972 ABNORMAL IMMUNE RESPONSES IN HOMOSEXUAL MEN WITH CHRONIC GENERALIZED LYMPHADENOPATHY. Anne K. Junker, Hans D. Ochs, Ann C. Collier, Hunter H. Handsfield, Gerald Schiffman, Ralph J. Wedgwood. Univ of Washington, Depts of Peds and Med, Seattle; Dept of Micro and Immunol, SUNY Downstate Med Ctr, Brooklyn.

Chronic generalized lymphadenopathy (CGL) in homosexual men may be a prodrome of acquired immunodeficiency syndrome (AIDS). Depressed cell mediated immunity including low T-helper:T-suppressor ratios are commonly detected. To evaluate humoral immune function we immunized 9 CGL patients with pneumococcal polysaccharide (PPS) vaccine and the T-dependent neoantigen bacteriophage ØX 174 (ØX 174). Antibody responses to PPS were abnormal in 7 of 8 patients immunized; baseline antibody titers were absent or low, post immunization titers rose minimally and titers declined rapidly. Three patients had abnormal primary and secondary and five patients had abnormal secondary responses to ØX 174 including failure to switch from IgM to IgG. One patient with a very low primary response to ØX 174 developed pneumocystis carinii pneumonia 5 months later. Peripheral blood mononuclear cells (PBM) of patients with abnormal antibody responses in vivo showed reduced antigen induced antibody synthesis in vitro. The direct hemolytic plaque assay was abnormal in all patients: 2 had excessive suppression; 1 lacked T help; 5 showed increased antibody synthesis suggesting a state of immune activation which might explain the reported hyperimmunoglobulinemia.

These studies indicate that homosexual males with CGL have both cellular and humoral immune deficiency, predisposing such persons to the opportunistic infections which characterize AIDS.

● 973 DECREASED Ia-AG ON HUMAN NEONATAL MACROPHAGES (A MARKER OF MATURATION?) S. Kalayanarooj*, Y. Bryson; UCLA Sch. Med., Los Angeles, Ca.

Neonates are susceptible to severe viral and bacterial infections which may be due to immaturity of their immune system. Macrophages are one of the critical elements in host defense and are important for many specific immune processes including gamma interferon production. Studies in our lab have shown that a functional immature neonatal macrophage is primarily responsible for the impaired production of PHA-induced gamma IFN by mononuclear cells of neonates (age 1-7 days). This defect improves with time of in vitro cultivation (>3 wks) and also is age related in vivo (>2½ mos). Neonatal mice have been shown to have a defect in antigen presenting function which is correlated with a decreased number of Ia AG bearing macros. To investigate factors associated with this macrophage defect in newborns, we studied adherent cells from Ficoll-Hypaque separated cord and adult cells in vitro for evidence of Ia AG by immunoperoxidase and monoclonal AB assay and morphological changes by scanning electron microscopy. We found that only 26% of 7 day old neonatal macros expressed Ia AG vs 90% in comparable adult macros. No gross morphological differences were seen by SEM of adult or neonatal macros at 5,6,7,8 days of age. However we found 2 populations of neonatal macros at 7 days with 10-15% large macros (diameter ~40u) compared to 20-30u adult macros. In an attempt to activate neonatal macros we pretreated 6 day old neonatal macros for 8 hrs with Meloy interferon (.1, 1 and 10 IU) added neonatal T cells ($2x10^5$/ml) and stimulated with PHA and found no enhancement of IFN production in 48 hr supernates. Further investigation into the nature and correction of this impaired function may suggest future therapeutic possibilities.

974 HYPOGAMMAGLOBULINEMIA IN CHILDREN WITH CHRONIC GRAFT VERSUS HOST DISEASE (GVHD) AFTER BONE MARROW TRANSPLANTATION (BMT). Naynesh Kamani, Charles S. August and Steven D. Douglas, Univ. Pa. Sch. Med., Children's Hospital of Philadelphia, Dept. Pediatrics.

Four boys aged 5-16 years developed acute GVHD following allogeneic BMT. All were treated with high-dose intravenous methylprednisolone; they then developed chronic GVHD and were given Prednisone 2 mg/kg/day. Three patients also received Azathioprine 2 mg/kg/day. After 2-3 months of therapy, immunoglobulin (Ig) levels had fallen dramatically (Table). The percentages of E and EAC rosetting cells, surface Ig bearing cells and lymphocyte mitogen responses were normal. All 4 received replacement therapy with gammaglobulin or fresh frozen plasma. The index patient(#1) was discovered after having multiple pyogenic infections. Patient #4 died of pulmonary aspergillosis 19 months post-BMT. After Prednisone was discontinued in patients 1, 2, and 3, Ig levels promptly returned to normal (#1,#3) or increased (#2). Since replacement therapy was instituted, the surviving three have remained free of infection. Conclusion: Although chronic GVHD is often characterized by elevated Ig levels, reversible hypogammaglobulinemia can occur during steroid therapy and may be related to increased susceptibility to pyogenic infection.

			Ig Level-Nadir (mg/dl)		
PT#	Age	1° Disease	IgG	A	M
1	7	ALL	65	14	48
2	6	ALL	144	5	5
3	5	ALL	60	5	5
4	16	CML	80	11	38

975 IMMUNE DYSFUNCTION IN CHILDREN WITH ACQUIRED IMMUNE DEFICIENCY SYNDROME. Prakash Kaur, Saveeta Pahwa, Franklin Desposito, Thomas Denny, Rajendra Singh, James Oleske. (Spon. by Franklin C. Behrle). University of Medicine and Dentistry of New Jersey. Department of Pediatrics. Newark, New Jersey.

Immunological studies were performed on 4 children age 2 to 2½ years with Acquired Immune Deficiency Syndrome (AIDS) born into households in which at least one patient had AIDS or AIDS prodrome. Total T cells were quantitatively normal but the ratio T4 (helper/inducer) to T8 (suppressor/cytotoxic) subsets was inverted (<1.0 N = >1.8). Proliferative responses of the peripheral blood lymphocytes to T cell mitogens PHA and Con A were markedly reduced in all children. Proportions of B cells were normal or increased and all patients had excessive levels of serum immunoglobulins (Ig). Ig production by B cells was evaluated in a reverse hemolytic plaque assay. Although spontaneous secretion of Ig by peripheral blood B cells were markedly increased, after polyclonal activation with pokeweed mitogen Ig secretions were greatly diminished. In allogenic co-culture experiments, admixtures of patient cells to normal cells resulted in suppression of Ig production. This suppression or activity was abolished following irradiation of patient cells with 1500 rads. The above studies demonstrate that children with AIDS manifest abnormalities of B cell activation and immunoregulation.

● 976 IgD - A MUCOSAL IMMUNOGLOBULIN? Margaret A. Keller, Douglas C. Heiner, Aileen S. Myers, Diane M. Reisinger UCLA School of Medicine, Harbor-UCLA Medical Center, Department of Pediatrics, Torrance, California.

Simultaneous colostrum(C) and plasma samples(P) from 17 women, two to five days postpartum were assayed for IgD, albumin and IgG, the latter two by radial immunodiffusion and IgD by radioimmunoassay. Colostrum samples were assayed for IgD using a serum standard curve and a curve derived by adding known quantities of IgD to an IgD deficient colostrum. The mean colostrum/plasma ratio for IgD[0.055±0.015] exceeded the albumin C/P ratio [0.020±0.002] and the IgG C/P ratio[0.015±0.003]. For 14 of the 17 patients, the individual C/P ratio for IgD exceeded the C/P ratio for albumin and for one subject the IgD C/P ratio was greater than 10 times the albumin ratio. The data for IgG differed in that the total IgG C/P ratio was lower than the albumin C/P ratio for 16 of the 17 subjects studied. For all subjects except an IgD deficient subject, the C/P ratio for IgD exceeded that for IgG. Correlation coefficients were calculated using logarithmically transformed data. For albumin vs. IgG in colostum, r=0.865, p=0.001. This differed from the correlation for albumin vs. IgD, r=0.489, p=0.046 or IgD vs. IgG, r=0.556, p=0.020. These data indicate that IgD is enhanced in colostrum in comparison with IgG and suggest that the mechanism of entry of IgD into milk differs from that of IgG or albumin. The data support our previous finding of enhanced specific IgD antibody in colostrum and suggest a role for IgD in the secretory immune system.

977 THE CONSEQUENCES OF INFLUENZA VACCINE ON HUMAN NEUTROPHIL FUNCTION. Richard J. Kemmy, Roger H. Kobayashi, and Roberta J. White. University of Nebraska Medical Center, Omaha, Departments of Microbiology and Pediatrics.

Although the neutrophil (PMN) has been viewed as an antibacterial effector cell, there is recent evidence which indicates that these cells may interact with various viruses. The effect of influenza virus antigens on cellular functions was investigated by adherence, chemotaxis, and chemiluminescence (CL) assays. The purpose was to determine whether the administration of influenza vaccine depressed PMN function in vivo, and secondly, whether in vitro incubation of the vaccine with PMNs altered their function. The assays were performed before and after vaccination using cells obtained from normal adults, patients with cystic fibrosis (CF), and those with asthma. This study demonstrated that the neutrophil CL response from controls and asthma patients, but not CF patients, was temporarily depressed after vaccination. PMN adherence and chemotaxis were unaffected over the same time period in all three groups. However, direct incubation of the cells with viral antigens consistently depressed PMN functions as assessed by the three assays. These results imply that PMN functions may be suppressed following influenza infection and this might predispose the host to secondary bacterial infections. However, PMNs from patients with asthma or CF were not impaired to a greater degree than controls. This infers that other factors are involved which might enhance the opportunity for complications following influenza infection in patients with chronic lung disease.