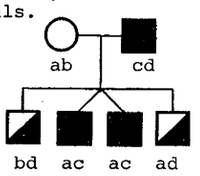
842 OCCURENCE OF ARTHRITIS FOLLOWING ERYTHEMA INFECTIOSUM(EI) IN HLA-B27+ MEMBERS OF A FAMILY. Peter LoGalbo, Gary Solomon, Robert Winchester (Spon. by Walter Henley). The Mount Sinai and N.Y.U. Schools of Medicine, Hosp. for Joint Disease, Dept. of Rheumatic Diseases, New York, N.Y.

We have identified a family in which typical EI developed in an epidemic setting in four male siblings and their father. Three individuals who were HLA-B27+ developed arthritis (Figure). Two nine year old twins developed joint pains and morning stiffness five days after the onset of rash and fever, and one of these has continued to have small joint arthritis of his hands and feet. Their father also became febrile and subsequently developed low back pain and joint stiffness. The ANA and RF were negative. Two other children with EI who were HLA-B27- had no arthritis. Human parvovirus has been shown to be the likely etiologic agent of EI. Our observations suggest that one form of arthropathy in EI has features in common with reactive arthritis, and occurs in genetically susceptible individuals.



■ = EI + arthritis

◪ = EI

a = A2	Cw3	Bw62	DR2
b = A1	Cw7	B8	DR3
c = A30	Cw2	B27	-
d = A2	CwX	B7	DR2

843 **SPECIFIC IgG ANTI-TETANUS TOXOID ANTIBODY SYNTHESIS BY HUMAN BONE MARROW MONONUCLEAR CELLS.** **Lawrence G. Lum, Jan E. Noges, Margaret C. Seigneuret, and Paul J. Martin. Medical College of Wisconsin, Milwaukee, WI, and Fred Hutchinson Cancer Research Center, Seattle, WA.**

Human marrow mononuclear (marrow) cells from normal donors who had not recently received tetanus toxoid (TT) booster immunizations were examined for their ability to produce specific IgG anti-TT antibody (anti-TT), polyclonal IgG and polyclonal IgM with and without in vitro tetanus toxoid (TT) stimulation. ELISA assays were used to measure specific and polyclonal IgG and IgM production. Marrow B cells were found to secrete anti-TT and nonspecific IgG and IgM spontaneously for up to 21 days of culture. Stimulation of marrow cultures with TT resulted in variable modulation of anti-TT production, yet had no detectable effect on polyclonal IgG or IgM production. Depletion of T cells from marrow caused a marked reduction of spontaneous and TT-induced anti-TT synthesis. Repletion of T cell-depleted marrow with autologous peripheral blood T cells and stimulation with TT were required to reconstitute anti-TT production. The results show that: 1) steady state marrow contains partially activated or differentiated marrow B cells capable of producing in vitro anti-TT; 2) spontaneous anti-TT synthesis by marrow B cells is regulated, in part, by T cells. Since antigen and T cells can alter the function of marrow B cells, these findings suggest that antigen-specific immune memory directed at pathogens or tumor antigens may be manipulable before, during, or after bone marrow transplantation.

844 EVIDENCE FOR MODULATION OF GRAFT-VS-HOST DISEASE BY INTRAUTERINE ENGRAFTED MATERNAL T LYMPHOCYTES. Robert J. Mamlok, Randall M. Goldblum, Smita Vaidya, and Armond S. Goldman. University of Texas Medical Branch, Departments of Pediatrics, Human Biological Chemistry & Genetics and Pathology, Galveston, TX.

A four month old dizygotic triplet with pseudomonas sepsis and disseminated intravascular coagulopathy necessitating platelet tranfusions was subsequently found to have severe combined immunodeficiency (SCID) with thymic dysplasia and mild cutaneous graft-vs-host disease (GvHD). T lymphocytes from the mother and both platelet donors but not from the triplets were identified in the patient's blood by HLA typing. Non-T lymphocytes displayed the same maternal and paternal haplotypes as the other triplet siblings and absorption of lymphocytotoxic HLA antibodies by the patient's platelets confirmed the patient's HLA genotype.

GvHD resolved during ablative therapy with anti-thymocyte globulin and cyclophosphamide and did not recur following bone marrow transplantation (BMT) from an HLA identical triplet. One month after BMT, T lymphocytes from one platelet donor, but not from the mother, were still found in the patient's blood.

Since GvHD from non-irradiated blood transfusions in SCID patients is fatal without treatment, we hypothesize that intrauterine engraftment of maternal cells and/or mild GvH reaction induced by these cells suppressed the GvHD from the platelet donor's lymphocytes. An exacerbation of the GvHD after maternal cells disappeared may have been prevented by immune function from the BMT. Thus, intrauterine engraftment of maternal T lymphocytes, which usually causes limited GvHD, may modulate the GvHD due to postnatally transplanted T lymphocyes.

845 NEUTROPENIA AND DEFECTIVE CHEMOTAXIS ASSOCIATED WITH BINUCLEAR, TETRAPLOID MYELOID-MONOCYTIC LEUKOCYTES. Robert J. Mamlok, Harinder S. Juneja, Frederick B. Elder, Mary E. Haggard, Frank C. Schmalstieg, and Armond S. Goldman. The University of Texas Medical Branch, Departments of Pediatrics, Internal Medicine and Human Biological Chemistry and Genetics, Galveston, TX.

A 13 year-old male presented with recurrent cellulitis and osteomyelitis and persistent neutropenia (blood neutrophils count of 100/mm^3). The bone marrow was normal except for two nuclei in the majority of metamyelocytes and bands and in all segmented neutrophils. No binucleated myeloblasts, myelocytes or other cell types were found. Granulocyte-monocyte colonies cultured from bone marrow had single nucleated and binucleated band forms and metamyelocytes, but no binucleated myelocytes or myeloblasts. Cytogenetic studies of bone marrow cells showed that the single nucleated cells were 46XY, while the binuclear cells were predominantly 92XXYY.

Chemotaxis of myeloid bone marrow cells was examined by a subagarose method. The patient's cells showed no directed movement towards zymogan activated serum (ZAS) or a synthetic peptide, F-Met-Phe, whereas directed and random movement of myeloid bone marrow cells from adult controls was detected (F-met-Phe, 13.2 u/mm and 9.5 u/min; ZAS 22 u/min and 11.7 u/min).

These findings suggest that defective egress of binucleated tetraploid neutrophils from the bone marrow resulted in chronic neutropenia. Further studies on the cytoplasmic architecture of these cells may help define a link between the failure of cytoplasmic splitting and the impaired chemotaxis of myeloid cells.

846 Fc RECEPTOR MEDIATED CLEARANCE, IN VIVO, IN PATIENTS WITH RECURRENT PULMONARY INFECTIONS. Evangelia C. Mantzouranis, Fred S. Rosen, Harvey R. Colten; Department of Pediatrics, Harvard Medical School; Department of Pediatrics, Washington University School of Medicine.

Previous studies established defective Fc receptor mediated clearance in patients with systemic lupus erythematosis (SLE), a disorder associated with circulating immune complexes (CIC). The finding of CIC in sera of patients with cystic fibrosis and speculation that progressive CF pulmonary disease may be due to immune complex deposition prompted the present study. Accordingly, we tested Fc mediated clearance in vivo in patients with CF (N=13), other chronic inflammatory lung diseases (N=6), SLE (N=6), hypogammaglobulinemics (N=6) and normals (N=10) with the use of ^{51}Cr labeled autologous erythrocytes sensitized with anti-Rh(D) antibody. CF patients, chronic pulmonary disease patients and hypogammaglobulinemics had accelerated Fc receptor mediated clearance rates (T 1/2 = 19.6 min, 16 min and 12.8 min, respectively) compared to normals (T 1/2 = 27.5 min) and patients with SLE (T 1/2 = 38.6 min). These data suggest that Fc mediated clearance in chronic inflammatory diseases other than SLE reflect enhanced reticuloendothelial cell function and not the depressed clearance function found in diseases thought to be due to immune complex deposition.

†847 PREMATURE & SECRETORY IgA. R. Mead, M.D., S.J. McGeady, M.D., Philadelphia, PA

Newborn infants have little or no secretory IgA (SIgA) at birth. Earlier studies suggest that premature infants do not significantly differ from term babies in their rate of acquisition of SIgA. No study to date has examined SIgA production as a function of gestational age or examined the kinetics of SIgA acquisition in premature infants of various ages and term infants. This study examined the unstimulated saliva in 40 infants including 13 term babies, 15 prematures with gestational ages 31-35 wks, and 12 less than 31 wks. A sensitive double antibody radiommunoassay was used to assay SIgA compared with a human colostrum derived SIgA standard. No baby was fed breast milk and formula preparations were shown not to inhibit the assay. Results of SIgA in mg/dl shown as mean and range:

Gest age	0-6 days	7-21 days	21 days
<31 wks	0 (0.0-0.0)	1.4 (0-4.1)	18.8 (1.2-36)
>31-35 wks	0.3 (0-1.5)	3.1 (1.2-5.2)	23.4 (14-38.2)
>36 wks	1.1 (0-2.7)	2.8 (0-6.3)	18 (13.5-20)

These results indicate that SIgA is absent in some prematures and term infants. The more premature the infant, the more likely is SIgA to be absent very early in life. After the first week of life, however, infants acquire SIgA at a comparable rate regardless of gestational age. The increased susceptibility to infection found in premature infants is most likely due to multiple factors, but a significant lag in SIgA production does not appear to be operative.