626 DECREASED GASTRIN SECRETION EVOKED BY FEEDING DURING ADMINISTRATION OF HUMAN GROWTH HORMONE (hGH). A.Root, A.Auritt and C.Livingston. Univ.South Florida,Dept. Pediatrics, and All Children's Hospital, St.Petersburg, Florida.

GH stimulates gastric mucosal growth and function (Crean, Vitamins Hormones 21:215,1963). In order to determine if hGH affected gastrin secretion, serum concentrations of this peptide were determined by radioimmunoassay for three hours during a standard test meal (2 boiled eggs, 2 pieces buttered toast, 8 ounces skim milk) in patients with hyposomatotropism prior to initiation of treatment (N=14) and after six (N=12) and 12 months (N=10) of hGH administration (0.1 U/kg thrice weekly). Basal and peak concentrations (pg/ml) of gastrin were:

	Pre hGH		+ 6 months		+ 12 months	
	Basal	Peak	Basal	Peak	Basal	Peak
$\bar{X}$	85	121[a]	88	114[a]	79	108[b]
SE	±6	±10	±10	±10	±9	±13

(a. p <0.01, b. p <0.05 versus basal by paired t test)
Gastrin concentrations increased during feeding at each period. The peak gastrin concentration after 12 months of hGH administration was significantly (p <0.05) lower than the peak value recorded prior to initiation of treatment. Peak serum insulin concentrations in response to the standard meal did not change significantly during hGH administration [pre Rx- 28 ±6 uU/ml (N=13); +6 months 39 ±12 (N=7); +12 months 31± 6 (N=9)]. It is concluded that long term administration hGH is associated with slightly depressed gastrin secretory response to feeding.

627 MALABSORPTION, FAT-FILLED ENTEROCYTES, HYPOCHOLESTEROLEMIA WITH NORMAL SERUM APOPROTEIN-B: A NEW SYNDROME. Claude C. Roy, Richard Deckelbaum, Allan Sniderman, Jean-P. Buts, Pierre Brochu, Jacqueline Orquin, Peter Green, Andrée Weber, Claude L. Morin and Jacques Letarte. Univ. of Montreal, Hôpital Ste-Justine, McGill Univ., Royal Victoria Hospital, Hadassah Hospital, Jerusalem, Columbia Univ. New York.

A celiac-like syndrome first became apparent during the first 3 months of life in 8 children who were studied between the ages of 3 1/2 mo to 14 years. A low one hr D-xylose was noted in 5/7, hypoalbuminemia (<3.5g%) in 5/8. Steatorrhea (11.6±3.1g/24h) was present in all patients along with low levels of carotene as well as of vitamins A and E. Jejunal biopsy changes were indistinguishable from those seen in abetalipoproteinemia. Psychomotor retardation (1/8), absent reflexes (2/8), abnormal electrooculograms (4/8) and electroretinograms (1/8) were noted. All patients had hypocholesterolemia (70.1±5.8mg/dl). There was no response of cholesterol nor of triglycerides (70.1±13.5mg/dl) to a fat load (50g/1.73m^2) over a 5hr period. Total (8.2±1.5) and residual (6.2 ±0.9) lipoprotein lipase activities (μmoles fatty acid/ml/hr) were essentially normal. Serum apoprotein-B was measured by a radial immunodiffusion method which could not detect any apo-B in a case of abetalipoproteinemia. Values ($\bar{X}$±SD) in the 8 patients (63.0±9.3mg/dl) were comparable to those obtained in a young adult population (80±10mg/dl). This study describes a new entity which is similar to abetalipoproteinemia with regard to its gastrointestinal manifestations. However CNS and ophtalmic findings are milder and less frequent.

● **628** INCREASED SECRETION RATE OF LECITHIN IN EXPERIMENTAL PANCREATIC INSUFFICIENCY. Claude C. Roy, Lyse-A. Fournier, Liette Chartrand and Guy Lepage. University of Montreal, Hôpital Ste-Justine, Dept. of Pediatrics, Montreal.

Since unhydrolyzed lecithin is known to interfere with cholesterol, fatty acid and bile salt absorption, it was of interest to examine the effect of pancreatic insufficiency on its biliary secretion rate. A pancreatic fistula (PF) was created in 2 groups of 10 male Sprague Dawley rats and in an equal number of controls (C). Following a 4 day intragastric perfusion of diet A (.4g of fat/24h) or diet B (3.4g of fat/24h) a bile fistula was created to study biliary lipid secretion rates (μmoles/100g/24h) and bile salt kinetics over a 6h period. There was little difference between PF and C on diet A. However on diet B, the PF group showed an increase (P<.01) in lecithin (79.5±6.8 vs 57.1±4.7) and little change in cholesterol (8.1±1.2 vs 9.6±1.5) and in bile salt (381.6 ±52.9 vs 349.2±32.5) when compared to C. Bile salt concentration and qualitative pattern as well as pool size and circulation frequency remained the same in PF and in C on diet A and on diet B except for the basal synthesis of bile salts which was increased (P<.005) in PF on diet B. In 2 PF animals on diet B, infusion of pancreatic juice led to normalization of lecithin secretion and of bile salt synthesis. These data suggest that in rats fed a low fat diet pancreatic insufficiency has no effect on biliary lipid and on bile salt metabolism. In conjunction with a high fat diet, it leads to an increase in the amount of lecithin circulating daily in the G.I. tract and to a significant interruption of the enterohepatic circulation of bile salts.

629 MECHANISM OF HYPOCHLOREMIC METABOLIC ALKALOSIS(HMA) IN DOGS FED A CHLORIDE-DEFICIENT FORMULA(CDF). Shane Roy, III, Fielding B. Stapleton and Billy S. Arant, Jr. Dept. Ped., Univ. of Tennessee Ctr. for Health Sci., Memphis, TN.

HMA, failure to thrive(FTT), deceleration of head growth and a Bartter-like syndrome was observed in 200 infants in the USA unintentionally fed a CDF([Na^+,K^+,Cl^-] of 17,25,<2mEq/L). HMA was corrected by adding chloride to the CDF. Suggested causes for the metabolic abnormalities include chloride depletion, alkali excess ([citrate$^-$]25mEq/L) and/or increased PGE_2. To better define pathophysiologic mechanisms, metabolic balance studies were performed in 3 mongrel puppies(A,B,C) fed the CDF(150ml/kg/day) for 11 wks. During 3 additional weeks, A was fed CDF, B received CDF+chloride ([Cl^-]14.4mEq/L) and C was fed another soy formula([Cl^-]11mEq/L). Mean laboratory values obtained before CDF(I-A,B,C), after 11 wks of CDF(II-A,B,C) and after 3 final weeks (III) were:

Plasma:	Na^+	K^+	Cl^-	HCO_3^-	pH	PRA[a]	PA[b]	ECFV[c]	$UPGE_2$	UNa^+	UK^+	UCl^-
	mEq/L									mEq/24hr		
I	139	5.1	110	21.2	7.33	1.0	36	342	155	12.3	10.7	5.4
II	134	3.6	86	30.2	7.40	7.9	45	273	182	8.3	8.3	0
III(A)	135	4.1	92	32.5	7.42	8.1	28	180	67	13.1	15.0	0
(B,C)	139	4.8	109	20.8	7.34	0.6	23	321	269	4.8	4.8	2.0

([a]ng/ml/hr);([b]ng/ml);([c]ml/kg);([d]ng/kg/24hr);(U=urinary excretion)
Each puppy exhibited FTT. These findings support the hypothesis that CDF and not chloride loss, alkali administration or increased PGE_2 resulted in hypochloremia, ECFV depletion with increased PRA and PA, increased renal conservation of $NaHCO_3$ and increased urinary potassium excretion.

630 RENAL RESPONSE TO ESSENTIAL AMINO ACID DEPRIVATION: ADAPTATION OF TAURINE (TAU) TRANSPORT. Rima Rozen and Charles R. Scriver. MRC Genetics Group, McGill University, Montreal, Quebec, Canada.

TAU behaves as an essential amino acid in the human neonate. Infants fed diets containing virtually no TAU have low [TAU] in plasma and urine (Gaull et al, J. Ped. 90:348, 1977). We reported (Rozen et al, Ped. Res. 14:624, 1980) that control and hypertaurinuric mouse strains, fed a low-protein diet, decrease fractional TAU excretion *in vivo* and increase TAU uptake by renal cortex slices and purified luminal membranes *in vitro*. We have now investigated the specificity of the renal adaptation to TAU deprivation. Mature C3H and C57Bl/6J mice, fed a selective low-sulfur-amino acid diet for 2 wks., also decrease fractional TAU excretion *in vivo* ($p < 0.01$) and increase uptake of TAU by renal cortex slices ($p < 0.01$) and by brush border membranes ($p < 0.01$). However, uptakes of glucose and alanine are also increased in the brush-border membrane vesicles of *in vivo* adapted mice. We then examined the hypothesis that the renal adaptation reflects deprivation of an essential amino acid (methionine). Mature mice, deprived of phenylalanine for 2 wks., also decrease urinary TAU excretion ($p < 0.02$) and increase uptake of TAU, glucose and alanine by brush-border membranes ($p < 0.05$). It appears that essential amino acid deprivation is associated with adaptation of the renal transporters for TAU and other solutes. Our findings in the mouse offer an *in vitro* explanation for the finding (Pentz, Biochem. Med. 2:70, 1968) that the human female decreases urinary TAU excretion during lysine deprivation.

631 BREAST FEEDING AMONG PRIVATE AND CLINIC PATIENTS: A RETROSPECTIVE STUDY OF 7,284 NEWBORNS. Timothy Ryan and Arturo Hervada. Thomas Jefferson Univ. Hosp. Phila

We could not find documented in the literature the commonly held belief that breast feeding is more common in upper and middle class than in the lower socioeconomic mothers. We retrospectively studied the incidence of breast feeding in our "private" and "clinic" newborn infants for a period of three years & nine months.

The study comprised 7,284 newborns delivered from October, 1976 to June, 1980. 3,807 (52.2%) were admitted to the "private" service and 3,477 (47.9%) to the "clinic" service. All infants were born at Thomas Jefferson University Hospital.

The mothers were questioned in the delivery room as to the type of nursing they had chosen. Three different answers were obtained: breast, bottle or undecided. The overall incidence of breast feeding was 55% in the private vs. 41% in the service group. Undecided mothers were 3.1% in the first group & 5.9% in the second.

Each successive year of the study showed an increase in the incidence of breast feeding. In the private group the incidence increased from 49.4% in the first year to 60.2% in the last one. There was also a rise in the "clinic" group from 22.7% to 31.7% over the same dates.

Our findings point to the need for better education regarding the importance of breast feeding for all women, especially those from lower socioeconomic backgrounds. For this purpose we recommend that human lactation should be introduced in the curriculum of all high schools and special emphasis should be given to breast feeding in all our maternity clinics.