842 ALLOGENEIC BONE MARROW TRANSPLANTATION IN THE SETTING OF A GENERAL CHILDREN'S HOSPITAL. W.L. Nix, K.A. Starling, J.D. Easley, C.P. Steuber, D.H. Mahoney, Jr., D.J. Fernbach. Departments of Pediatrics and Radiology, Baylor College of Medicine, Houston.

Bone marrow transplantation (BMT) has been performed in specialized centers because of perceived needs for supportive care measures such as laminar flow rooms (LFR), gut sterilization (GS), and leukocyte transfusions (LT). Hospitalizations in such settings are costly. Since June 1979, allogeneic BMT has been performed 11 times in 10 patients (pts) at Texas Children's Hospital: For aplastic anemia (2 pts) (1 pt transplanted twice); acute nonlymphocytic leukemia (4 pts); and acute lymphocytic leukemia (4 pts). All pts were cared for with standard reverse isolation techniques without LFR, GS, or prophylactic LT. Recipients were conditioned with cyclophosphamide; leukemia pts also received total body irradiation (TBI). Identical sibling donor bone marrow was infused 8-12 hours post TBI. All pts required antibiotic treatment for suspected sepsis. Fatal pseudomonas sepsis occurred in 1 pt despite antibiotic therapy and LT. All pts were transfused daily with irradiated platelet concentrates and received methotrexate to prevent graft versus host disease; all achieved marrow engraftment, identified by rising blood counts by 7-20 (mean 13) days post BMT. Eight surviving pts (9 BMT procedures) were discharged 18-30 (mean 24) days post BMT with satisfactory blood counts. These data demonstrate that BMT can be safely performed in a children's hospital without expensive ancillary equipment or procedures.

843 EFFECT OF VINCRISTINE UPON YEAST PHAGOCYTOSIS BY NEUTROPHILS IN LEUKEMIC CHILDREN DURING REMISSION. Uri Z. Nordan, James R. Humbert, Cameron K. Tebbi, Linda L. Moore. State University of New York at Buffalo, Roswell Park Memorial Institute and Children's Hospital of Buffalo, (Departments of Pediatrics), Buffalo.

During remission, patients with acute lymphoblastic leukemia (ALL) may develop fungal infections. We investigated the possible contribution of drug-induced neutrophil (PMN) dysfunction to such infections before and after a single dose of vincristine (VCR) in 9 leukemic children in remission. At the time of "pulse" therapy, PMN yeast phagocytosis was performed before, 1 hour after and 24 hours after injection of VCR (2 mg/M^2). Prednisone was omitted for 24 hours. Baker's yeast (Candida saccharomyces) phatocytosis was determined by a cytochemical technique. Slides were coded and read in blind fashion. Seven normal adults served as controls. Results are reported as number of yeast particles per 200 PMN ($x \pm SE$). Yeast phagocytosis before VCR injection in ALL patients (865 ± 47) was not significantly lower than in the controls (948 ± 46, $P > 0.1$). Results obtained 1 hour (715 ± 67) and 24 hours (632 ± 63) after VCR administration showed a progressive and significant decrease in phagocytosis as compared with pre-VCR values ($P < 0.025$). In ALL patients during remission a significant defect in yeast phagocytosis develops immediately after VCR administration; this defect progresses further in the next 24 hours and may contribure to the propensity toward fungal infections observed in some patients.

844 SUCCESSFUL TUMOR IMMUNOTHERAPY WITH CIMETIDINE BY ABROGATION OF SUPPRESSOR CELLS. M.E. Osband, P. Lavin, A. Brown, R. McCaffrey, Boston University Medical Center, and Sidney Farber Cancer Institute, Boston, MA 02115; Smith Kline Corp., Philadelphia, PA (spon. by Joel Alpert)

Immunotherapy has generally been aimed at stimulation of the patient's anti-tumor immune response. Since tumor growth may be associated with development of suppressor cells which could undermine an otherwise effective anti-tumor immune response, we have reasoned that an alternative immunotherapeutic strategy is the inactivation of suppressor cells by pharmacologic means, thereby allowing a more effective host anti-tumor immune response. Suppressor cells have a histamine H2 receptor on their surface which can be blocked by the H2 antagonist cimetidine (C). We therefore studied the effect of C on 3LL tumor in C57Bl/6 mice. Tumors were allowed to grow until 7.5 mm at which time they were surgically removed, and mice were randomized to control or C groups. The latter received C in their drinking water at 0.2, 2.0 or 10.0 mg/ml. The mice receiving 10 mg/ml C had a significant decrease in metastatic development (25 and 54 surface pulmonary metastases in control mice at 5 and 12 days after surgery; 6 and 16 in the C group, $p<.01$). C at all three dosages produced a significant prolongation in survival ($p<.01$ for 0.2 and 2.0 mg/ml groups; $p<.001$ for 10.0 mg/ml C group). These effects were associated with suppressor cell inactivation (47% suppression in control; 21% in 10 mg/ml C group). We conclude that C slows metastatic development and prolongs survival by abrogation of suppressor cells. This suggests that a similar immunotherapeutic approach in human neoplasia is appropriate.

✳ **845** THE HEMOLYTIC EFFECT OF LEAD ON GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENT ERYTHROCYTES. M. Osband, D. Shipman, E. Cohen, R. McCaffrey, J. Alpert, Boston City Hospital and Boston University Medical Center, Boston, MA 02118

Based on our experience of a G6PD deficient patient with recurrent episodes of severe hemolysis associated with plymbism, we studied the effect of lead on erythrocytes from 3 normal and 4 stable G6PD deficient patients (3 with A-, 1 with B- variants). RBC's were washed, resuspended 5% v/v in culture medium, incubated for 2 hours at 37°C with lead acetate and/or phenylhydrazine (PH), and the percentage hemolysis determined. At concentrations tested (10-80ug/100ml) lead caused hemolysis only in the G6PD deficient cells. For example, normal RBC's demonstrated $.60 \pm .10\%$ hemolysis in the absence of lead and $.53 \pm .05\%$ in its presence (40ug/100ml); G6PD deficient RBC's showed $.69 \pm .05\%$ and $2.11 \pm .57\%$ hemolysis under similar conditions, $p<.001$. When cells were exposed to PH (0.5mg/ml) as an oxidant stress, the additional presence of lead (40ug/100ml) significantly exacerbated ($p<.01$) the resultant hemolysis in the G6PD deficient cells:

	% hemolysis		
	No Additive	PH Alone	PH + Lead
Normal	$.67 \pm .01$	$.49 \pm .01$	$.55 \pm .08$
G6PD deficient	$.61 \pm .09$	$.87 \pm 02$	$1.21 \pm .05$

We conclude that lead, at levels seen in minimally poisoned children, represents a heretofore unrecognized hemolytic stimulus in G6PD deficiency. Since the incidence of G6PD deficiency is high in children at risk for plumbism, it is important to recognize lead as being capable of both causing and exacerbating hemolytic crises in these patients.

● **846** EFFECT OF IRON DEFICIENCY WITHOUT ANEMIA ON INFANT BEHAVIOR. **Frank A. Oski, Alice S. Honig, Brenda M. Helu, Peter H. Howanitz. Upstate Medical Center, State University of New York & Syracuse Univ., Syracuse, N.Y.**

We have reported that treatment of infants with iron deficiency anemia produces a prompt increase in infant performance as measured by the Bayley scores of mental development (MDI). The present study was designed to determine if iron deficiency in the absence of anemia (Hb ≥11.0 g/dl) was also associated with behavioral alterations. Infants, 9-13 months of age, were classified based on hemoglobin, serum ferritin, erythrocyte protoporphyrin (FEP) and red cell size (MCV) as follows:

	Hb	Ferritin	FEP	MCV
Normal	≥11.0	>12 ng/ml	<30 µg/dl	>70 fl
Iron depleted	≥11.0	<12 ng/ml	<30 µg/dl	>70 fl
Iron deficient-I	≥11.0	<12 ng/ml	≥30 µg/dl	>70 fl
Iron deficient-II	≥11.0	<12 ng/ml	≥30 µg/dl	≤70 fl

Subjects were administered the Bayley MDI, treated with intramuscular iron, and then retested exactly one week later. All tests were administered by the same psychologist without knowledge of the infant's iron status. The mean increase in score in the test-retest for the 10 normals was 7.2; for those with iron depletion (8) it was 3.7 while the 15 infants with iron deficiency had a mean increase of 21 points ($p<0.005$). This study demonstrates that iron deficiency with biochemical (FEP) or cellular alterations (MCV) produces alterations in behavior that are independent of anemia and are rapidly reversible with iron therapy.

847 THE SYNERGISTIC EFFECT OF MICROWAVES, HYPERTHERMIA AND HYDROCORTISONE SUCCINATE ON L1210 LEUKEMIC CELLS Mark J. Ottenbreit, Susumu Inoue, James C. Lin and Ward D. Peterson Jr., Wayne State Univ., Dept. of Pediatrics, Detroit, MI

Mouse Leukemia L1210 is an extensively used model for cancer chemotherapy studies. An unique microwave irradiation exposure system was used to assay the effects of hyperthermia, microwave irradiation (MWR), and hydrocortisone succinate (HCS) on L1210 cells ability to form colonies in a methylcellulose culture system. MWR was given at 2450 MHz frequency with 1000 mW/cm^2 power output for 30 min. L1210 cells were suspended in α medium and then exposed as follows: Sham at 37°C and 42°C; MWR at 37°C and 42°C. All 4 exposure sets were then plated at a cell concentration of 5,000 cells per plate either with no drug, or with 10^{-5}M, 10^{-6}M, 10^{-7}M or 10^{-8}M HCS per plate. At day 8 of culture the colony number was scored in each of the plates. Pre-exposure to MWR, hyperthermia or combination of the two had no effect on the cell's ability to form colonies. HCS when added to culture plates inhibited the colony formation in a dose-dependent fashion. However, no further inhibition was observed by pre-exposing cells to MWR or hyperthermia alone. A significant inhibition (50%) in the colony formation at each HCS concentration was observed by pre-exposing the cells to both MWR and hyperthermia proceeding culture in HCS. We conclude that MWR and hyperthermia when employed together interact with HCS in a synergistic fashion to inhibit growth of L1210 cells.