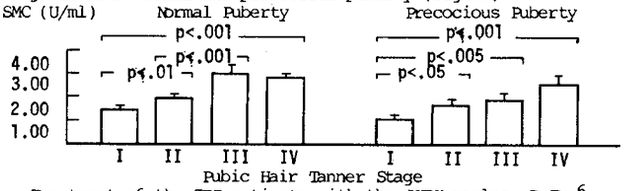
468 CORD SERUM THYROID STIMULATING HORMONE (TSH) AND THYROGLOBULIN (Tg) LEVELS DO NOT DECLINE WITH INCREASING WEIGHT IN NORMAL NEWBORNS WITH MEAN + SD BIRTH WEIGHT OF 3685 + 623 GRAMS OR GREATER. Robert Penny, Carole A. Spencer, John T. Nicoloff. Depts. of Pediatrics and Medicine, Univ. of So. Calif., Los Angeles, California.

Tg, TSH, free T_4 index, and free T_3 index were determined in cord serum and related to birth weight and sex in normal newborns, 12 females and 12 males. Free index values are the product of the T_3 resin uptake ratio and the appropriate thyroid function indice. Mean + SD birth weight of female infants (3685 + 623 grams) was significantly ($p < 0.025$) less than that of male infants (4104 + 248 grams). In contrast, the mean + SD free T_4 index value of female infants (8.4 + 0.7 µg/dl) was significantly ($p < 0.025$) greater than that of male infants (7.3 + 1.6 µg/dl). Free T_3 index values (58.1 + 4.2 vs 60.5 + 6.0 ng/dl), TSH values (7.0 + 3.8 vs 8.4 + 3.2 µU/ml, and Tg values (39.1 + 8.1 vs 38.3 + 12.0 ng/ml) of female and male infants did not differ significantly ($p > 0.1$). Free T_4 index values of female ($r = 0.804$, $p < 0.005$) and male ($r = 0.704$, $p < 0.01$) infants correlated positively with free T_3 index values. The log of TSH levels did not correlate with birth weight in female infants ($r = -0.291$, $p > 0.1$). However, male infants showed a positive correlation between the log of TSH levels and birth weight ($r = 0.539$, $p < 0.05$). Tg levels of female ($r = -0.282$, $p > 0.1$) or male ($r = 0.254$, $p > 0.2$) infants did not correlate with birth weight. These data, together with our prior observations, are consistent with a hypothesis that body composition may influence cord serum Tg and TSH levels.

●469 SOMATOMEDIN C IN PRECOCIOUS PUBERTY. Ora H. Pescovitz*, Florence Comite*, Karen Hench*, Gordon B. Cutler*, D. Lynn Loriaux*, Raymond L. Hintz†, Ron G. Rosenfeld†. *Developmental Endocrinology Branch, NICHD, NIH, Bethesda, Md., and †Stanford U., Dept. Pediatrics, Stanford, Cal.

Plasma somatomedin C (SmC) increases gradually throughout childhood and sharply at puberty. To assess the possible contribution of SmC to the accelerated growth of children with true precocious puberty (TPP), we have compared SmC levels in 41 1-7 year old children with TPP (33 girls, 8 boys), 87 age-matched controls, and 110 normal pubertal children. SmC levels were significantly greater in the TPP patients than in age-matched controls ($p<.01$). SmC levels correlated with Tanner stage in both normal and precocious puberty (Figure).



Treatment of the TPP patients with the LHRH analog, D-Trp^6-Pro^9-NEt-LHRH (4 ug/kg/d s.c.), decreased both the growth rate and the SmC level ($p<.02$) as determined by paired t-testing.

We conclude: (1) SmC levels are elevated in TPP, (2) SmC correlates with pubertal stage in TPP, and (3) LHRH analog therapy lowers the SmC level.

† 470 EFFECT OF FETAL THYROIDECTOMY ON THE METABOLIC RESPONSE TO BIRTH IN LAMBS. D.H. Polk, J.F. Padbury, C.C. Callegari, J. Newnham, A. Reviczky and A.H. Klein, Dept. of Pediatrics, Harbor-UCLA Medical Center, UCLA School of Medicine, Torrance, CA

The effect of perinatal changes in thyroid function on adaptation at birth as reflected in plasma free fatty acids (FFA's), catecholamines, glycerol and biophysical parameters was assessed in thyroidectomized (TX) (n=5) and sham (n=4) operated (control) fetal sheep. Surgery was done at 133±1 days gestation followed by cesarean delivery at 146±1 days gestation into a room held at 72°F. Measurements were made in utero before delivery and at timed intervals after birth. Blood gases, rectal temperature, pulse, respiratory rate, mean blood pressure, plasma glycerol and plasma norepinephrine levels were similar. Plasma epinephrine levels were markedly elevated after delivery in the TX animals with a mean (±SEM) peak level of 1849±593 pg/ml compared to a mean peak level of 263±129 pg/ml ($p<0.01$) in controls. FFA's were decreased in the TX animals with lower levels measured at 15 min (237±81 (SEM) µEq/L vs 688±79, $p<0.01$) and 30 min (439±66 vs 1026±230, $p<0.01$). T3 increased after delivery in controls (260-360 ng/dl) and was measurable but did not increase after delivery in the TX animals (8-45 ng/dl). Conclusions: 1) thyroid hormones play an important role in non-shivering thermogenesis after birth as reflected in decreased plasma FFA's in the TX animals; 2) altered thyroid status results in an augmented epinephrine response to birth; 3) absence of the perinatal T3 surge does not affect body temperature when the animal is exposed to a mild cold stress.

471 CHANGES IN ADRENAL GLAND MOTILIN CONTENT DURING DEVELOPMENT AND WITH PHARMACOLOGIC DENERVATION. Merrily M. Poth, Diane A. Proia, Jill Gustafson (Spon. by Roger E. Johnsonbaugh). Department of Pediatrics, Uniformed Services University, Bethesda, MD.

Motilin is a peptide first isolated from small intestine based on its effects on gut motility. It is widely distributed in neuroendocrine tissue including adrenal gland but its function in these tissues is not known. We measured adrenal gland motilin during development (day 20 of fetal life thru adulthood) and after treatment with drugs known to effect adrenal function. Adrenal glands from Sprague-Dawley rats were extracted and motilin content measured by radioimmunoassay. In drug studies young adult male rats were injected I.M. for 5 days with either dexamethasone 0.1 mg daily or reserpine 1 mg every other day. Control rats received saline. Motilin was not detectable in adrenal until 1 week of age. The concentration then increased until 5 wks of life when it reached adult levels. Reserpine, but not dexamethasone, had a marked effect on motilin content.

Treatment	Motilin picogm/pair adrenal (mean + SEM)
Saline (n = 12)	26.8 + 4.5
Dexamethasone (n = 12)	26.8 + 2.9 N.S.
Reserpine (n = 12)	51.1 + 4.6 $p < 0.001$

This increase after pharmacologic denervation of adrenal gland is consistent with a function for this peptide in the adrenal medulla. It is of interest in this regard that enkephalin, another neuropeptide found in adrenal, is released from adrenal gland during hypovolemic shock. Understanding of the specific function for motilin in this system awaits further study.

●472 EFFECT OF HUMAN GROWTH HORMONE (HGH) ON THE IMMUNE SYSTEM (IS) OF GH DEFICIENT CHILDREN. Robert Rapaport, James Oleske, Sally Solomon, Carol Delfaus, Raj Singh, Houman Ahdieh (Spon. by F. Behrle). UMD-New Jersey Med School, Children's Hospital of New Jersey, Dept Pediatrics, Newark, N.J.

We measured serum immunoglobulins, B cells, total, suppressor (S) and helper (H) T cells, lymphocyte and polymorphonuclear leukocyte (PMN) function in 8 GH deficient children before and at 1-3 month intervals for 1 year of HGH treatment (tx). The patients' ages were 1-17 years. HGH was provided by the National Pituitary Agency. The growth rates of all patients increased on tx. Prior to therapy the IS was normal in all. Treatment with HGH resulted in a significant fall in % B cells in 7/8 patients. By 12 months B cell values returned to pre-treatment levels in 4/7 and to higher than lowest values in all. (See Table)

Patient #	Mean(S.E.)	1	2	3	4	5	6	7
B)pre-tx	17.7 (2.3)	26	22	12	18	10	14	16
cell)lowest/mo.	*7.8 (1.6)	6/6	12/6	8/9	5/9	2/6	3/6	7/3
%)12 mo. tx	13.8 (1.3)	10	20	12	12	15	16	12

*6 mo. T test paired observat pre vs 6 mo $p<.01$, pre vs 12 mo N.S.

Lymphoblast responses to phytohemagglutinin (PHA) decreased in all 8 patients during the course of tx, in 7/8 by 6 months. At 12 months PHA response was suppressed in all 4 tested. A transient decrease in T H to T S cell ratio was noted in 4/8 patients. The rest of the immune parameters tested remained unchanged. None of the patients had an increase in number of infections during tx. We report here evidence that HGH tx has a profound influence on the immune system, affecting % B cells, PHA response and T H/S ratio.

●473 HYPERPROLACTINEMIA IN ADOLESCENCE: Geoffrey P. Redmond, Gita Gidwani, Leslie R. Sheeler (Spon. by Paul G. Dyment). Depts. of Endocrinology, Gynecology, and the Center for Children and Youth, The Cleveland Clinic Foundation, Cleveland, Ohio

Hyperprolactinemia is recognized commonly as a cause of menstrual dysfunction and hypogonadism in adults but is often assumed to be rare in adolescents. The clinical features of this in adolescence have not been well characterized. We have studied 20 patients with adolescent onset of hyperprolactinemia. Initial results on 16 (13 females and 3 males) are as follows: The cause of hyperprolactinemia was microadenoma in 6, macroadenoma in 7, pituitary cyst in 1 and tumor hydrocephalus in 2. Onset was between 9 and 19 years with a mean of 14.6 years but the diagnosis was delayed almost 5 years to a mean age of 19.3 years. Prolactin levels varied from 33 to 3450 ng/ml. Presenting complaints in females were: Amenorrhea in 64%, galactorrhea in 18%, cystic acne in 9%. Galactorrhea was present in 73% but only 1/3 of these were aware of it. Headaches were present in 82%. 6 of 8 patients had withdrawal bleeding after Provera.

3 patterns of pubertal progression were seen: 1)primary amenorrhea 18%, 2)normal menarche with only a few periods 36%, 3) normal menarche, irregular menses for several years and amenorrhea in 46%. 4 of 5 patients with macroadenomas have had surgery, radiotherapy or both and 3 of these have residual hyperprolactinemia and other residua. Although hyperprolactinemia is a common cause of menstrual or pubertal disturbance in adolescence, diagnosis is usually delayed. Because outcome is often suboptimal, an effort toward timely diagnosis is indicated.