944 ABNORMALITIES IN THE ALTERNATIVE PATHWAY OF COMPLEMENT IN PSORIASIS. Wayne M. Marley, Patricia W. Belew, E. William Rosenberg, Ann E. Stitzel and Roger E. Spitzer.

Univ. of Tennessee Center for the Health Sciences, Departments of Medicine and Pathology, Memphis, TN and SUNY, Upstate Medical Center, Department of Pediatrics, Syracuse, NY. The complement system of twenty-four patients with psoriasis was studied in detail. Quantitation of classical pathway components (C4, C3, C3-C9) and the ability of the patient's serum to support lysis of antibody-coated target cells and C3-C9 consumption after incubation with a preformed immune precipitate were either normal or elevated. Alternative pathway evaluation involved quantitation of serum levels of factor B and properdin by radial immunodiffusion; in addition, the ability of the patient's serum to support C3-C9 activation with zymosan and cobra venom factor as well as lysis of unsensitized rabbit erythrocytes was also ascertained. Rabbit CH50 titers and factor B levels were normal or elevated in 23/24 and 24/24 patients respectively. Abnormally low properdin levels more than 2 standard deviations below the normal mean were seen in 14/24 patients. Mean C3-C9 consumption after zymosan or cobra venom factor incubation was significantly less than normal ($p < .001$ and 0.025); in 11/24 sera incubated with zymosan, the extent of C3-C9 activation was more than 2 S.D. below the normal mean. Thus, abnormalities of the complement system are clearly present in patients with psoriasis and seem limited to the alternative pathway. Further, these abnormalities suggest that this pathway may be involved in the genesis of this disorder.

945 ONTOGENY OF PHAGOCYTE MOVEMENT IN THE OVINE. Michael E. Miller, Rukmani Raghunathan, Judy Faust. Harbor-UCLA Medical Center, Dept. Peds, Torrance, California.

Movement of phagocytic cells constitutes a major host defense mechanism. Previous studies from this and other laboratories have demonstrated diminished movement of neonatal PMNs and MNLs. Nothing, however, is known of the ontogeny of PMN and MNL movement. We now report such studies in the ovine. Fetuses ranging in age from 120 to 150 days (term) were delivered of date bred ewes by Caesarean section. PMNs were obtained from venous blood and chemotaxis was measured by a modified Boyden technique, utilizing 3 μ cellulose acetate filters. Endotoxin activated pooled normal sheep plasma was the chemoattractant. Pulmonary alveolar macrophages (PAM) were obtained from the first 10-12 cc of tracheobronchial aspirate. PAM were suspended in McCoy's medium to a final concentration of 1.5×10^6 /ml. Chemotaxis was studied by a modification of the method of Snyderman utilizing 8 μ nuclepore filters. The data showed: 1) Regardless of age at delivery, PMNs and PAMs failed to show any chemotaxis. This correlated with marked neutropenia in the first blood sample obtained. Bone marrow examination, by contrast, showed normal granulocyte stores. Maternal ovine control PMNs and PAMs showed normal migration; 2) Following delivery, lamb PMNs developed chemotactic responses within several days. This correlated with rise in the peripheral neutrophil count to normal adult sheep levels. The absence of chemotactic response in utero was entirely unexpected and suggests a regulatory role on phagocyte function occurring during gestation and the birth process.

946 ANTIBODY DEPENDENT CELL MEDIATED CYTOTOXICITY (ADCC) IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) AND JUVENILE DERMATOMYOSITIS (JDMS) AND THE EFFECT OF *IN VITRO* PREDNISOLONE. M.L. Miller and L.M. Pachman. The Children's Memorial Hospital, Chicago.

Children with clinically inactive SLE and JDMS may have abnormal immune function reflecting underlying defects. ADCC, mediated by Fc receptor bearing cells, may play a role in these diseases by eliminating autoantibody coated cells; it is abnormal in some adult SLE patients. ADCC and its inhibition by prednisolone have not been studied in children with SLE or JDMS. We have shown that ADCC is inhibited by *in vitro* prednisolone in healthy adults; the purpose of this study is to determine if ADCC mediated by peripheral mononuclear cells from children with SLE and JDMS is inhibited by prednisolone. Chang cells labelled with 51-chromium were used as targets. Neither 100 nor 50 μ g/dl prednisolone significantly decreased ADCC (72.0 ± 18.8 at 64:1 effector:target (E:T) ratio). 6 of these children had clinically inactive disease. 6 SLE patients studied showed a correlation of ADCC with total hemolytic complement ($r=.62$). There was no significant decrease in ADCC when prednisolone was added to cells from 4 children with DMS (70.3 ± 14.3 at 64:1 E:T), only 1 of whom had active disease. It is concluded that *in vitro* prednisolone inhibits ADCC from normal adults but not 7 of 9 children with SLE or 4 children with DMS. It is speculated that lack of ADCC response to *in vitro* prednisolone may reflect altered cell subpopulations in children with inactive and active SLE and JDMS.

947 THE EFFECT OF SPECIFIC ANTISERA ON VIRUS-INITIATED NEUTROPHIL CHEMILUMINESCENCE. E.L. Mills, P.G. Quie, Univ. of Minnesota School of Medicine, Minneapolis, and J. Verhoef, University of Utrecht, Utrecht, The Netherlands.

Host resistance to influenza A virus (INF-V) correlates with the titre of serum IgG hemagglutination inhibition (HI) antibody but the mechanisms responsible for protection in the presence or absence of HI antibody are not clear. Human neutrophils (PMN) incubated with live INF-V consume oxygen, generate superoxide and produce chemiluminescence (CL) in the absence of serum. PMN exposed to virus incubated in 10% heated pre-immune human sera (PRE sera), in heated post immune sera (POST sera) or in buffer generated comparable peak CL. When PMN were desialylated by pretreatment with neuraminidase to increase the number of available PMN Fc receptors and the cells incubated with virus opsonized in PRE-serum, no CL was emitted; When desialylated PMN were incubated with virus opsonized in POST-serum, peak CL was 2 - 3 fold above control PMN. When PMN Fc receptors were blocked by pretreating the cells with immune complexes and the cells exposed to virus opsonized in PRE-sera, peak CL was equal to that of control PMN; in contrast, these PMN exposed to virus opsonized in POST sera emitted very little CL. Electronmicrographs of PMN incubated with unopsonized virus for 20 min demonstrate intracellular virus particles. Thus, in the absence of specific antibody, INF-V enters the PMN and stimulates CL via PMN sialic acid-associated receptors; in the presence of antibody, INF-V stimulates via Fc receptors. These data suggest a previously unrecognized role for PMN-viral interaction in INF-V infections.

948 GENERATION OF CONCAVALIN A (Con-A)-INDUCIBLE SUPPRESSIVE FACTORS IN ACUTE LYMPHOCYTIC LEUKEMIA (ALL). Nalini Mukhopadhyay and Donald J. Fernbach (Intro. by Kenneth A. Starling). Department of Pediatrics, Baylor College of Medicine, Houston.

Leukemic blasts (LB) obtained at diagnosis from 6 non-T, non-B, and 2 T-cell patients with ALL were studied for their ability to suppress or enhance the proliferative capacity of normal allogeneic peripheral blood lymphocytes (PBL). LB or control lymphocytes (Ly), preincubated at 37°C for 48 hrs with 100 μ g/ml Con-A (Ca) or without Con-A (Co), were treated with mitomycin-C. One hundred thousand Ca- or Co-treated LB or Ly were added to 1×10^5 PBL and cultured with or without supernatants of pre-incubated cultures in the presence of phytohemagglutinin, Con-A, and pokeweed mitogen. The response of PBL in the presence of supernatants from LB or Ly was markedly suppressed for all stimulations ($P < .001$). However, in the absence of supernatants, 2 patients with non-T, non-B ALL and 1 T-cell ALL suppressed the PBL response for all mitogens (% suppression range 10-73.9; control range 19.3-81.1). One T cell and 4 non-T, non-B ALL showed enhancement (range 11.6-232.0). We conclude that T-cell factors are bifunctional molecules recognizing the stimulant by one end, selecting the cell type by the other end, leading to suppression or augmentation with their restriction specificity.

949 INABILITY OF CORD BLOOD B CELLS TO DIFFERENTIATE INTO PLASMA CELLS: SUPPRESSION VS CELLULAR DEFECTS. K.R. McIntosh and J.A. Beilanti. Georgetown Univ. Sch. Med., Wash., D.C.

Cord blood mononuclear cells fail to differentiate into plasma cells when stimulated by pokeweed mitogen (0% cord blood plasma cells vs 13.5% plasma cells in adults). In order to distinguish between an intrinsic defect in cord blood B cells and active suppression of B cell differentiation, co-cultures of cord blood lymphocyte subpopulations and adult peripheral blood mononuclear cells were performed. The following cord blood populations were found to be suppressive (percent suppression in parentheses): Unfractionated ficoll-hypaque separated cells (68%), T cells (87%), T γ cells (43%), T non-gamma cells (97%), T cell depleted non-adherent cells (39%). Of the non-T, non-adherent cells, only those expressing the Fc receptor for IgG suppress (46%). PWM-stimulated co-cultures of adult helper T cells and cord blood B cells have failed to produce plasma cells in our system, even when cells expressing Fc receptors for IgG have been removed from the cord blood B cell fraction. Therefore, it appears that the inability of cord blood B cells to differentiate into plasma cells is due to both an intrinsic cellular defect and active suppression mediated by various cell populations. NIH Grant 5 T32 HD07185-02.