun exposure studies have not been done in relation to season of birth. We hypothesized that breast-fed infants <1 yr of age have serum 25-OHD varying directly with sun exposure & that opposite patterns occur in summer vs winter-born infants. 25 term infants exclusively breast-fed without vitamins were followed longitudinally from birth for > 6 mos: 13 born in summer and 12 winter. Sun exposure was monitored for 1 wk of every mo until 12 mos. A sun exposure score, previously verified, quantified time & surface area exposed (e.g., 0=no exposure, 5= 13hrs/wk, diaper only). 25-OHD was determined by protein-binding assay (N 11-68ng/ml; CV 11%). Mean (±sem) values by age were:

Summer-born	1mo	3mos	6mos	12mos
Sun exposure	4.0 (0.2)	3.2 (0.4)	1.3 (0.4)	4.9 (0.2)
25-OHD	37 (3)	33 (6)	22 (4)	41 (3)
Winter-born				
Sun exposure	1.5 (0.3)	3.2 (0.5)	4.2 (0.1)	2.3 (0.3)
25-OHD	16 (2)	26 (4)	46 (6)	28 (5)

Monthly exposure scores peaked in summer, were low in winter ($p<.01$), & intermediate in fall & spring. 25-OHD paralleled sun exposure, irrespective of age; 25-OHD was correlated with exposure ($r=.54$, $p<.001$). Thus, summer vs winter-born infants had mirror image patterns of both sun exposure and 25-OHD; large seasonal differences in both sun exposure and 25-OHD were observed.

●1265 SEASONAL CHANGES IN SERUM VITAMIN D BINDING PROTEIN IN INFANCY: RELATION TO SUN EXPOSURE. Bonny Specker, Mona Ho, Reginald C. Tsang. U. Cincinnati.

Vitamin D binding protein (DBP) is the major carrier for vitamin D and its metabolites in serum. Its physiologic regulation is unclear; DBP increases in pregnancy and decreases in cirrhosis; no seasonal variation has been reported in adults. We hypothesized that serum DBP in infants would not vary by season. 41 exclusively breast-fed, non-D supplemented infants <6 mos of age were studied. DBP was measured by radial immunodiffusion: adult range, 276-505 ng/ml, cv 2.9% intra- and 7.6% inter-assay. 25-Hydroxyvitamin D (25-OHD) as an indicator of vitamin D status was measured by protein binding assay. Winter DBP exceeded summer: 377±12 vs 302±7 ug/ml (x±sem, p=.003). Serum 25-OHD exhibited an opposite pattern: 14±2 vs 26±2 ng/ml (p<.001). Maternal DBP did not differ by season: 374 and 373 ug/ml for winter and summer. An ultraviolet exposure score, previously verified, was used to document time and body surface exposed to sun. DBP was inversely related to sun exposure ($r=-.41$, $p=.01$). Infant DBP was significantly & negatively correlated with 25-OHD ($r=-0.38$, $p=0.02$). Two subsequent independent studies, 1 cross-sectional & 1 longitudinal, yielded similar results: infant DBP being significantly higher in winter vs summer. Thus vitamin D binding protein in exclusively breast fed infants is elevated in winter (vs summer), in low sun exposure, and in low vitamin D status as reflected by low serum 25-OHD; we speculate that serum vitamin D binding protein fluctuations are a response to varying vitamin D needs: increased DBP occurs in low vitamin D status to maximize uptake of vitamin D from skin.

●1266 ABNORMALITIES IN VASCULAR ARACHIDONIC ACID (AA) METABOLISM IN THE INFANT OF THE DIABETIC MOTHER (IDM) Marie J. Stuart, Yamaja Setty, Shirazali Sunderji, Sherry Boone, Carolyn Ganley. SUNY, Upstate Medical Center, Department of Pediatrics, Syracuse, N.Y. 13210

Studies of coagulation have not identified the cause(s) for the prothrombotic tendency in the IDM. Our work was designed to evaluate AA metabolism in IDM vessels, and to assess circulating PGI_2 levels in these neonates (n=17) compared to control infants (n=15). When endogenous vascular 6KPGF$_{1\alpha}$(the metabolite of PGI_2) was measured by RIA, control neonates produced 6.5±1.5(1SD) pmol per mg of umbilical arterial tissue. Although IDMs born to mothers in glucose homeostasis were similar to controls (6.4±1.8), IDMs born to mothers with ↑ HbA_{1c} levels demonstrated a decrease in vascular 6KPGF$_{1\alpha}$(4.3±1.1; $p<0.02$). However, when ^{14}CAA was utilized as substrate by vascular microsomes, ^{14}C6KPGF$_{1\alpha}$ was similar in controls (975±478 pmol per mg) and in IDMs born to mothers in normal or abnormal metabolic homeostasis (1199±479; 1092±577). A significant decrease ($p<0.02$) in the circulating plasma level of 6KPGF$_{1\alpha}$was observed in the IDM (0.75±0.24 pmol per ml) compared to controls (1.65±1.2). Correlation in the IDM between endogenous vascular 6KPGF$_{1\alpha}$ production,and plasma levels of PGI_2 was seen ($r=0.7$; $p<0.05$). This latter finding demonstrates that the invitro deficiency in vascular 6KPGF$_{1\alpha}$in the IDM reflects an invivo abnormality as well. Besides its antiplatelet-aggregatory properties, PGI_2 is an endogenous pulmonary vasodilator. The deficiency in PGI_2 could thus contribute not only to a prothrombotic tendency, but to pulmonary vasoconstriction and to the transient respiratory distress seen in the IDM.