1194 PREVENTION OF OSTEOPENIA IN EXTREMELY LOW BIRTH WEIGHT INFANTS (ELBW, BW<1000 GM) WITH SPLIT BOTTLE TOTAL PARENTERAL NUTRITION. Vang Kamtorn, Shyan Sun, AmeIia Bautista (Spons. F. Behrle) UMD-New Jersey Med. School, Division Neonatal-Perinatal Medicine, Newark,New Jersey

Deficiencies of calcium (Ca) and phosphorus (P) have been implicated as etiological factors among others in osteopenia of prematurity. It is not possible to supply the amount of Ca & P normally acquired transplacentally in a TPN solution without forming calcium phosphate precipitate. To avoid this, we used split bottle technique. One bottle of TPN solution contains only Ca (60 mEq/L) and the other only P (26.4 mM/L). Each bottle is infused simultaneously at different site or alternately. We compared the outcome in terms of bone mineralization of single vs split bottle TPN at 8 weeks of age. Single bottle TPN solution (Ca 10 mEq/L P 13 mM/L) was used in previous years

TPN	Year	Pt No	x-ray (+) (%)	Fx (%)	Alk P	Alk P >1000	P
Single	1978-81	20	14 (70)	6 (30)	1270	8	4.2
Split	1982-83	14	4 (29)	0 (0)	721	2	5.7
p				0.056	<0.005	<0.05	<0.005

Bone demineralization and fractures were more common and alkaline phosphatase levels were significantly higher in infants with single bottle group compared to split bottle group. There was no difference in BW, duration of NPO, TPN, IMV, 0_2 therapy, dose of furosemide and number of BPD in two groups. It is our impression that split bottle method of TPN might have contributed to reduce the severity of osteopenia.

1195 GLYCOSYLATED ALBUMIN (GLY-ALB) AND GLYCOSYLATED TRANSFERRIN (GLY-TRANS) AS SHORT-TERM MARKERS OF CONTROL IN DIABETES. Stephen F. Kemp, Timothy R. Horn, Robert H. Creech, (Spon. by Robert C. Boerth), University of South Alabama, Department of Pediatrics, Mobile, AL

Glycosylated hemoglobin (gly-hgb) is widely used as a marker of long term diabetic control (2-3 months), while glycosylated albumin (half-life 14 days) is a marker of short term control (2-4 weeks). We separated glycosylated and non-glycosylated proteins by an affinity column method (Glyc-Affin™, Isolab) and determined levels of albumin (colorimetrically) and transferrin (radial immunodiffusion). Serum was collected from children with type I diabetes before and 10 days after attending a camp session in which blood glucose levels were carefully controlled. Gly-hgb in these subjects ranged from 4.6 to 14.6% (mean ± SEM= 8.1 ± 0.2%). Mean pre-camp gly-alb in 73 subjects was 16.4 ± 0.6% (SEM), which was elevated compared to the mean of levels in 20 non-diabetic control subjects (8.7 ± 0.3% SEM), and correlated well with levels of gly-hgb (r=0.71). After 10 days mean gly-alb fell to 14.6 ± 0.5% (SEM) ($p < 0.00001$), near the predicted post-camp value of 13.4% if control had been ideal for the 10 days. Initial levels of gly-trans in 44 of these subjects ranged from 4.5% to 22.3% (mean ± SEM=11.4 ± 0.6%), and was higher than the mean of 3.8 ± 0.3% (SEM) in 20 non-diabetic controls. Mean post-camp gly-trans fell to 8.2 ± 0.3% (SEM) ($p< 0.00001$), near the predicted post-camp mean of 7.0 ± 0.4%. Both gly-alb and gly-trans appear to be reliable markers of short term glycemic control; gly-trans (half-life 8 days) was more sensitive over this 10-day period than gly-alb.

1196 GLUCOSE, INSULIN AND LIPID STUDIES FOLLOWING CHRONIC GROWTH HORMONE ADMINISTRATION. A.Kershnar, B. Buckingham.(Spon. by Beverly Morgan)Childrens Hospital of Orange County, Orange, CA. 92668 University of California at Irvine, CA. Department of Pediatrics 92714

Fifteen patients with documented growth hormone deficiency treated for 1-5 years with 0.1 units/kg growth hormone IM three times each week were studied with respect to lipid and carbohydrate metabolism. Lipid levels did not vary significantly pre and post treatment except for two patients with familial hyperlipidemia who showed improvement in both lipid levels and glucose tolerance. In the other patients, data confirmed a diabetogenic effect following chronic growth hormone administration. Peak glucose levels and peak insulin levels were significantly higher post therapy at the <0.02 level using the paired "t" test of two correlated samples. Similarly, the sum of insulin values on 5 hour oral glucose tolerance testing suggested relative insulin resistance with significance at the <0.02 levels following 3, 4, and 5 years of growth hormone therapy. One patient showed decreased insulin production following 5 years of growth hormone therapy associated with diminished glucose tolerance and a peak glucose value of 230 mg% on the 5 hour glucose tolerance test. Thus, all patients appear to confirm reports of increased insulin resistance and/or diabetogenic effect associated with prolonged growth hormone therapy.

1197 FATTY ACID OXIDATION BY DEVELOPING RAT INTESTINE IS CONTROLLED BY CHANGES IN MITOCHONDRIAL [NADH]/[NAD]. Robert E. Kimura (Spon. by M. Simmons), Department of Pediatrics, University of Utah Medical Center, Salt Lake City.

At the time of weaning there is an eight-fold increase in palmitate oxidation to CO_2 by rat intestinal mitochondria. Oxidation of acetyl-CoA also increases indicating that the tricarboxylic acid (TCA) cycle may be rate-limiting during the suckling period. An increase in mitochondrial (mito) [NADH]/[NAD] inhibits isocitrate dehydrogenase, a control enzyme in the TCA cycle. In the suckling rat mito [NADH]/[NAD] is five-fold greater than postwean ratios. If the increase in mito [NADH]/[NAD] during the suckling period regulates TCA cycle activity, decreasing the mito [NADH]/[NAD] should increase TCA cycle activity and fatty acid oxidation. We have determined that high concentrations of potassium inhibit ketone oxidation which results in a decrease in mito [NADH/NAD] by altering the equilibrium of ß-hydroxybutyrate dehydrogenase. The oxidation of palmitate and acetate by mitochondria of suckling rat small intestine is activated by the addition of high concentrations of potassium (100 mM) to the mitochondrial isolation mixture. These data suggest that the fatty acid oxidative mechanism is present during the suckling period and an increased mito [NADH]/[NAD] during this period decreases TCA cycle activity and fatty acid oxidation.

AGE (Day)	Estimated Mitochondrial [NADH]/[NAD]	C16CoA→CO_2 Control	C16CoA→CO_2 +K	AcetylCoA→CO_2 Control	Acetate→CO_2 +K
		(n mol CO_2/mg prot/hr + S.E.M.)			
13-18	4.8+0.7	3.6+0.7	26.1+2.9	9.0+1.8	69.5+13.0
25-Ad	1.0+0.2	33.5+3.7	34.4+2.7	69.5+5.1	93.0+ 6.4

●1198 GLUTAMINE IS THE PREFERRED OXIDATIVE SUBSTRATE FOR SMALL INTESTINE OF SUCKLING RAT. Robert E. Kimura (Spon. by M. Simmons), Department of Pediatrics, University of Utah Medical Center, Salt Lake City, UT.

We determined the rate of oxidation of glutamine and ß-hydroxybutyrate (ßHß), the preferred substrates in adult intestine, in developing rat. ßHß oxidation is low during the suckling period and increases four-fold at the time of weaning. In contrast glutamine oxidation is high during the suckling period and decreases at the time of weaning. Acetyl-CoA, an intermediate product of ketone oxidation is oxidized by intestinal mitochondria of suckling rat pups at 1/8 the rate of postwean indicating that tricarboxylic acid (TCA) cycle activity may be rate-limiting in ßHß oxidation to CO_2 during the suckling period. A high mitochondrial (mito) [NADH]/[NAD] inhibits isocitrate dehydrogenase, a rate limiting step of the TCA cycle. The mito [NADH]/[NAD] decreases four fold at the time of weaning which is consistent with an increase in TCA cycle activity at this time. Since glutamine oxidation enters the TCA cycle in the form of α-ketoglutarate, the inhibition of isocitrate dehyrogenase by an increase in mito [NADH]/[NAD] would not affect glutamine oxidation. These data suggest that an increase in mito [NADH]/[NAD] results in inhibition of oxidation of substrates which enter the TCA cycle at the level of acetyl-CoA and results in the utilization of glutamine as a primary substrate during the suckling period.

Age day	MITO [NADH]/[NAD]	ßHß → CO_2	Glutamine → CO_2	AcetylCoA → CO_2
		(n mol CO_2. mg/hr ± S.E.M)		
15-20	4.8±0.7	0.092±0.012	1.45±0.150	9.0±1.8
23-ad	1.0±0.2	0.368±0.017	1.13±0.099	69.5±5.1

1199 SEQUENTIAL INTRAHEPATIC METABOLIC EFFECTS OF ENTERIC GALACTOSE ALIMENTATION IN NEWBORN RATS. S.Morton, R.M.Kliegman, Case West.Res.U.,Dept.Peds., Cleve,OH

Galactose(GAL) may regulate neonatal carbohydrate utilization. To define the intrahepatic(HEP) regulation during enteric alimentation, newborn rats were fed 0.6 g/kg of glucose(GLU) or GAL. Blood GLU and HEP tissues were sampled at 0, 15 and 30 min and hourly until 300 min (N=6 pups/time). Blood GLU increased in both groups but was lower after GAL at 15-120 min. Insulin increased in both groups but was lower at 30-60 min in GAL fed pups. HEP glycogen (GLYC) increased in GAL fed pups at 30 min and peaked between 120-300 min. In GLU fed pups, GLYC increased at 120 min and achieved a higher peak value at 300 min (86.8± 8.2 vs 55.8±6.0 µmol/g, p=0.01) compared to GAL fed pups. HEP GAL-1-P increased in both groups but persisted in the GAL fed pups. Fructose-6-P was lower at 120 and 180 min in GAL fed pups while fructose Di-P was not altered. Of note, ATP was lower at 30 min in GAL fed pups (1.58±0.06 vs 1.86±0.11 µmol/g, p=0.02). The active component of GLYC synthase increased at 60 min in GAL fed pups and achieved higher values (at 240 min) than the GLU fed pups (46.8±8.2 vs 27.5±3.1 ng/mg protein min, p=0.01). The percent state of activation of synthase was greatest in GAL fed pups while there was no effect on the percentage of active phosphorylase. In conclusion, in newborn rats enteric GAL alimentation resulted in an attenuated glycemic response and, earlier onset of GLYC synthesis due in part to activation of GLYC synthase. Nevertheless, GAL alimentation also resulted in a temporary decline of HEP ATP and a more persistent increase of HEP GAL-1-P. Values mean±SE