589 HEMOGLOBIN CONCENTRATION (Hb) DIFFERENCES BETWEEN BLACKS AND CAUCASIANS: GENETIC OR ENVIRONMENTAL? Peter R. Dallman, George D. Barr, Constance M. Allen and Henry R. Shinefield. University of California Medical Center, Department of Pediatrics, San Francisco and Kaiser Permanente Medical Center, San Francisco.

Blacks have a Hb 0.5 to 1.0 g/dl lower than that of income-matched caucasians in several large surveys. This difference could be a racial characteristic of blacks or it might be due to a higher frequency of genetic traits such as thalassemia minor and hemoglobinopathies or to environmental factors such as iron deficiency. To help in making this distinction, we analyzed the data from multiphasic examinations (1973-75) on 1718 caucasians and 741 black, healthy, non-indigent children between 5 and 14 yr of age. Anthropometric measurements (height, weight, skin fold thickness), Hb, red cell indices and Hb electrophoresis were analyzed. In the entire population, the median Hb at each age averaged 0.5 g lower in blacks than in caucasians of both sexes ($p<0.001$). The difference still averaged 0.5 g/dl ($p<0.001$) after exclusion of all those with abnormal Hb electrophoresis (Hb S and C) and those whose mean corpuscular volume (MCV) was more than 5% below the normal mean for age (to exclude iron deficiency or thalassemia minor). Abnormal Hb was found in 1% of caucasians and 12% of blacks. Exclusions based on MCV were made in 10% of caucasians and 21% of blacks. There was no association between obesity and Hb. The data strengthen the impression that blacks normally have a Hb about 0.5 g/dl less than in caucasians. If this proves to be the case, 10% of normal blacks would be mistakenly designated anemic unless different norms were adopted.

590 TREATMENT OF HYPOPLASTIC ANEMIA WITH PLACENTAL TRANSPLANTS. Joseph Dancis, Valerie Jansen, George F. Brown, Fred Gorstein & M. Earl Balis, N.Y.U. School of Medicine, Dept. of Pediatrics & of Pathology & Memorial Sloan-Kettering Institute, New York City.

A genetic mutation in mice (W/W^v) causes an autosomal recessive disease characterized by hypoplastic anemia which lasts throughout life. Homozygous W/W^v anemic mice were sublethally irradiated to facilitate repopulation of marrow with transplanted cells and injected IV with suspensions of 5-10 million placental cells of 15 days gestation derived from normal, isogeneic donors. RBC fell promptly after irradiation and then rose progressively over a period of weeks reaching normal levels for the non-mutant. Mean corpuscular volume and hemoglobin electrophoresis pattern of RBC in recipient W/W^v mice resembled those of normal donor animals. The therapeutic effect lasted for the duration of the observation period, in some instances over 7 months. W/W^v mice that were administered Hanks' solution or fetal blood, instead of placental transplants, remained anemic. Late gestation placentas (13 days) were also ineffective. The placental cells with hemopoietic potential could not be identified by routine histological examination.

591 ADRENAL INTEGRITY IN ACUTE LYMPHOCYTE LEUKEMIA (ALL) AND NON-HODGKINS LYMPHOMA (NHL). Pedro A. DeAlarcon and James A. Stockman, III, Dept. of Ped., SUNY, Syracuse, N.Y. (Spon. by Frank A. Oski).

Present treatment schedules for ALL and NHL include steroid therapy. While Wilson (Lancet 1:610, 1976) demonstrated impaired adrenal responses in 2/10 with myeloma and Hodgkins disease, adrenal integrity and the need for steroid tapering have not been thoroughly evaluated in ALL and NHL. To determine whether adrenal suppression occurs following short courses of steroid, a study was undertaken in 10 subjects (6 ALL, 4 NHL) receiving prednisone or dexamethasone during remission maintenance. Previously all had received induction steroid and currently received steroid either 1 wk. monthly or 2 wks. quarterly. Other drugs given included 6 mercaptopurine, methotrexate and in some cytoxan. Plasma cortisols were determined prior to each steroid course and 24 hrs. after a non-tapered discontinuation of steroid. It was found that prior to periodic steroid therapy 5/10 subjects had abnormally low levels of cortisol, 3/10 had low normal levels while only 2/10 had clearly normal cortisol. Following steroid, 8/10 showed a definite decrease in cortisol and overall 8/10 demonstrated clear evidence of adrenal suppression. The suppression was equivalent with either dexamethasone or prednisone and was not related to the time from diagnosis. Clinical adrenal insufficiency was not noted even at the time of severe stress. Adrenal suppression is present throughout much of maintenance therapy in ALL and NHL. These studies indicate that steroid tapering should be undertaken and supplementation considered at times of illness.

592 GRISEOFULVIN THERAPY AS A CAUSE OF LYMPH NODE PROLIFERATION RESEMBLING MALIGNANT HISTIOCYTOSIS. Louis P. Dehner, William Krivit; University of Minnesota School of Medicine and Hospitals, Departments of Laboratory Medicine and Pathology, and Pediatrics, Minneapolis.

The relationship between lymphadenopathy and certain drugs especially the hydantoins has been well documented. Although the histologic appearance of the lymph node may be quite disturbing and suggest a malignant lymphoma, most cases, but not all, are clinically and pathologically benign. This report documents the occurrence of a lymph node proliferation in a child which resembled malignant histiocytosis. The differential diagnostic problem between malignant histiocytosis and immunoblastic lymphadenopathy in childhood is illustrated and explored in the context of this case.

A 12-year-old male developed a generalized rash, fever, diffuse lymphadenopathy (including mediastinal) and hepatosplenomegaly soon after the onset of Griseofulvin therapy. An enlarged cervical lymph node revealed a predominatly sinusoidal proliferation of large, atypical "histiocytoid" cells and abundant mitotic activity. Partial obliteration of the follicular architecture had occurred. Following an interpretation of probable malignant histiocytosis, Griseofulvin therapy was discontinued and it was elected to withhold further treatment. All abnormal clinical findings regressed and the patient has remained well for over 2 years. A repeat lymph node biopsy was unremarkable. Retrospectively, the atypical "histiocytoid" cells represented, in fact, "immunoblasts" or transformed lymphocytes and the process more appropriately belongs to the category of immunoblastic lymphadenopathy (N. Engl. J. Med, 292: 1, 1975. Am J Med 59:803, 1975).

593 INSIGNIFICANCE OF PROGNOSTIC INDICATORS AFTER 2-YEAR SURVIVAL IN ACUTE LYMPHOBLASTIC LEUKEMIA (ALL). Nancy L. Dunn, Nancy B. McWilliams, and Harold M. Maurer. Medical College of Virginia, Department of Pediatrics, Richmond, Virginia.

52% (11/21) of our children with ALL in marrow remission for >4 years had been classified as "high risk" because of WBC > 30,000/mm^3 and/or age < 2 or > 7 years at diagnosis. Because of the surprisingly high percentage of "high risk" patients among our long-term survivors, we retrospectively studied 61 children with ALL followed for 2 - 12 years post-diagnosis, to determine whether there was a limited interval during which these unfavorable indicators were operative. 24 patients with WBC > 30,000/mm^3 had shorter first remissions, a shorter mean survival time and there were fewer survivors at 2 years ($p < .005$ for each). After 2 years, high initial WBC had no significant predictive value ($p > .3$). Survival in 14 patients with unfavorable age at diagnosis at no time differed significantly from those of favorable age.

There was no correlation between WBC and frequency of CNS leukemia; but 64% ($p < .1$) of CNS relapses occurred in the "high risk" age group, 2/3 within 2 years of diagnosis.

We conclude that: 1) the initial WBC is a valid predictor of survival for 2 years after diagnosis, but is invalid if survival exceeds 2 years; and 2) unfavorable age at diagnosis predisposes to CNS relapse in the first 2 years.

594 RED BLOOD CELL (RBC) SURVIVAL IN IRON DEFICIENT INFANTS DETERMINED BY ANALYSIS OF RBC SIZE DISTRIBUTION CURVES. F.R. Ellwanger, A.L. Lightsey, H.M. Koenig (spon. by W.L. Nyhan), Naval Regional Medical Center, San Diego.

Shortened survival of iron-deficient RBC has been demonstrated by isotope labeling and cross-transfusion techniques. Survival of iron-deficient RBC in 7 infants was determined by analyzing serial changes in RBC size distribution curves during treatment. RBC size (volume) distribution curves were measured on a Coulter Channelizer. Pretreatment RBC were microcytic and had unimodal, log-normal size distribution curves. During treatment size distribution curves became bimodal as a new, larger sized population of RBC entered the circulation. Bimodal curves were separated by curve matching into large and small size RBC population curves. The area under each curve was proportional to the number of cells in that population. Initial hemoglobin (Hb) and RBC half-life determined by analysis of cell size distribution curves in the 7 infants were:

Hb (g/dl)	3.1	4.0	5.1	7.4	7.6	7.9	8.3
RBC half-life (days)	16	18	18	34	30	50	36

Severity of anemia correlated with shortened RBC survival ($r=0.86$) RBC survival in these 7 infants was shorter than survival of ^{51}Cr labeled cells in non-anemic infants ($p < 0.001$). Analysis of RBC size distribution curves is a useful method of measuring RBC survival when correction of anemia produces a change in RBC size. Measurement can be accomplished on microliter quantities of blood without using isotopes.