594 EVALUATION OF ERYTHROCYTE RIBOFLAVIN IN LOW BIRTHWEIGHT INFANTS: RELATIONSHIP TO MODE OF NUTRITION. Martin A. Docherty, Kathleen B. Schwarz (spon. by Thomas Aceto, Jr.). St. Louis University School of Medicine, Cardinal Glennon Memorial Hospital for Children, Depts. of Peds. and Dietetics, St. Louis, MO.

Riboflavin requirements for total parenteral nutrition (TPN) regimens have not been adequately determined. We investigated the riboflavin status of low-birthweight infants maintained by TPN formulation which provided 4 mg/day of riboflavin to each infant compared to that of infants fed orally on formula containing 1.0 to 5.0 mg riboflavin/liter.

15 newborn infants (birthweight 570-2350 grams) had baseline blood samples drawn prior to TPN or formula feeding. The patients had second blood samples drawn after they had been fed either orally (n=7) or by TPN (n=8) for at least 7 days.

	Riboflavin status		
Feeding Regimen	Erythrocyte Glutathione Reductase(AC)*		t-test
	Baseline	After	
TPN	0.98 ± 0.12	1.00 ± 0.09	N.S.
ORAL	1.00 ± 0.11	1.06 ± 0.11	N.S.

*AC=Activity Coefficients

None of the 15 infants showed abnormal riboflavin status and the range established for these low-birthweight infants (AC 0.86-1.19) corresponds to that established for healthy full-term newborns(0.9-1.2). (Proc. Soc. Exp. Biol. Med 143:326, 1973).

From this pilot study, it appears that 4 mg/day of riboflavin provided to low-birthweight infants in a TPN solution maintains riboflavin status during the time period studied (7 days).

595 **ENDOGENOUS ETHANOL PRODUCTION IN A CHILD WITH SHORT BOWEL SYNDROME.** Jeremy M. Drelich, Amy Fox, Elinor E. Reese, Brad D. Berman (Spon. by Samuel P. Gotoff). Michael Reese Hospital and Medical Center, Department of Pediatrics, Chicago.

Although ethanol occurs naturally as a product of fermentation, there are few reports of significant production in humans. We report a child with ethanol intoxication, apparently caused by alcoholic fermentation within his gastrointestinal tract.

A 3 8/12-year-old male with short bowel syndrome on continuous nasogastric (CNG) 3/4-strength Vivonex was admitted for dehydration from gastroenteritis. Otitis media was diagnosed, and the patient was placed on ampicillin. While on nasogastric ampicillin and CNG feedings, the patient developed symptoms of acute ethanol intoxication. Ampicillin was discontinued. Over the following 10 days, blood ethanol rose to 452 mg% before feedings were discontinued. Gastric ethanol (max. 185%) was repeatedly elevated, although less than blood ethanol. Stool ethanol (max. 575 mg%) was greater than blood and gastric ethanol. An extensive search for exogenous ethanol sources was unsuccessful. Stool cultures grew Klebsiella pneumoniae and had a predominance of the yeasts Saccaromyces cerevisiae and Torulopsis glabrata. In vitro studies confirmed the ability of the three organisms to ferment the patient's formula into ethanol. Subsequent feeding did not result in elevations in blood ethanol when stools no longer had yeast overgrowth.

Abnormalities of gut anatomy and motility are known to predispose humans to colonization by atypical flora. Fermentation by unusual gut flora can produce near-lethal blood ethanol levels in patients with short bowel syndrome being fed conventional formula.

596 NUTRITIONAL ADEQUACY OF FORTIFIED PRETERM HUMAN MILK (PTHM) IN VLBW INFANTS. Richard A. Ehrenkranz, Mildred A. Chamberlin, Patricia A. Gettner, and Catherine M. Nelli (Spon. by I. Gross). Dept of Pediatrics and Children's Clinical Research Center, Yale Univ Sch of Med, New Haven, CT.

The ability of mother's own PTHM fortified with a protein-mineral supplement (Mead Johnson) to approximate in utero requirements of fat, nitrogen (N_2), calcium (Ca), phosphorus (P), and zinc (Zn) was assessed and compared to a premature formula (Mead Johnson) in 5 VLBW infants (BW 948±67 gm, GA 27.8±0.9 wks, mean ± SEM). Urine and stool were collected separately during a 72 hr interval bracketed by carmine red. Three studies were performed in 3 infants (wt 1230-1540 gm, age 41-74 days) fed fortified-PTHM and 3 in 2 infants (wt 1120-1490 gm, age 27-40 days) fed formula. Fat balance and absorption were 6.31±0.72 gm/kg/d and 93±5% with fortified-PTHM, and 5.63±0.06 gm/kg/d and 93±2% with formula respectively. N_2, Ca, and P balance (mg/kg/d) and % retention are shown below. Zn balance and retention ranged from 256 to 1084 µg/kg/d and 14 to 64% with fortified-PTHM, and -1074 to +258 µg/kg/d and -88 to +22% with formula respectively. In conclusion, the nutritional requirements for fat, N_2, Ca, and P are comparably met by fortified-PTHM and premature formula. However, fortified-PTHM meets Zn needs better.

	Fortified-PTHM		Premature Formula	
	Balance	Retention	Balance	Retention
Nitrogen	427±35	87±3.0	478±30	85±4.9
Calcium	79±6.0	67±6.3	85±7.5	61±6.5
Phosphorus	46±3.0	81±5.2	56±0.8	79±2.6

597 CALCIUM (Ca), PHOSPHORUS (P), ZINC (Zn), AND COPPER (Cu) CONTENT OF PRETERM HUMAN MILK (PTHM). Richard A. Ehrenkranz, Barbara A. Ackerman and Catherine M. Nelli (Spon. by I. Gross). Dept of Ped, Yale Univ Sch of Med, Yale-New Haven Hospital, New Haven, CT.

The Ca, P, Zn, and Cu content of PTHM was determined on cross-sectional and longitudinal samples of PTHM collected on days 1-2, 4±1, 7±1, 14±2, 28±2, 42±2, 56±2, and 70±2 of lactation from 74 women who delivered at or before 34 wks of gestation. Ca, Zn, and Cu were analyzed by atomic absorption spectroscopy. P was analyzed by the spectrophotometric method of Bartlett. The findings are displayed in the Table. Changes in content over time were tested by analysis of variance. Zn content decreased significantly with duration of lactation. The content of Ca, P, and Cu increased slightly during the early days of lactation, but then decreased slightly with the duration of lactation. Comparing these data with the estimated third trimester intrauterine accretion rates for these 4 minerals suggests that although the Zn content of PTHM might meet those rates, the content of Ca, P, and Cu does not.

Day (n)	Ca (mg/dl)	P (mg/dl)	Zn (µg/dl)	Cu (µg/dl)
1-2 (37)	30.0±1.4	12.2±0.7	909±73	28.1±2.0
4±1 (31)	33.6±1.6	15.4±0.8	715±44	42.2±3.5
7±1 (22)	27.6±1.9	18.6±1.9	592±44	38.0±3.6
14±2 (31)	24.1±1.2	14.6±1.0	467±30	34.6±1.8
28±2 (23)	24.2±1.0	13.7±0.7	370±25	30.6±1.8
42±2 (10)	27.6±1.3	14.5±0.7	329±31	30.1±2.8
56±2 (10)	25.7±0.9	12.9±0.8	282±36	24.0±2.4
70±2 (6)	23.7±0.8	10.0±0.6	260±29	---

† **598** MEASURING THE DIETARY BIOAVAILABILITY OF CALCIUM (Ca) IN PREMATURE INFANTS WITH THE STABLE ISOTOPE ^{46}Ca. Richard A. Ehrenkranz, Barbara A. Ackerman, Catherine M. Nelli, and Morteza Janghorbani (Spon. by I. Gross). Yale Univ Sch of Med, Yale-New Haven Hosp, Dept of Ped, New Haven, CT and Nuclear Reactor Lab, Dept of Nutr and Food Sci, Massachusetts Institute of Technology, Cambridge, MA.

The dietary bioavailability of Ca was determined on 12 occasions in 10 AGA premature infants (GA 29.9±0.3 wks, BW 1198±28 gm mean ± SEM) with stable isotope techniques. A tracer dose of ^{46}Ca that provided a 5 to 7-fold enrichment of dietary ^{46}Ca was given during one scheduled intermittent gavage feeding for each determination. Five studies were performed in 4 infants (wt 1100-1500 gm, age 15-37 days) fed a whey-predominant premature formula; 3 in 2 infants (wt 1455-1560 gm, age 26-30 days) fed their own mother's preterm milk (PTHM); and 4 in 4 infants (wt 1500-1690 gm, age 20-25 days) fed fortified-PTHM, a 1:1 (V/V) mixture of the premature formula and their own mother's milk. A 72 hr stool collection, bracketed by carmine red, was initiated with the administration of ^{46}Ca. The stool contents of ^{46}Ca and ^{48}Ca were determined with neutron activation analysis on each stool from 3 studies and after pooling in 9. Pooling was found to slightly underestimate ^{46}Ca absorption because of reentry of about 0.5% of the absorbed ^{46}Ca into the GI tract/24 hr. The % ^{46}Ca absorption was 80±4% (range 65-87%) from formula, 89±4% (85-97%) from PTHM, and 81±6% (range 69-96%) from fortified-PTHM. Thus, assuming that preterm infants handle ^{46}Ca like dietary Ca, about 80% of dietary Ca is bioavailable. This value is comparable to data from standard Ca balances.

† **599** THE CRYING BABY SYNDROME: THE ROLE OF GASTRO-ESOPHAGEAL REFLUX. Alex F. Flores, Aubrey J. Katz, (Spon. by Harvey Colten), Dept. of Ped., Harvard School of Medicine, Children's Hospital Med. Center, Boston.

Crying babies present many diagnostic dilemmas. We studied 10 infants, ages 5-24 months (mean 10.8 mo.) who presented with persistent nocturnal crying;some also with postprandial irritability. None had vomiting or weight loss.Mean duration of symptoms was 5.5 mos. Criteria for a GE reflux workup included severe disruption of family life and failure to respond to a milk and soy free diet. Methods: Infants were evaluated by GI series, overnight intraesophageal pH monitoring, and endoscopy. Results:Upper GI series revealed no anatomical abnormalities and significant GE reflux in 3 patients. Overnight pH probe study showed abnormal acid clearance in all patients, correlating with crying and irritability. Endoscopy and biopsy revealed esophagitis in one patient. Follow-up: All patients responded to antacid therapy. 6-24 months after therapy indicates no evidence for recurrence of GER, or chronic esophagitis. Conclusions:1. GER is an important and not uncommon cause of the nocturnal and/or postprandial irritability in infancy.2. Intraesophageal pH probe monitoring appears to be the best method for detecting GER in these patients and correlates well with symptoms. 2. Only 1/10 (10%) of patients had esophagitis on biopsy. 4. None have progressed to chronic esophagitis.