908 SUPPRESSOR CELL ACTIVITY IN NEONATAL PERIPHERAL BLOOD Anne E. Dyson, Stanley E. Read and Joseph Algrant. The Rockefeller University, New York and SUNY, Upstate Medical Center, Department of Pediatrics, Syracuse, NY.

A technique for assessing suppressor cell activity in peripheral blood of healthy premature neonates was established using ConA induced suppression of the PHA response in vitro. Neonatal lymphocytes were separated in routine fashion and placed in preculture with ConA (1µg/ml). Cells were also precultured without ConA to provide information on spontaneous suppressor cell activity. After 48 hours incubation, cells were collected, washed and irradiated with 3000r. Effect of precultured cells on PHA stimulated blastogenesis was studied by microculture technique using 10^5 inactivated cells and an equal number of fresh, autologous lymphocytes (responder cells). Controls were provided by addition of PHA to responder cells alone. Normal adult cells were used for comparison cultures. Data were expressed as one minus the ratio (x 100) of the observed mean cpm of the precultured plus fresh cell blastogenic response to the observed mean cpm of the neonatal control response. Preculture of neonatal lymphocytes yielded suppression in the majority of samples, 73% (mean 33 ± 24) with and 55% (mean 23 ± 15) without ConA; conversely, stimulation was seen in 18% and 27% respectively. Adult studies showed suppression in 25%, unchanged by ConA, but showed stimulation in 50% (mean 45 ± 35) with and 58% (mean 35 ± 25) without ConA. We conclude that neonatal lymphocytes, as studied in this system, demonstrate a propensity to suppress PHA induced blastogenesis.

● **909** MOLECULAR BASIS OF A GENETIC DEFICIENCY OF THE SECOND COMPLEMENT COMPONENT (C2). L.Peter Einstein, Gabriel Goldberger, F.Sessions Cole, Dieter Bitter-Suermann, Harvey R.Colten. Harvard Medical School, Dept. Pediatrics, Boston.

C2 deficiency is the most prevalent human genetic disorder of the complement proteins. We reported that monocytes from C2 deficient humans failed to secrete functional C2. Availability of a strain of C2 deficient (C2D) guinea pigs permitted a study of the molecular basis of this defect. Production of C2, factor B, and C4 by peritoneal macrophages from normal, heterozygous and homozygous C2D animals was monitored functionally and immunochemically. Homozygous C2D macrophages failed to secrete functional C2. C2 protein with an apparent molecular weight less than normal was found intracellularly. Extracellular C2 antigen secreted by the C2D cells was present primarily as apparent catabolic fragments on SDS-PAGE. Mixtures of normal radiolabeled C2 with conditioned medium from C2D cells suggested that this finding was not due to increased proteolytic activity in the C2D medium. Cells from each animal produced comparable amounts of C4 and factor B, and were similar in morphology, phagocytosis, nonspecific esterase staining, and C3b and Fc receptor function. These data indicate that C2D guinea pig macrophages synthesize a defective, unstable C2 protein.

910 TRANSIENT LAZY LEUKOCYTE SYNDROME. Senih M. Fikrig, Yasmin Bhassin, Juana C. Phillips, Pulluru S. Rao. S.U.N.Y., Downstate Medical Center, Department of Pediatrics, Brooklyn, New York.

A Puerto Rican girl with history of impetigo at 3 weeks of age was admitted with diagnosis of pneumonia at 5 months of age. Her WBC: 5800 with Poly:1 Eo:1 L:79 M:19%. The bone marrow was normal with increased number of myeloid cells, without maturation arrest. The subcutaneous epinephrine as well as intravenous hydrocortisone injections did not increase the total WBC's as well as percentage of PMNs. The PMN chemotaxis and random motility was grossly abnormal. For the next 10 months the WBC and % of PMN did not change, and she had a number of URI's and gingivitis, then she was lost to follow up. At 3 years of age when she was seen again with WBC:7150 Poly:45 Eo:1 L:48 M:6%. The epinephrine stimulation showed a 116% increase in absolute PMN count at 5' and hydrocortisone injection increased the PMN count 4560 above the baseline at 3 hours. PMN chemotaxis was normal.

	Random Motility(u)	Zymosan Chemotaxis(u)
Control	183	115
Patient (5mos.)	47	61
Control	97	119
Patient(3yrs.)	60	110

911 IgE AND IgG ANTI-POLLEN ANTIBODIES IN DOGS AFTER VIRUS INFECTIONS. Oscar L. Frick and Dale Brooks, University of California, San Francisco and Davis, Departments of Pediatrics and Veterinary Medicine, San Francisco and Davis, CA.

In infants born into allergic families, we reported a co-incidental association between viral infections and onset of allergy. Dogs are the only animals with frequent naturally occurring allergies. A colony of allergic dogs was established by screening 220 hunting dogs for high titers of IgE antibodies to grass and weed pollens. Six animals (4 females, 2 males) that had 10^{5+} IgE-PCA titer were bred and their offspring also inbred. At 4,8,12 weeks of age, puppies received their live attenuated distemper virus vaccine and 2 and 9 days later, 10 µg of 5% mixed grass and ragweed pollen extracts in alum were injected s.c. The vaccinated puppies made significantly more IgE antibodies to pollens than did their unvaccinated littermates. In May 1980, a parvovirus diarrhea epidemic struck the colony affecting all the dogs; 2 litters of young puppies died. During the diarrhea, all dogs were given 10 µg 5% pollen extracts in alum. IgE and IgG antibodies to pollens were measured by RAST using rabbit anti-canine ε and γ-chain sera, respectively. In 6 litters (23 dogs), there was universally, a brisk 2 to 5-fold rise in IgE antibodies to one or both pollens within the next 6 weeks; in another 9 dogs the rise occurred in 3 months. In 8/32 dogs, IgG antibodies rose 2-fold. All infected dogs had a 4-fold$^+$ rise in parvovirus antibodies. The high IgE anti-pollen antibodies have been maintained in 80%. Eczematous rashes developed in 10%. We suggest that certain virus infections cause "allergic breakthrough" by depressing T_S-E cells for IgE antibody production.

912 A MONKEY MODEL FOR IgE MEDIATED OTITIS MEDIA WITH EFFUSION. Roger Friedman, William Doyle, James Fagin, Charles Bluestone and Philip Fireman. Univ. of Pittsburgh School of Medicine, Dept. of Ped., Pittsburgh, PA.

An allergic (IgE) pathogenesis for otitis media with effusion (OME) has been suggested but not confirmed. Monkeys have been used previously to study allergic reactions by passive sensitization with serum from allergic patients. A method of insufflation of the eustachian tube (politzerization) by ragweed pollen grains was confirmed by recovery of pollen from the middle ear. Three normal juvenile monkeys were passively sensitized I.V. with human sera (high RAST anti-ragweed IgE titer), 40 cc/kg over 2 to 3 days and demonstrated positive skin tests and serum IgE antibodies to ragweed. Antigen challenge by politzerization with 0.1 gm ragweed pollen for 3 to 5 days induced OME as measured by tympanometry and confirmed by tympanocentesis in all animals. Similar studies in infant monkeys induced middle ear effusion (MEE) in 2 of 3 antigen challenges. In a single study without tympanocentesis, the OME resolved spontaneously in 5 days. A monkey given non-allergic serum did not develop OME after ragweed antigen challenge via politzerization as described above. Another juvenile monkey passively sensitized with allergic serum was challenged by nasopharyngeal insufflation without eustachian tube entry of the ragweed pollen and no OME developed. Studies thus far of the MEE obtained by tympanocentesis have shown no bacterial organisms by gram stain or culture. A monkey model of IgE mediated MEE has been developed to study the pathogenesis and therapy of OME.

913 IMMUNE COMPLEXES IN SLIPPED CAPITAL FEMORAL EPIPHYSIS. Gene L. France, Raymond T. Morrissy, Daniel J. Marmer, and Russell W. Steele. Univ. of Arkansas for Medical Sciences, Depts. of Pediatrics and Orthopaedics, Little Rock, AR.

Serum, synovial fluid, and synovial biopsy material were examined in 9 patients with slipped capital femoral epiphysis (SCFE), in 7 controls, and in 3 patients with acute toxic synovitis (ATS) for the presence of immune complexes (IC), immunoglobulins, and complement components. The serum was assayed for IC by the Raji cell indirect fluorescent method and by the C1q binding assay (C1q-BA). Total hemolytic complement (CH_{100}), C_3, and C4 were quantitated by radial diffusion, and quantitative immunoglobulins (IgG,A, and M) were measured by laser nephelometry. Biopsies of the synovium and the junction of the femoral neck and articular cartilage were studied by light microscopy and direct immunofluorescence. IC in the synovial fluid were demonstrated in 6 of 7 SCFE patients by Raji cell method and 5 of 7 by C1q-BA, in 3 of 3 ATS patients by Raji cell method and C1q-BA, and in 1 control by Raji cell method, but not by C1q-BA. Fluorescent staining for deposition of immunoglobulins and complement on synovial tissue was negative in all study cases.

All serum immunologic parameters (IgG, IgA, IgM, C_3, C4, and CH_{100}) were normal in patients with SCFE, and IC were not detected in SCFE patients' serum by either the Raji cell method or C1q-BA. However, the serum from 1 of 3 ATS patients had IC by Raji cell method. Immunoglobulins and complement of the synovial fluid were comparable in all 3 groups (SCFE, ATS, and control). This preliminary data suggest that immune complexes may play a pathologic role in SCFE and ATS.