65 GROWTH FALTERING DURING EARLY INFANCY AND LACTATION INADEQUACY

NF Butte*, WW Wong, S Villalpando-Hernandez, S Flores-Huerta, C Garza. USDA/ARS Children's Nutr Res Ctr, Dept Pediatr, Baylor Coll Med, Houston, TX, and Div de Crecimiento y Desarrollo, Inst Mexicano del Seguro Social, Mexico DF.

To determine whether growth faltering during early infancy was attributable to inadequate lactation, we measured milk production and composition in 30 rural Indian women living in Capulhaua, Mexico. Milk production and maternal total body water (TBW) were measured by the dose-to-the-mother ^{2}H dilution method at 4 and 6 mo postpartum. Energy, protein, lactose, and fat in human milk were analyzed by standard techniques. Maternal and infant anthropometric measurements were taken serially. Mean (SD) maternal weight, height, and body mass index (BMI) were 50.3(6.0) kg, 147(6.0) cm, and 23.4(3.1), respectively. Infant weight gain averaged 16(3) g/d at 4 mo and 8(3) g/d at 6 mo. Milk production rates were 825(126) g/d at 4 mo and 838(94) g/d at 6 mo. Energy, protein N, lactose, and fat levels in human milk were 0.55(0.07) kcal/g, 1.24(0.16) mg/g, 66.6(2.9) mg/g, and 2.2(0.6) mg/g, respectively. Maternal weight was a significant determinant of infant weight gain ($r=.44$, $p<0.01$). Maternal weight, BMI, and TBW were significantly correlated with milk concentrations of energy and fat ($p<0.01$). Milk production rates tended to be inversely correlated with maternal weight, BMI, and milk concentrations of energy and fat. These relatively high milk production rates only partially compensated for the low concentrations of milk fat. Growth faltering in this population may be partially attributed to lactation inadequacy.

66 ENERGY REQUIREMENTS (ER) DURING INFANCY.

WW Wong*, NF Butte, S Villalpando-Hernandez, S Flores-Huerta. USDA/ARS Children's Nutr Res Ctr, Dept Pediatr, Baylor Coll Med, Houston, TX and Div de Crecimiento y Desarrollo, Inst Mexicano del Seguro Social, Mexico DF.

To evaluate the ER of infants, 20 formula-fed and 20 breast-fed infants (10 ea at 1 mo and 10 ea at 4 mo) from the USA and 8 4-mo-old breast-fed infants from Mexico were studied. Energy expenditures (EE) were estimated by the $^{2}H_{2}{}^{18}O$ method. After collection of a predose urine sample, each infant received by mouth 200 mg $^{2}H_{2}O$ and 300 mg ^{18}O/kg body wt. One daily postdose urine sample was collected from each infant for 7-10 d. ^{2}H and ^{18}O content of urine samples were measured by isotope-ratio mass spectrometry. CO_2 expiration rates (rCO_2) were calculated from the fractional turnover rates and isotope dilution spaces of ^{2}H and ^{18}O. Oxygen consumption rates (rO_2) were calculated from rCO_2 using the measured respiratory quotient or the estimated food quotient values. EE was calculated from the rCO_2 and rO_2 using Weir's equation. Energy deposited for growth (EDG) was calculated from fat and protein gained by these infants using Fomon's reference data and gross energy equivalents of 9.25 kcal/g for fat and 5.65 kcal/g for protein. Energy requirement (ER) was the sum of EE and EDG. ER of the 1-mo-old infants were 103±17 kcal/kg/d. At 4 mo of age, no difference in ER was observed between the American and Mexican infants. However, ER of the 4-mo-old infants (79±15 kcal/kg/d) were significantly lower than the current recommendation for energy intake (105-115 kcal/kg/d) during infancy. Therefore, revision of the current recommendation is warranted.

67 EFFECT OF CARBOHYDRATE(CHO) CONCENTRATION IN A "SEMI-ELEMENTAL" INFANT FORMULA ON TOLERANCE AND MACRONUTRIENT ABSORPTION IN INFANTS WITH CHRONIC DIARRHEA.

CH Lifschitz, FR Carrazza*, A Cordano+. USDA/ARS Childr Nutr Res Cent, Dept Pediatr, Baylor Coll Med, Houston, TX, *Inst da Crianca, Sao Paulo, Brazil, +Mead Johnson, Evansville, IN.

To determine the effect of CHO concentration in infant formula on tolerance and macronutrient absorption, we studied 12 lactose-intolerant males (age x±SD 5.7±2 mo) with chronic diarrhea and malnutrition. In 2 successive metabolic balances infants received a semi-elemental formula that contained high CHO (HCHO) (9.1 g/dl) or low CHO (LCHO) (6.7 g/Dl). Formulas were isocaloric; the difference in CHO content was counterbalanced by the amount of fat. Breath was collected for H2. Fecal fat, osmolality, N, and energy were determined and energy-derived from fecal CHO was calculated. Mean daily caloric intake was not significantly different between the 2 periods (x±SD was 114±26 vs 117±28 kcal/kg/d; HCHO vs LCHO, respectively). No significant difference was seen between the 2 periods in: wt gain/g body wt during the 3 d of each balance, peak H2 levels, fecal N, and osmolality. LCHO formula was tolerated better than HCHO as shown by a significantly lower stool output (x±SD 387±230 g vs 764±443, respectively, [$p<0.05$]), higher fecal pH ($p<0.05$), and lower amount of fecal CHO determined at bedside ($p<0.05$). Macronutrient absorption was also better during the ingestion of LCHO formula as shown by significantly lower amounts of total fecal energy, lower CHO excretion ($p<0.05$), and a better coefficient of fat absorption ($p<0.001$). CHO content had an overriding effect on nutrient absorption and formula tolerance in infants with severe chronic diarrhea.

68 SLEEP STAGE AND ENERGY EXPENDITURE DIFFERENCES IN BREAST-FED AND FORMULA-FED INFANTS.

CL Jensen, NF Butte, JK Moon, DG Glaze, JD Frost, Jr. USDA/ARS Children's Nutr Res Ctr, Dept Pediatr, and Sect Neurophysiol, Dept Neurol, Baylor Coll Med, and The Methodist Hospital, Houston, TX 77030.

To investigate physiologic processes that contribute to observed variations in sleeping metabolic rates, sleep stage and energy expenditure were monitored simultaneously in 9 breast-fed and 6 formula-fed 4-mo-old infants. Electroencephalogram, electrooculogram, arm and leg movement by triaxial accelerometry, heart rate, and O_2 saturation were monitored during an overnight sleep session. Behavioral observations were recorded by video tape and technologist. Sleep stages (nonrapid eye movement [NREM] and rapid eye movement [REM]) were scored by standardized criteria. Energy expenditure during sleep was measured by indirect calorimetry. Mean (SD) energy expenditures were 0.036 (0.004) and 0.041 (0.004) kcal/kg/min during NREM and REM, respectively ($p<0.001$). Mean sleep time, 442 (78) min, accounted for 74 (14)% of the monitoring time. No significant differences were noted between breast-fed and formula-fed infants in the percentage of time spent in sleep during monitoring. However, the mean percentage of sleeping time spent in NREM was 65.3 (4.4) (range 59-70) for breast-fed infants compared with 54.7 (5.0) (range 48-60) for formula-fed infants ($p<0.01$). Significant differences in the distribution between NREM and REM partially explain differences in sleeping metabolic rates of breast-fed and formula-fed infants.

69 BRUSH BORDER LACTASE EXPRESSION AND ACTIVITY DURING LACTATION.

BL Nichols, MA Dudley, M Putman, P Johnston, S Perkinson, PJ Reeds. USDA/ARS Children's Nutr Res Ctr, Dept Pediatr, Baylor Coll Med, Houston, TX

Although lactase activity in adult rats is believed to be low, we have found it to be elevated during lactation. Our study relates lactase activity measured enzymatically and histochemically with immunological and morphological changes in the jejunum of lactating, pregnant, and nongravid adult rats, suckling and weanling pups. Jejunal sections were collected for light and fluorescent microscopy. Villus measurements were made on H&E sections using Jandel Video Analysis. Lactase was demonstrated by histochemistry and immunofluorescence (IF). Fluorescent intensity was graded 0-4+. Lactase specific activity (SA) (μmol/min/g protein) was performed using scraped, homogenized mucosal membranes.

	Age(d)	n	Lactase SA	Villus Length(μm)	IF Intensity
Suckling	10	14	124±5*	163± 7	4.0+
Weanling	22	9	53±4	177± 6	3.9+
Nongravid	--	4	33±4	244±21	1.3+
Pregnant	--	4	34±5	263± 6	2.9+
Lactation	10	8	42±3	384±13**	3.3+
Lactation	22	7	35±6	323±22	2.1+

Mean±SEM *P<0.05 vs all other groups **P<0.05 vs nongravid

Histochemically, lactase was present in all animals. Our results show that during 1) weaning, lactase activity decreases independently of changes in villous morphology, 2) early lactation, both villous length (+57%) and lactase SA (+30%) increase, and 3) late lactation, lactase SA decreases to adult values independent of villus length.

70 DIAGNOSIS OF WILSON DISEASE USING DNA MARKERS

R.H.J. Houwen*, G.D. Billingsley, E.A. Roberts and D.W. Cox. Depts. of Genetics and Paediatrics, The Hospital for Sick Children, Toronto, Canada and *Wilhelmina Childrens Hosp., Utrecht, The Netherlands

Wilson disease (WD) is an autosomal recessive disorder of copper metabolism. The basic defect in WD is unknown, but the gene has been assigned to chromosome 13 at q14-q21 by using closely linked markers.

Clinical presentation of WD is variable. Symptomatic patients have a high urinary copper output and decreased serum ceruloplasmin. Atypical or presymptomatic patients may not be diagnosed by these criteria. Distinction from heterozygotes can also be difficult. A liver biopsy is almost always conclusive, but is invasive. Therefore the incorporation of 67Cu into ceruloplasmin (absent in WD) has been advocated as a non-invasive diagnostic test.

Nevertheless a genetic diagnosis is more desirable. So we used 12 DNA markers from the 13q14-q21 region to study 6 patients with a questionable diagnosis of WD. We could confirm the diagnosis in 2 of them. However in the remaining 4 individuals, all previously classified as having WD (based on a moderately increased urinary copper excretion, a somewhat increased liver copper, a low ceruloplasmin and no incorporation of 67Cu), the diagnosis had to be revised. Our present results reveal that they are in fact heterozygotes.

This demonstrates that DNA markers can now be used to establish the diagnosis of WD. Moreover copper isotope studies can be abnormal in heterozygotes with a low ceruloplasmin.