† 690 DISTRIBUTION OF EPIDERMAL GROWTH FACTOR (EGF) RECEPTORS(R) IN THE FETAL(F) GASTROINTESTINAL TRACT (GI) F.Sadiq, J.Gunn, V.Chechani and U.Devaskar (Spons. W.J.Keenan) Dept. of Peds., St. Louis University, St. Louis, Missouri

EGF stimulates mitosis and enzymatic function of GI. Regional differences of EGF R in F GI might clarify the role that EGF plays in GI maturation. Plasma membrane bound EGF R of the 27d. F rabbit stomach(S), jejunum(J), ileum(I) and colon(C) were isolated and characterized by protein con., % recovery, DNA and 5'-nucleotidase. Optimal binding of ^{125}I-EGF was obtained at 37° C, and pH 7.45 after 45 min. of incubation. All Scatchard plots were linear. (All data X̄±SEM, DNS-displacement not seen)

	Stomach (n=6)	Jejunum (n=6)	Ileum (n=6)	Colon (n=6)
Protein (mg/gm)	42±1.6	52.5±2.6	50.5±1.3	46±.2
% recovery	6.2±.37	11.69±.84	9.12±.8	7.8±.7
% Sp. Binding .2mg of prot.	1.18±.23	1.04±.12	.622±.058	*2.95±.344
R no.x10^{-10} / mg Protein	DNS	3.48±.78	2,7±.81	*8.65±2.15
R No.x10^{-10} / mg DNA	DNS	7.8±.23	4.27±.79	*16.9±1.5
Kd	DNS	4.2±.9	4.0±.56	3.33±.72

*p < 0.05

At 27d. there was very little EGF binding in the stomach while the highest density of EGF R was observed in the colon. EGF probably plays an important differential role in maturation of the fetal GI.

● 691 STORAGE OF MEDIUM CHAIN TRIGLYCERIDES (MCT) IN ADIPOSE TISSUE OF FORMULA FED INFANTS. Pierre Sarda, Guy Lepage, Philippe Chessex. (Spons. by Claude C Roy). Univ. of Montreal, Hôpital Ste-Justine, Dept. of Ped., Montreal.

The rapid portal transport and hepatic oxidation of MCT point to their utilization as a readily available source of energy. This is supported by observations showing decreased fat storage in MCT fed rats. However the composition of adipose tissue (ADIP) has not been studied. The fatty acid (FA) pattern of ADIP from infants operated on for inguinal hernia was determined by gas chromatography using the internal standard, C9:0, to correct for losses of medium chain fatty acids (MCFA: C8:0, C10:0, C12:0) during the preparative phase. The pattern obtained was correlated with that of human milk (HM) or 67 kcal/dl formulas (F1 or F2) fed ad libitum from birth to 3 groups of infants (A,B,C). Group A consisted of 8 infants with a mean age (X̄±SD) of 75±29d and weight of 5.3±1.6 kg. Corresponding figures for group B comprising 7 infants, were 63±32d and 4.9±1.7 kg, while for the 7 subjects of group C they were 50±15d and 4.1±0.9kg. % of total FA:

	A		B		C	
	HM	ADIP	F1	ADIP	F2	ADIP
C 8:0	0	0	4.6	0	7.8	0.7±0.2
C10:0	2.3±0.3	0.5±0.1	3.0	0.6±0.2	4.9	1.2±0.1
C12:0	4.9±0.8	2.6±0.7	11.3	4.5±0.3	21.8	10.9±0.9

These data show in ADIP a surprisingly large % of MCFA, linearly related ($p<0.05$) with the dietary intake of C10:0 and C12:0. Since this relationship did not hold for C8:0, it is suggested that C8:0 may be more completely oxidized. The metabolic fate of MCFA stored in ADIP needs to be explored.

692 PEDIATRIC ULCER DISEASE: NEW PERSPECTIVES. L. Sanipkin and F.C. Rothstein (Spon. by J. Blumer), Case Western Reserve Univ., Cleveland, Ohio.

We retrospectively studied 70 children with endoscopically proven ulcers (age 1day-18yrs) to determine whether gastric ulcers (GUD) were different from duodenal ulcers (DUD) with respect to epidemiology, aspirin (ASA) use and clinical presentation. GUD occurred in 64% and DUD in 36% of patients. GUD occurred in all age groups but DUD was rare in the young child. GUD was present in 51% of children under 6 yrs of age, compared to 8% of DUD. GUD showed a strong seasonal influence with 80% occurring between November and April, the period of increased viral illnesses and presumed increased ASA use. DUD showed no such seasonal predilection. Of the 36 patients with GUD during these months, 67% were acute (symptoms < 1 mo), 36% had a prodromal illness and 47% were exposed to ASA. Of the remaining 9 GUD patients who presented in the summer months, 78% were acute in onset but only 18% had a prior illness or were exposed to ASA. In contrast, of the 25 DUD patients, 52% presented with chronic symptoms, 16% had an antecedent illness and only 28% had ASA. Symptomatically, pediatric DUD presents similarly to adult DUD, with epigastric pain (76%) as the primary symptom and GI bleeding in only 16%. GUD more commonly presents with vomiting (53%) and GI bleeding (49%) with pain in only 44%. In conclusion, it appears that pediatric GUD is a unique problem, distinctively different from DUD. GUD usually occurs in the winter months and commonly follows a viral illness. More importantly, GUD is seen after the use of ASA which may in part account for the high frequency of acute gastrointestinal bleeding found in these children.

●693 'TRUE' RATES OF GLUCOSE OXIDATION IN NEONATES. P.Sauer J.Van Aerde, J.Smith, P.Pencharz, P.Swyer, Dept Paed & Med Eng, U of Toronto, Res Inst, Hosp Sick Children, Toronto, Canada.

We have previously studied substrate utilization in neonates using indirect calorimetry (IC). As IC might overestimate glucose oxidation rate (GOR) due to conversion of glucose to fat, we compared GORs measured by IC and U-^{13}C-glucose utilization. 7 AGA neonates fed only glucose-amino acids IV were studied. Glucose intake was 15.1±3.0g/kg/d (mean SD), protein intake 2.6±1.3g/kg/d BW 2.4±0.4kg, gestational age 37±2 wks, study weight 2.4±0.4kg, age 4-30d. IC was performed for 6h metabolic rate (MR) and GORs were calculated from protein free $\dot{V}O_2$ and RQ. Simultaneously a primed constant infusion of U-^{13}C-glucose was given. GOR was calculated from the increase in $^{13}CO_2$ excretion above baseline. A plateau was obtained after ≈2h.

RESULTS: (n=7, mean±SE)

	MR kcal/kg/d	GOR g/kg/d	% of MR derived from glucose	fat	protein
IC	45.5±1.6	10.3±0.9*	85	6	9
^{13}C		7.0±0.4*	58	33	9

* paired difference 3.3±0.7 g/kg/d, p<0.001

CONCLUSIONS: 1) IC shows a significantly higher glucose oxidation rate than ^{13}C-glucose; 2) glucose oxidation rate of neonates is lower and endogenous fat oxidation higher than previously measured; 3) a significant amount of ingested glucose presumably is converted into fat.

694 PLASMA AMINO ACIDS IN PRETERM INFANTS FED FORTIFIED MOTHER'S MILK. Richard J. Schanler, Cutberto Garza, and Buford L. Nichols. Baylor College of Medicine, USDA/ARS Children's Nutrition Research Center, Department of Pediatrics, Houston, TX.

Eleven infants [BW=1230(97)g; GA=29(0.7)wk; mean (SD)] were fed their mother's milk fortified with skim and cream components derived from heated, lyophilized donor milk for approximately 8 weeks. At 6 wk of age, in conjunction with metabolic balance studies, plasma amino acids levels were measured at 0, 1, and 2 hr after a nasogastric bolus feeding. Mean nitrogen and energy intakes were 480(45) mg/kg/d and 134(6) kcal/kg/d, respectively. Postprandial levels of THR, GLU, ALA, VAL, ILE, LEU, TYR, PHE, LYS, ARG, and MET rose significantly above baseline ($P < 0.05$). Of these, the greatest increments at 1 hr above baseline were observed for ILE, VAL, LEU, and MET. No amino acids dropped below baseline. The smallest increments were observed for GLY, GLU, TRY, and HPR. When corrected for present estimates of requirements for "essential" amino acids, the apparent order of limiting amino acids was LEU, ILE, PHE + TYR, LYS, THR, VAL, MET + CYS, TRY, and HIS. BUN levels and acid-base status were normal. Albumin and total protein levels were 3.1 (0.6) and 4.4 (0.6), respectively. Weight gain and N retention matched fetal growth and body composition data. These data support the conclusion that to double N concentrations in maternal milk does not overwhelm the abilities of preterm infants to catabolize amino acids. The small increments in amino acids traditionally labeled as "non-essential" suggest that the level of intake or the capacity for their synthesis may be limited.

695 GASTROESOPHAGEAL REFLUX(GER) IN CYSTIC FIBROSIS (CF). R. Brent Scott, Edward V. O'Loughlin, D. Grant Gall, University of Calgary and Alberta Children's Hospital Department of Pediatrics, Calgary, Alberta, Canada.

GER is a recognized problem in CF. The aim of this study was to define the frequency of GER in a clinic population of CF patients and to assess the effect of supplemental nocturnal nasogastric (NG) feedings on the frequency of GER. Children in the CF Clinic were surveyed to determine the incidence of symptomatic GER. A subgroup of patients with poor nutritional status were studied with esophageal manometry and 24-hr esophageal pH recording before and after initiation of supplemental continuous night-time NG feeds. Of 68 CF patients ≥5 yr of age (13.1±7.1 yr, X̄±SD) 20.6% experienced regurgitation and 26.5% heartburn. There was no significant association between symptoms of GER and bronchodilator therapy. Eight patients studied with manometry and 24-hr pH recording (15.5±6.6 yr) had normal lower esophageal sphincter pressure of 24.8±8.8 mmHg and thoracoabdominal pressure gradient of 11.4±4.6 mmHg. Peristalsis and upper esophageal sphincter function were normal. There was a significant increase in reflux episodes, episodes >5 min duration, and % time esophageal pH was <4 in CF patients compared to published control data for the entire 24-hr period or during sleep. During sleep continuous NG feedings significantly increased episodes of reflux. Our findings indicate that symptoms of GER, heartburn and regurgitation, are frequent in CF patients and when quantitated by 24-hr esophageal pH monitoring GER is significantly more common in CF compared to controls. GER was not related to bronchodilator therapy but was aggravated by night-time NG feedings.