DIHYDROTESTOSTERONE (DHT) BINDING ACTIVITY OF CULTURED SKIN FIBROBLASTS FROM FAMILIAL MALE PSEUDOHERMAPHRODITES WITH VIRILIZATION AT PUBERTY (MP-VP). Walter J. Meyer, Bruce S. Keenan, James I. Park, Howard W. Jones, Claude J. Migeon. The Johns Hopkins Univ. School of Medicine, The Johns Hopkins Hospital, Depts. of Ped. and OB-GYN. Baltimore, Maryland.

Typically, patients with male pseudohermaphroditism due to complete androgen insensitivity (MP-CAI) feminize at puberty (Testicular feminization). However, male pseudohermaphrodites with partial masculinization of the external genitalia may feminize or masculinize at puberty.

A highly specific low capacity DHT binding protein has been identified in the fibroblasts cultured from skin explants of normal males and females. The binding capacity (Bmax) and apparent dissociation constant (Kd) were determined by linear regression analysis of a double reciprocal plot of a saturation curve (see table). Similar binding activity is present in the fibroblasts from MP-VP but is not present in those who have MP-CAI and feminize at puberty. The specific DHT binding activity in cultured skin fibroblasts seem to be a useful tool in distinguishing and characterizing some types of male pseudohermaphrodites.

Subjects	Kd ($M \times 10^{-9}$)	Bmax ($M \times 10^{-18}$/ug DNA)
Normal males	0.33 to 1.50	173 to 2583
MP-CAI's	Unmeasurable	0 to <10
MP-VP's	1.05 to 1.36	314 to 1685

IS THERE A STEROID HORMONE CAPABLE OF PRODUCING HYPERTENSION IN MAN? Maria I. New, Paul H. Saenger, Lenore S. Levine, Ralph E. Peterson. Cornell Univ. Med. Coll., Depts. of Ped. and Med., New York City.

The hypothesis that a steroid can produce hypertension in man was tested in an 18 yr. old male with dexamethasone-suppressible hypertension. Reinvestigation after 6 yrs. of little or no treatment demonstrated that the hypertension and mild hyperaldosteronism were promptly decreased by a small dose of dexamethasone. During dexamethasone treatment, when aldosterone secretion was suppressed to less than normal and he was normotensive, steroids were given by constant infusion in an attempt to reproduce the hypertension of the dexamethasone-free state. Neither 5 days of aldosterone (A) nor 18-hydroxy desoxycorticosterone (18-OH DOC) at 1 mg/day nor DOC at 30 mg/day caused hypertension. However, Na^+ retention and K^+ loss was observed during A and DOC infusion. Hypertension was produced within 5 days during infusion of ACTH or oral metyrapone. The hypertensive effect of the latter was eliminated by addition of aminoglutethimide treatment. These studies suggest that an ACTH dependent steroid other than A, 18-OH DOC, or DOC may be the cause of the hypertension in this patient. The aminoglutethimide data suggest that it is a steroid, and the metyrapone studies suggest that steroid is an 11 desoxysteroid. Urine and blood collected under ACTH stimulation and metyrapone treatment is a rich source from which we may characterize this hormone.

PLASMA GROWTH HORMONE (GH) DURING CATCH-UP GROWTH (CUG) AFTER FASTING OR CORTISONE INJECTIONS. H. David Mosier, Jr., and Regina A. Jansons, Univ. California (Irvine) Col. Med., Dept. Ped., and Memorial Hosp. Med. Ctr., Long Beach, California.

This work was undertaken to determine whether GH is involved in CUG after fasting or cortisone injections in the postweaning period. Male Long-Evans rats, 40 d of age, were fasted 2 d or injected sc with 5 mg cortisone acetate daily for 4 d. Untreated rats served as controls. We have shown in these models complete CUG after fast but only return to normal growth rate after cortisone. Plasma GH in decapitation neck blood was determined by radioimmunoassay (Ref NIAMD-Rat-GH-RP-1) before, at the end of treatment, and weekly for 4 w. Pituitary weight and GH content, body size, and cell size and number of heart, skeletal muscle, and liver were determined. The results showed a significant increase of mean values as well as an increased occurrence of very high (>250 mμg/ml) levels of GH during the posttreatment period in both experimental groups. Control plasma GH was 42 mμg/ml± 11(SE). At 4 w plasma GH remained significantly elevated in fasted rats. It was normal in cortisone rats. Soft tissue parameters were markedly different between fast and cortisone rats at the end of treatment. These approached normal by 4 w in fasted rats. Cortisone rats had reduced liver size and cell number at 4 w. The results are compatible with the view that GH metabolism is involved in CUG. The mechanism responsible for the increased plasma GH values during the posttreatment period and the possible role of GH in CUG remain to be determined.

CORRELATION OF SERUM LUTEINIZING HORMONE (LH) AND FOLLICLE STIMULATING HORMONE (FSH) CONCENTRATIONS WITH HEIGHT AND WEIGHT. Robert Penny, N. Olatunji Olambiwonnu and S. Douglas Frasier. Univ. So. Calif. Sch. Med., Los Angeles County-USC Med. Ctr., Dept. Pediatrics, Los Angeles.

Serum LH and FSH concentrations were determined in 16 normal girls (6-16 yrs of age) and 16 normal boys (9-18 yrs of age) and plotted against height and weight. There was a positive correlation between serum LH and height in girls ($r=0.60$; $p < .01$) and in boys ($r=0.89$; $p < .005$). A similar positive correlation was found between serum FSH and height in girls and boys ($r=0.63$; $p < .005$ for both sexes). A positive correlation was also observed between serum LH and weight in girls ($r=0.60$; $p < .01$) and boys ($r=0.91$; $p < .005$) and between serum FSH and weight in girls ($r=0.47$; $p < .05$) and boys ($r=0.71$; $p < .005$). More than two-thirds of the observed concentrations of LH and FSH fell within one standard deviation of calculated regression lines. Speculation: Cell mass can be predicted from the relationship between body composition and height and weight. From infancy to adolescence, there is a linear relationship between basal heat production and visceral cell mass. Serum LH and FSH concentrations would be expected to increase with height and weight if it is assumed that the attainment of a critical metabolic rate may be associated with reduced sensitivity of the hypothalamus to sex steroids giving rise to a change in the gonadal-hypothalamic feedback set-point.

LOW LEVELS OF TRIIODOTHYRONINE IN ANOREXIA NERVOSA. Thomas Moshang, Jr., John S. Parks, Vanitha Vaidya, Robert Utiger and Alfred M. Bongiovanni. Hahnemann Med. Coll., Dept. of Ped., and the Univ. of Pa. Sch. of Med., Dept. of Ped. & Med.

There are many biochemical and clinical findings in patients with anorexia nervosa to suggest altered thyroid hormone activity but laboratory tests of thyroid gland function are generally normal or low normal. TSH, total T_4, total T_3 and the response to TRH were determined in 6 patients with anorexia nervosa in an effort to evaluate discriminately thyroid hormone activity at the physiologic levels of the hypothalamus, pituitary gland, thyroid gland and peripheral tissue. The results, expressed as the mean value ± SEM, are summarized in the following table:

	T4(μg%)	T3(ng%)	TSH(μU/ml)	Δ TSH
Patients	7.2 ± 1.01	49.7 ± 10.7	3.0 ± 0.52	9 ± 2.38
Controls	9.5 ± 0.29	106.0 ± 7.5	3.6 ± 0.43	12 ± 1.37

The most striking finding was the markedly depressed T_3 levels. Animal studies suggest that in acute starvation, peripheral deiodination of thyroxine is increased. Our data suggest that during chronic starvation, peripheral deiodination of T_4 to T_3 is diminished. Sullivan et al have suggested that severely ill but euthyroid patients have decreased deiodination and decreased intracellular T_3. Teleologically, decreased deiodination may be a protective mechanism during chronic stress.

EPISODIC SECRETION OF GONADOTROPINS IN CHILDREN AND ADOLESCENTS. Robert Penny, N. Olatunji Olambiwonnu and S. Douglas Frasier. Univ. So. Calif. Sch. Med., Los Angeles County-USC Med. Ctr., Dept. Pediatrics, Los Angeles.

The concentrations of LH and FSH were determined in serum samples obtained from 7 normal girls (ages 9.5-16.5 years) and 8 normal boys (ages 9.0-16.8 years) at 15 minute intervals during a 4 hour period. An episodic pattern of LH but not FSH secretion was seen in all subjects. The number of episodes per subject ranged from 1 to 3. Time of occurrence of secretion episodes ranged from 0 (initial sample) to 240 minutes with a mean (± SD) of 133.8 ± 65.7 minutes. The incremental rise in LH concentration ranged from 1.1-5.3 mIU/ml with a mean (± SD) of 2.8 ± 1.3 mIU/ml. The mean (± SD) percentage increment was 41.6 ± 9.8% with a range of 31.3-58.0%. No sex difference in the number of secretion episodes, incremental rise in LH concentration or time of occurrence of secretion episode was demonstrated. Mean LH and FSH concentrations increased with age and stage of sexual development. All LH and FSH concentrations were within the range previously reported for normal girls and boys. This data indicates that FSH is not secreted episodically in normal children and adolescents. The previous observation of episodic LH secretion is confirmed. It is speculated that differential feedback of the sex steroids is responsible for the lack of concurrence in LH and FSH secretion patterns.