383 CORTICOSTEROID BINDING GLOBULIN (CBG) IN FETAL AND NEWBORN SHEEP. P.L. Ballard, A.C.G. Platzker, R.D. Bland, J.A. Kitterman, R.I. Clyman, P.D. Gluckman, S.L. Kaplan, and M.M. Grumbach. Dept. Ped., Childrens Hosp., Los Angeles, and Dept. Ped. & Cardio. Res. Inst., Univ. Calif., San Francisco.

To examine the ontogeny and regulation of CBG in the perinatal period, we determined plasma cortisol binding capacity using a charcoal absorption assay. CBG capacity increased progressively from 1.6 µg/dl at 75 days to 7.1 µg/dl at 141 days (n=249), with the greatest increase from 121 days to term. There was a similar increase in CBG (+41.1%) and total proteins (+36.7%) in 6 fetuses during the 4-6 days before spontaneous delivery. After birth, both CBG and proteins decreased during the first half day; thereafter CBG decreased ($t_{½}$= 5 days) to 1.0 µg/dl at 14 days while proteins did not change. In 7 fetuses with loss of pituitary function there was no increase in CBG during the 22-35 days after surgery. Infusions of hydrocortisone for 2 days, estradiol for 5 days, prolactin for 5-8 days and ACTH for 3 days to intact fetuses did not affect CBG levels.

We conclude that the pituitary controls the major increase in CBG after 121 days; there is an additional prepartum increase in all serum proteins with labor. The pituitary hormone(s) which stimulate CBG production are not identified, but this hormonal influence apparently ceases with birth.

384 THE INFLUENCE OF THYROXINE (T_4) CONCENTRATION IN HUMAN MILK (HM) ON NEONATAL THYROID SCREENING (NEO-T_4). Raul C. Banagale (Spon. by A. P. Erenberg) Dept. of Ped., Univ. of Iowa Hospitals and Clinics, Iowa City, IA and Dept. of Ped., Iowa Methodist Medical Center, Des Moines, IA.

In HM T_4 is excreted in significant amounts after the first post-partum week and may delay the clinical recognition of a hypothyroid infant (Acta Paediatr Scand, Supp 277:54, 1979). The same study has shown that the HM T_4 concentration during the first 5 days post-partum to be low (0.7 ± 0.3 ug/dl). However, the T_4 measurements in HM were accomplished on a limited number of artificially expressed samples. Prior to completion of the present study, speculation existed whether breast feeding interfered with the results of our NEO-T_4 (^{125}I T_4 RIA) which is done routinely on the 3rd day of life. The table shows results of a 1 year study comparing the NEO-T_4 values of formula fed (FF) and breast-fed (BF) infant.

	MALES		FEMALES		MEAN	
	ug/dl	n	ug/dl	n	ug/dl	n
FF	15.88	(340)	16.77	(340)	16.32	(680)
BF	16.32	(566)	16.61	(513)	16.46	(1079)
MEAN	16.16	(906)	16.67	(853)	16.41	(1759)

The observed difference ($p < 0.01$) between BF and FF (lower values) males does not have a biological explanation. However, the overall difference between BF and FF infants (0.14 ug/dl) does not approach statistical significance and confirms that HM T_4 concentration does not interfere with the NEO-T_4 results done on the third day of life.

385 ABNORMALITIES IN GROWTH HORMONE SECRETION AFTER CRANIAL IRRADIATION IN PRIMATES. B.B. Bercu, G. Chrousos, T. Brown, D. O'Neill, J. Schwade, and D.G. Poplack, NICHD and NCI, NIH, Bethesda, MD 20205 (SPON: J.D. Schulman)

Abnormalities of GH secretion have previously been reported in children given cranial irradiation for CNS prophylaxis in acute lymphocytic leukemia or for brain tumors. To better define these neuroendocrine abnormalities, prospective longitudinal studies of GH secretion were performed in young adult male rhesus monkeys (N=4 in each group). Two doses of cranial irradiation were given (2400 or 4000 rad in 10 fractions over 2 weeks). Three consecutive provocative tests of GH secretion were used (arginine infusion, insulin induced hypoglycemia and L-Dopa stimulation) prior to radiation and 10, 30 and 50 weeks after radiation. The 2400 rad group at 10 weeks had an excessive and prolonged GH response to arginine (GH at 30, 60 and 90 min was 15±15, 37±11 and 30±11 ng/ml vs 22±4, 15±4 and 6±1 ng/ml for 13 controls, mean ± SE) and insulin (GH at 30 and 60 min was 46±13 and 49±7 ng/ml vs 40±9 and 8±1.5 ng/ml for 9 controls). In subsequent studies, GH responses to arginine and L-Dopa were normal, but GH response to insulin was blunted. In the 4000 rad group the arginine and L-Dopa tests were normal throughout, but the response to insulin was consistently blunted. In conclusion, there are abnormalities of GH secretion after cranial irradiation with doses frequently used in clinical practice, but their clinical significance is not yet clear. This may be a useful model for the study of neuroendocrine regulation of GH secretion.

386 TSH HYPERSECRETION AND TSHα AND TSHβ SUBUNIT MEASUREMENTS IN NEPHROPATHIC CYSTINOSIS. Barry B. Bercu and Joseph D. Schulman. Neonatal and Pediatric Medicine Branch, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD 20205

We have previously reported partial pituitary resistance to thyroid hormone in cystinosis (J. Clin. Endocr. Metab. 51:1262, 1980). In 10 of these same patients and 3 others we measured basal serum concentrations of T_4, T_3, TSH, TSHα and TSHβ. In cystinotics, mean concentrations of T_3 (204 ± 9 SEM ng/dl) and TSH (19 ± 5 µU/ml) were elevated compared to age matched controls but mean T_4 (9.1 ± 0.7 µg/dl) was normal. In 7 patients the TSHα was elevated beyond the normal range (> 1.8 ng/ml). TSHβ levels were minimally increased in 2 patients and were normal in 11 others. All patients with elevated TSHα had increased TSH levels and the molar ratio of TSHα to TSH (2.2 ± 0.5) in their serum was equivalent to that of normal children. All cystinotics with increased TSHα were 5-10 years of age with moderate to severe renal impairment; some younger patients had abnormally elevated TSH but all had normal TSHα. Kouridis et al. reported high TSHα in patients with pituitary tumors but not in patients with isolated pituitary resistance to thyroid hormone (J. Clin. Endocr. Metab. 45:534, 1977). In contrast, most of the cystinotics with pituitary resistance had high TSHα levels. The extent to which renal impairment or other factors may account for the increased TSHα levels in cystinotics remains to be defined.

387 Thyronine Metabolism in Fetal Pulmonary type II Cells M. Segall-Blank, W.Douglas, R.Sanders, K.Hitchcock. (Spon. C.Anast.) Dept. Anat. Tufts U.Sch.Med. Boston,MA

The importance of thyroid hormone in fetal lung development and surfactant production is well known. Yet, triiodothyronine (T3), the active hormone, is present in low concentrations in fetal rat serum. We therefore studied metabolism of ^{125}I T_4 in surfactant-producing type II cells maintained in organotypic culture (Douglas, W.H.J. et al. In Vitro 12:373-381, 1976). The type II cells were obtained from fetal rat lungs at 16 and 19 days of gestation (term, 22-23 days). Cultures were incubated for 36 hrs. in medium enriched with ^{125}I T_4 at 37°C. Products of ^{125}I T_4 metabolism in medium, cell homogenate and a subcellular fraction enriched with nuclear material were assayed by chromatography. Confirmation by radioautography was performed. The distribution of radioactive compounds expressed as % of ^{125}I T_4 added to 10^8 day 19 cells and corrected for spontaneous degradation, follows:

	Subcellular fraction	medium	cell homogenate
%T_4 degraded	21.4±12.6	12.5±8.5	11.9±3
%Iodide formation	6.3±4.1	12.4±3.5	3.5±2.0
%T_3 of total activity	7.7±2.3	0.18±0.2	4.6±1.5

The results obtained from day 16 cells were similar. This suggests that fetal type II cells can deiodinate T_4. The high percentage of ^{125}I T_3 present in the cells suggests intracellular T_3 generation. There is a total net gain of ^{125}Iodide in the system. This excess iodide formation suggests pathways of thyronine metabolism which could result in formation and degradation of T_3. Such phenolic ring deiodination may terminate in 3,3'-T_2 or 3'-T or T_0.

● **388** PRIMARY HYPERPARATHYROIDISM (HPT) IN INFANCY. Casey Jason, Sara B. Arnaud, Michael R. Harrison, Dennis M. Styne, and Selna L. Kaplan. University of California, Department of Pediatrics, San Francisco, CA.

Hypercalcemia (↑Ca) due to HPT in infancy is rare and its laboratory diagnosis difficult. We report a case with unusual features in which the assay of parathyroid hormone (iPTH) aided in the diagnosis and in the evaluation of the novel therapeutic technique of autotransplantation. An 18 mo old female was noted to have ↑Ca (16.7 mg/dl) and hypophosphatemia (2.5 mg/dl) during evaluation for retardation. She was the first child of 5th cousins, both of whom had asymptomatic ↑Ca (11.4 and 10.4 mg/dl) and 'normal' levels of iPTH (31 and 35 µl eq/ml). The infant's head circumference was small (44 cm) and skeletal films showed little evidence of increased bone resorption in spite of high serum iPTH (125 ul eq/ml, normal, < 56). Treatment with dietary calcium restriction, saline, furosemide, phosphate, and calcitonin decreased serum calcium to 12 mg/dl; steroids did not influence the course. At surgery, 3 hyperplastic and one normal sized parathyroid glands were removed (140 mg, total wt). Portions of one gland were implanted into the brachial muscle. Normocalcemia was maintained by decreasing doses of Dihydrotachysterol postoperatively. This is the first known instance of HPT due to parathyroid hyperplasia in infancy in which both parents were affected with milder forms of the disease and, we believe, the youngest in whom the disease was treated by autotransplantation.