

1021 REDUCTION OF CORD BLOOD POLYMORPHONUCLEAR (PMN) LEUKOCYTE CHEMOTAXIS BY INDOMETHACIN. Jesse D. Roberts, Jr., Alfredo Jalowayski and T. Allen Merritt. University of California, San Diego, School of Medicine, Department of Pediatrics, San Diego.

Protracted exposure to high oxygen tensions has been associated in preterm neonates with egress of leukocytes and alveolar macrophages into pulmonary interstitium and effluent obtained by bronchoalveolar lavage. Pulmonary effluent serine elastase concentrations are increased in infants exposed to $F_{iO_2} > .4$. Serum indomethacin concentrations of 10^{-5} to 10^{-7} molar have been associated with closure of the patent ductus arteriosus. We hypothesized that indomethacin, a potent anti-inflammatory agent, might alter leukocyte chemotaxis in infants. Using modified Boyden chambers human term cord blood PMN leukocyte chemotaxis was altered ($F=6.68, p=0.025$) with linear attenuation ($r=-0.77$) to zymogen activated human serum. We speculate that indomethacin treatment of neonates may reduce oxygen induced inflammation through reducing leukocyte induced cytotoxic parenchymal lung damage and airways inflammation in addition to closing the patent ductus arteriosus. (HD 16292)

1022 A LYMPHOBLASTOID B-CELL LINE (LBL) DERIVED FROM A 12-YEAR-OLD BOY WITH SEVERE COMBINED IMMUNODEFICIENCY (SCID). Howard M. Rosenblatt, Jerome Ritz, Nirmi Parikh, Dorothy Lewis, and William T. Shearer, Baylor College of Medicine, Department of Pediatrics, Houston, Texas.

We describe the phenotypic and functional characteristics of an LBL derived from the bone marrow of a patient who died with an Epstein-Barr virus (EBV) related lymphoreticular malignancy 4 months after bone marrow transplantation for SCID. This LBL, designated DV-1, arose spontaneously 2-3 weeks after transplantation and while initially dependent on autologous fibroblasts, subsequently has been maintained in standard suspension cultures. The B-cell origin is suggested by the absence of T-cell markers and the presence of DR and BI antigen on 80-90%, surface IgM on 20-30%, and IgD on 20-30% of the cells. The predominant light chain type is κ (72%) with a small percentage of cells expressing λ . The cells show a normal 46XY karyotype and the host's HLA phenotype. Greater than 90% of the cells are positive for EBV nuclear antigen and show the typical appearance and growth characteristics of EBV transformed LBL. Phytohemagglutinin inhibited (12-39%), pokeweed mitogen had no effect on, goat anti- μ chain antiserum stimulated (57%), and staphylococcus aureus Cowan strain A inhibited (20-80%) thymidine incorporation by DV-1 in a dose related manner. These characteristics are similar to those described for other EBV derived LBL's reported in the literature and to an independently derived LBL (LA350) to which we compared DV-1. This line holds useful potential for future functional and morphological studies on B-cell lines derived from immunodeficient patients.

1023 IgA RHEUMATOID FACTOR (RF) IN HENOCH-SCHONLEIN PURPURA (HSP). Frank T. Saulsbury. (Spon. by Robert L. Chevalier) University of Virginia Medical Center, Department of Pediatrics, Charlottesville, VA

Recent evidence indicates that circulating immune complexes play a role in the pathogenesis of HSP. The elaboration of RF appears to be an important host response to circulating immune complexes. Accordingly, sera from 23 children with acute HSP were tested for RF of various isotypes using a diffusion-in-gel enzyme-linked immunosorbent assay. Controls consisted of 15 normal children and 10 normal adults. Heat aggregated human or rabbit IgG was used for the test substrate and peroxidase-conjugated F(ab')₂ fragment of goat anti-human IgG, IgA, or IgM served to determine the RF isotype. None of the HSP patients had IgG or IgM RF. Of the controls, one child had IgM RF, and one adult had IgG RF. IgA RF was present in the sera of 12 of 23 HSP patients (52%), in contrast to 1 of 25 controls ($p<0.0005$). When present, IgA RF titers tended to be highest during the acute phase of the illness. Although 64% of HSP patients had increased serum concentrations of IgA, there was no correlation between IgA concentration and the titer of IgA RF ($r=0.25, p>0.10$). Moreover, there was no association between the presence of IgA RF and any of the clinical manifestations of HSP such as nephritis, arthritis, abdominal pain, or gastrointestinal bleeding. These results indicate that a substantial proportion of HSP patients have IgA RF. The role of IgA RF in the formation of circulating immune complexes in HSP remains to be elucidated.

1024 PENICILLIN HYPERSENSITIVITY IN CHILDREN GIVEN PENICILLIN OR PLACEBO AT BIRTH. V. Schauf, P. Nell, K. Ghaey, M. Tolpin, M. Rathi, B. Kontelas, N.F. Adkinson Jr. Depts of Pediatrics, U. of Ill., Christ, U. of Chicago Hospitals, Dept. of Medicine, Johns Hopkins Univ. School of Medicine, Chicago and Baltimore.

Penicillins are frequently used in neonates; however, the potential for inducing hypersensitivity is unknown. Subjects participated in a placebo-controlled trial of parenteral penicillin prophylaxis of neonatal streptococcal disease 2 years earlier. Parents were interviewed to determine allergic history and antibiotic use. Sera from the 2 year olds were tested for IgG and/or IgE antibody to benzylpenicilloyl antigen (BPO) by radioimmuno-precipitation and RAST, respectively. Half the infants in each group had received a β -lactam antibiotic since the injection at delivery. Possible β -lactam allergic reactions occurred in approximately 10% of each group. Food allergies were reported more often in placebo recipients (23 of 220 vs. 14 of 200, $p=.02$). IgG-BPO was found in 3 of 61 penicillin and 4 of 46 placebo recipients who later received β -lactam therapy. None of the IgG-BPO positive sera contained IgE-BPO. Additional, randomly selected IgG-BPO negative sera were also negative for IgE-BPO. IgG-BPO was present in only one child with no known later β -lactam exposure. Early exposure to a single dose of parenteral penicillin does not appear to predispose to subsequent penicillin allergy in the first 2 years of life.

† 1025 CONCAVALIN A BINDING PROTEINS OF THE HUMAN POLYMORPHONUCLEAR LEUKOCYTE PLASMA MEMBRANE. Frank C. Schmalstieg, Helen E. Rudloff, Donald C. Anderson, and Timothy A. Springer. The University of Texas Medical Branch, Galveston, TX, Baylor College of Medicine, Houston, TX, and Harvard Medical School, Boston, MA.

The plant lectin, Con A, has been used extensively as a membrane probe in the study of blood leukocytes including PMNL. At least twenty proteins (¹²⁵I-labeled human PMNL) with this capability are visible after separation on SDS-PAGE and subsequent autoradiography. In contrast to these findings, Williams and Becker recently described a 140 Kd membrane glycoprotein as the major Con A binding protein in rabbit PMNL. The observation of an accompanying 95 Kd protein prompted us to examine the existence of a similar glycoprotein complex in human PMNL and to determine whether the 140 Kd and 95 Kd proteins were related. Human PMNL membrane glycoproteins were allowed to bind to Con A and released with α -methyl-D-mannoside. The released proteins were immunoprecipitated with a variety of membrane protein specific antibodies. The 140 Kd glycoprotein was identical to the alpha subunit of Mac-1 (iC3b complement receptor). Furthermore, the 95 Kd protein was identical to the beta subunit of the Mac-1 molecule. NaB³H labeling of the PMNL surface was undertaken to better delineate the position of Mac-1 in the membrane. This experiment suggested the carbohydrate portion of the molecule was clearly exposed. Although Mac-1 may be an important Con A binding protein, other plasma membrane proteins contribute to this binding since a patient with Mac-1 deficiency was able to bind and cap Con A in a qualitatively normal fashion.

1026 HISTAMINE CONCENTRATION IN MIDDLE EAR EFFUSIONS David P. Skoner, William J. Doyle, Ernest P. Tanner and Philip Fireman. Children's Hospital of Pittsburgh, Pittsburgh, Pennsylvania

The presence of a number of inflammatory mediators in middle ear effusions collected from children with otitis media has recently been detected. In one study, histamine levels in middle ear effusions were assayed using a modified fluorometric technique and a range of 0-3650 ng histamine/ml effusion was reported (Berger et al, Rec. Adv. OME, 1984). Since this amine is a biologically active vasodilator affecting mucosal permeability, its presence may have profound consequences to the pathogenesis and treatment of the disease. In an effort to confirm these findings, 22 effusions collected from 17 children aged .7-8.7 years with chronic non-suppurative otitis media were assayed for the presence of histamine using a modified single isotopic enzymatic assay. Histamine concentrations in these effusions ranged from 1.3 to 112 ng/ml with a median value of 21 ng/ml. As with the previous study, no correlation was found between the concentration of histamine and the age or sex of the child, effusion culture or hemoglobin content. While the concentrations reported in this study are an order of magnitude lower than those previously reported, our data support the hypothesis that histamine may be involved in the production and maintenance of inflammatory reactions associated with otitis media.