498 INSULIN LEVELS IN NEONATES OF DRUG ADDICTED MOTHERS. M.Vadapalli, A.Zubrow, G.Bhatia, S.W.Nicholas, B.Thyson*, D.Bateman, I.Fennoy. Department of Pediatrics, Harlem Hospital Center, College of Physicians & Surgeons of Columbia University, New York,N.Y. *Lab.Medicine, OBS-GYN, Albert Einstein School of Medicine.

Fetal insulin is considered to be an intrauterine growth promoting hormone. Intrauterine growth retardation has been one of the major problems of newborn of drug addicted mothers. Umbilical cord venous blood was collected from full term small for gestational age babies (SGA) born to drug addicted mothers (DA). Controls (C) include normal full term appropriate for gestational age (AGA) and small for gestational age babies of non-drug addicted mothers. Gestational age was assessed by DUBOWITZ and lens examination in all the newborns. The results are as follows:

INSULIN VALUES (per units/ml)

AGA-C	AGA-DA	SGA-C	SGA-DA
n = 7	n = 7	n = 7	n = 7
34.55*	14.06+	8.5#	5.1@
±10.8	±6.38	±1.4	±1.4

n ± SD * vs. + P<.05 # vs. @ P<.05

There is significant decrease in insulin leves in SGA newborns as compared to AGA group. The insulin levels of AGA-DA group are significantly lower than AGA-C but significantly higher than SGA-C or SGA-DA. Insulin levels in SGA-DA were significantly lower than in SGA-C. These results suggest that intrauterine growth retardation of newborns of drug addicted mothers is a process mediated by insulin. Further studies are needed.

499 PROLACTIN HOMEOSTASIS IN VLBW INFANTS: EFFECT OF HYPOCALCEMIA (HC) AND CA INFUSION ON PROLACTIN RELEASE AND SECRETION IN NEONATES. P.Venkataraman, K.Blick, M.Parker, H. Fry. U. Okla.(Spon. O.M.Rennert).

In vitro, prolactin secretion but not release is influenced by media Ca. Prolactin homeostasis in VLBW infants and effect of neonatal HC on secretion and release of prolactin are unclear. In 6 preterm AGA neonates, birthwt. 1050 ± 96 gms(mean ± sd), gestation < 32 wks, we studied the theses: 1)serum prolactin would be high; 2)serum Ca <6.0 mg/dl would be associated with decline in serum prolactin; 3)Ca infusion would not affect prolactin release; but may increase prolactin secretion. Serum Ca, iCa, Mg, P, and prolactin were determined on entry; when serum Ca <6.0 mg/dl; immediately post infusion of 18 mg/kg of Ca as 10% Ca gluconate over 10 minutes; +8 hrs post Ca infusion. Serum prolactin was determined by RIA (N <18 ng/ml in males, < 25 ng/ml in females). Serum total Ca was 7.4 ±0.2 at 11 ± 2.6 hrs of age, and declined to 5.1 ±0.2 mg/dl (mean ± se,p <0.025) at 46 ±9 hrs and rose to 9.2± 0.7 and 7.1 ± 0.5 mg/dl immediately and +8 hrs post Ca infusion; serum iCa was 5.24 ± 0.3, 3.8 ± 0.2, 6.9 ± 0.6 and 4.2 ± 0.2 mg/dl at these times, serum Mg and P did not change. Basal serum prolactin was elevated 209 ±41 ng/ml ($p < 0.05$ vs adult control) and declined to 121 ± 23 ng/ml, $p < 0.05$, associated with hypocalcemia; immediately post Ca serum prolactin was 124 ± 28 ng/ml and 129 ± 20 ng/ml at +8 hrs post Ca. Thus, in VLBW infants, 1) serum prolactin is extremely high; 2) serum prolactin declines postnatally associated with decline in serum Ca low as 5.0 mg/dl; 3)in HC Ca infusion does not cause acute or subacute changes in serum prolactin. We speculate that although serum Ca and prolactin show similar temporal changes, serum Ca, low as 5.0 mg/dl with low serum iCa does not modify prolactin homeostasis.

500 HEMOGLOBIN LEVELS IN HYPOTHYROID INFANTS. Mark Weinblatt, Pavel Fort and Joseph Kochen. Cornell Univ. Med. Coll. and North Shore Univ. Hosp., Depts. of Pediatrics, Manhasset, NY.

Children and adults with long-standing hypothyroidism frequently develop anemia, often of a macrocytic nature. The incidence of anemia has been reported to be between 21 and 61% in adults. We investigated newborns with hypothyroidism for the presence of anemia. All infants were initially determined to have abnormal thyroid function by the N.Y. State Screening Program with heel stick blood specimens obtained by day 3 of life. Newborns with elevated TSH levels (>20 μU/ml) or low T_4 levels (<8 ug/dl) were included in the study. Repeat thyroid studies were performed at 10 to 55 days of life, and blood counts with RBC indices were measured by Coulter Counter. All children with other causes of anemia or polycythemia were excluded from analysis. Of 23 infants who fit these criteria, none were found to be anemic, nor did any have macrocytic indices. Surprisingly, 6 children (26%) were discovered to have polycythemia, 4 with significantly elevated hemoglobins as high as 23 g/dl. All children with polycythemia had normal red cell indices. The hemoglobin showed no correlation with T_4 or TSH levels. Anemia in patients with hypothyroidism is likely to be a result of chronically abnormal thyroid function, and as such, would not appear to be helpful in screening infants for thyroid dysfunction.

501 SERUM THYROXINE (T4), FREE T4 (FT4) AND TSH IN PREMATURE INFANTS LESS THAN 1000 GRAMS DURING THE FIRST THREE MONTHS OF LIFE. W. Howard Whiteside, Paul R. Williams, Allen W. Root, Jack Strzlecki, John S. Curran, and Keith S. Kanarek, Department of Pediatrics, College of Medicine, University of South Florida, Tampa, Florida.

The incidence of transient hypothyroidism in very low birth weight infants has not been delineated. We studied 22 premature infants <1000 grams and <30 weeks gestation. Eight infants died within four days and their data excluded.

Age	T4 μg/dl*	N	Free T4 ng/dl	N	TSH μU/ml	N
Cord	7.10 ± 1.22	6	1.40 ± 0.26	6	4.28 ± 2.23	6
Day 1	7.77 ± 0.81	9	1.52 ± 0.10	9	3.53 ± 1.55	7
Day 2	5.29 ± 0.48	7	1.19 ± 0.07	7	1.08 ± 0.33	5
Day 3	9.45 ± 2.88	6	1.49 ± 0.20	6	1.60 ± 0.33	5
1 Wk	5.32 ± 1.41	13	1.09 ± 0.24	13	2.83 ± 1.07	11
2 Wks	3.20 ± 0.98	10	0.79 ± 0.15	10	5.89 ± 1.97	10
4 Wks	4.64 ± 0.83	9	1.03 ± 0.14	9	2.90 ± 0.61	9
8 Wks	7.43 ± 1.28	8	1.30 ± 0.14	8	3.16 ± 1.14	7
12 Wks	7.88 ± 1.75	5	1.53 ± 0.14	6	2.20 ± 0.46	5

*All values expressed as mean + S.E.M.

The nadir of T4 and FT4 and the peak TSH levels occurred at 2 weeks. Eight of 10 babies had T4's <6μg/dl and 7 had FT4's <0.8ng/dl (below accepted normal values). No TSH value was >20μU/ml. The FT4's were lower than anticipated. These data are compatible with transient hypothyroxinemia. However, it is possible that these babies had transient hypothyroidism with a blunted TSH response secondary to immaturity of the hypothalamo-pituitary-thyroid axis.

●502 ADRENARCHE AND GROWTH DURING LHRH AGONIST ($LHRH_a$) ADMINISTRATION. Margaret Wierman, Donna Beardsworth, John Crawford, John Crigler, Hans Bode, David Kushner and William Crowley, Depts Gyn, Med and Ped, Vincent Res. Labs, Mass. General Hospital, Children's Hospital, Boston, MA.

During puberty the effect of adrenal androgens on skeletal maturation and growth is obscured by the influence of gonadal steroids. Suppression of gonadarche with an $LHRH_a$ affords an opportunity to explore the association between adrenarche, skeletal development and adult stature. In 16 girls with central precocious puberty (CPP), gonadarche was suppressed by $LHRH_a$ administration for 12 to 36 months. During treatment dehydroepiandrosterone sulfate (DS) levels, an index of adrenarche, were constant or increased in an age expected fashion. Rate of bone maturation (ΔBA) for change in chronologic advancement (ΔCA) decreased as shown by $\Delta BA/\Delta CA < 1$ in all subjects. DS levels were positively correlated with $\Delta BA/\Delta CA$ ($R = 0.07$, $P < 0.002$). Statural growth also decreased but less than bone maturation so that predicted mature height (Bayley and Pinneau) was increased in all but one subject. A significant negative linear regression of DS levels with increase in predicted height was observed ($R = -0.70$, $P < 0.003$). Conclusions: 1) In girls with CPP, adrenarche progressed during $LHRH_a$ suppression of gonadarche. 2) In preadrenarchal girls, $LHRH_a$ administration was associated with a more striking slowing of bone maturation with relatively lesser effects on height velocity. In those girls with the onset or progression of adrenarche in the absence of gonadal steroids, therapy was associated with less evidence of restraint on bone maturation. 3) These data are compatible with a direct effect of adrenal androgens upon skeletal maturation and growth.

503 **SUBCUTANEOUS VERSUS INTRAMUSCULAR HUMAN GROWTH HORMONE THERAPY: GROWTH AND ACUTE SOMATOMEDIN RESPONSE.** **DM Wilson, BK Baker, RL Hintz, and RG Rosenfeld, Stanford University, Dept. of Pediatrics, Stanford, CA**

Although growth hormone (GH) has traditionally been administered intramuscularly (IM), concerns regarding patient comfort and compliance have led to the consideration of the subcutaneous (SQ) route. To compare SQ and IM administration of GH, we have examined the acute insulin-like growth factor I (IGF-I) & II response to 4 daily injections of GH (0.1 U/kg) in 21 GH deficient children. Subjects were off GH for at least 2 weeks prior to randomization to SQ (11) or IM (10). Samples were obtained at baseline and 20 hr after the 4th injection. IGF-I and IGF-II were measured by specfic RIAs. Growth rate and antibody levels were determined before and after 6 months of three times a week therapy. We evaluated parent and subject preference in those who switched to SQ by questionnaire. IGF-I levels tripled after 4 days of GH in both groups (43±58 (±SD) to 149±106 ng/ml SQ, 43± 27 to 144±65 IM) while IGF-II levels nearly doubled (537±326 to 1014±332 ng/ml SQ, 617±394 to 1088±426 IM). In those who had completed 6 months of therapy, there was no significant difference in the growth rates (5.04±1.26 cm/yr SQ, 6.72±1.09 cm/yr IM) and only one patient developed GH antibodies (IM group). Both parents and subjects expressed a strong preference for the SQ method (8 of 9 thought SQ better than or equal to IM). The identical rises of both IGF-I and IGF-II following a brief course of either SQ or IM GH, the similar growth rates and lack of antibody development all suggest that the two routes are equivalent, while patients and parents clearly prefer SQ therapy.