745 QUANTITATIVE IMMUNOSORBENT ASSAY FOR SIgA. J.H.van Bavel, R.M. Goldblum, F.C.Schmalstieg and A.S. Goldman. Depts.Human Biological Chem.& Genetics and Pediatrics, The University of Texas Medical Branch,Galveston,Texas.

Studies of the secretory immune system have been hampered by the lack of an assay to specifically measure SIgA in secretions which also contain serum IgA and free secretory componet (FSC). We have developed an assay for SIgA utilizing commercially available acrylamide beads coated with antibodies against human alpha chains (Immuno-Fluor,Bio-Rad Labs). After incubation with IgA containing solutions, SIgA bound to the beads was labeled with a fluorescein conjugated antibody against secretory component. The fluorescence measured was directly proportional to the added purified SIgA, and was linear over a 10-fold range of concentrations with a sensitivity of approximately 50 ng. The effect of serum IgA was studied using mixtures containing varying proportions of SIgA and serum IgA. Quantitation of SIgA was not affected by serum IgA when these mixtures were assayed in the presence of excess antibody coated beads. Knowing the SIgA concentration, serum IgA levels in a sample could be calculated from the fluorescence developed with an anti-alpha fluorescein conjugate. An immunosorbent assay utilizing antibodies of two different specificities for binding and detection thus allows the specific quantitation of complex proteins (SIgA) in the presence of their subunits (FSC and IgA). This assay for SIgA will allow further investigation of the role of secretory immunity in both health and disease states.

746 RECURRENT MENINGOCOCCAL MENINGITIS AND ABSENCE OF THE SIXTH COMPONENT OF COMPLEMENT. Larry B. Vogler, Simon L. Newman, Rutherford B. Polhill, Robert M. Stroud, Richard B. Johnston, Depts. of Pediatrics and Medicine, University of Alabama in Birmingham, Birmingham, Alabama.

A healthy 5½ year old girl developed Neisseria meningitidis, group Y meningitis and bacteremia; the organism was sensitive to the antibiotics employed. Ten months after full recovery a second episode of group Y N. meningitidis meninigitis occurred. Appropriate leukocytosis and neutrophilia were present. Serum immunoglobulin levels were normal, as were antibody titers to blood group and typhoid antigens. Class specific fluorescent antibody titers to autologous meningococci, bactericidal antibody, and antibody to meningococcal protein antigen were elevated. There was a complete lack of serum hemolytic complement activity due to absence of the sixth component (C6). C3 was transiently depressed in association with a disseminated coagulopathy; C8 levels were 1/3 of normal; the remaining components were comparable to reference sera. Complement mediated opsonization of zymosan was normal, as was polymorphonuclear leukocyte killing of opsonized S. aureus and E. coli. The early acting components of the alternative complement pathway were functionally intact. T and B lymphocyte numbers and response to mitogens was normal. No coagulation defects were demonstrable. The patient's mother and uncle had half normal levels of C6. The two previously reported cases of C6 deficiency had recurrent N. gonorrhea and meningitidis infections respectively, underscoring the significance of this late complement component in host defense against Neisseria species. (Grants HD 00413, AI 07051 and AI 10286)

747 THE EFFECT OF IMMUNE COMPLEXES ON GOBLET CELL MUCUS RELEASE: A possible new mechanism of gastrointestinal host defense. W. Allan Walker and Kurt J. Bloch. Harvard Medical School, Mass. Gen. Hosp., Pediatric GI and Clin. Imm. Units, Boston.

In previous in vitro studies, we noted that immune complexes prepared in antibody excess and exposed to everted gut sacs consistently stimulated excessive mucus release. In the present study, the effect of immune complexes on mucus release was investigated in intact rats. Complexes (bovine serum albumin (BSA)-rat anti-BSA antibody) formed in two-fold antibody excess were injected at laparotomy into the duodenum of normal rats. Rats injected with BSA or purified anti-BSA antibody served as controls. In comparison to the controls, there was a marked increase ($P < 0.01$) in percentage of disrupted goblet cells (an index of mucus release) in segments from the intestine of rats exposed to immune complexes in vivo. Similarly, there was a significant increase in radiolabelled ($Na_2{}^{35}SO4$) mucus released into intestinal contents, rinse and mucosal homogenate fluids from rats exposed to immune complexes compared to those from rats exposed to BSA ($p < 0.001$) or purified rat anti-BSA antibody ($p < 0.001$) alone. Cholera toxin, used as a positive control, also produced a significant increase in percentage of disrupted goblet cells and in radiolabelled mucus release into each of the fluids tested. These findings strongly suggest that immune complexes can stimulate mucus release in intact rat small intestine; enhanced release of mucus may have an important role in clearing the intestine of immune complexes formed on the surface. (Supported in part by USPHS grants).

748 HYPERIMMUNE PLASMA THERAPY FOR ECHO TYPE 5 CHRONIC VIRAL MENINGITIS IN X-LINKED HYPO-γ-GLOBULINEMIA. L.S.Weiner, S.Baron, M. Langford, A.S. Goldman and R.A. Lord. Depts. Pediatrics and Microbiology, The University of Texas Medical Branch, Galveston, Texas.

Certain children with x-linked hypo-γ-globulinemia have been observed to be chronically infected with enteroviruses. We report experimental therapy of such a patient with hyperimmune human plasma. This 16-year-old affected male developed a chronic basilar meningitis characterized by motor, sensory and cognitive dysfunction after 12 years of γ-globulin therapy. In the CSF, protein was increased,glucose decreased and lymphocytes (principally T cells) were increased. Echo virus type 5 was cultured repeatedly from CSF and occasionally from the blood over a 12 month period, but no virus was detected in cultures of the pharynx, sputum, stool, urine or liver biopsy. Interferon was found in the CSF but not in serum.The patient's serum contained low titers of neutralizing antibodies (Ab) which behaved in an in vitro neutrallization test differently from those in human γ-globulin or serum in that the protection was of short duration.

Therapy with human plasma containing high titers of Ab to Echo virus type 5 was instituted 10 months after the virologic diagnosis. A 100-fold fall in CSF viral titers paralleled a rise in serum and CSF immunoglobulins and specific Abs, and neurologic improvement. The finding that hyperimmune plasma partly reversed this infection in an Ab deficient individual suggests that Abs protect against picornavirus infections of the CNS and that circulating Abs play a role in recovery from such infections.

749 DETERMINATION OF OPSONOPHAGOCYTIC DEFECTS IN HUMAN NEONATES BY GRANULOCYTE CHEMILUMINESCENCE. Mark E. Wilson, Michael A. Trush, Knox Van Dyke, Martha D. Mullett and William A. Neal (Spon. by Barbara Jones). W.Va. Univ. Med. Ctr., Depts. of Pharmacol. and Pediatrics, Morgantown, WV.

Alternate pathway complement activation and metabolic responsiveness of polymorphonuclear leukocytes (PMNs) were studied in eight premature neonates, gestational age 27 to 36 wks., by chemiluminescence (CL) assay. Zymosan, a yeast cell wall fragment known to activate alternate pathway, was used as the phagocytosable particle following opsonization by neonatal or adult serum. In the presence of adult opsonins both neonatal and adult PMNs gave comparable CL responses. However, in the presence of neonatal opsonins, a marked reduction in CL response by neonatal or adult PMNs resulted.

Granulocyte Chemiluminescence (cpm x 10^{-4}/5 x 10^6 adult PMNs)

	1 min	5 min	10 min
Adult serum	3.57 ± 0.4	13.26 ± 1.2	14.32 ± 1.5
Neonatal serum	0.86 ± 0.2	3.43 ± 1.1	4.90 ± 1.4

The results suggest that neonatal PMNs exhibit normal increases in oxidative metabolism (as reflected by CL responses) following the initiation of phagocytosis, but that neonatal serum is markedly deficient in its ability to generate opsonins via alternate pathway. This deficiency may constitute an important determinant in the predisposition of premature neonates to sepsis. Supported by WVU Senate and Institutional grants and NIH Training grant GM07039-02.

750 DEFECTIVE ACTIVATION OF THE TERMINAL COMPLEMENT (C') COMPONENTS IN NEWBORN SERA. Jerry Winkelstein, Larry Kurlandsky and Andi Swift, Johns Hopkins Univ Sch of Med, Dept of Ped, Baltimore.

Activation of the terminal C' components, C3-9,plays an important role in host defense against infection. The following study was performed in order to determine if the activation of C3-9 is normal in newborn sera. E coli, isolated from newborn blood or CSF, were incubated in sera at 37° for 30 min and the percent of available C3 that was activated was measured. Using one strain of E coli, 32% (mean) of the C3 was activated in sera from 18 newborns, as compared to 85% in sera from their mothers and 79% in sera from 13 normal adults ($P < .005$). In contrast, using another strain of E coli, the percent of C3 activated in newborns (83%) was the same as in their mothers (81%) or in normal adults (73%). The inability of some E coli to activate C3 in newborn sera was unrelated to the presence of the K1 antigen; in studying 7 additional strains some K1 positive strains activated C3 in newborn sera while some K1 negative strains did not. The defect in newborn sera appeared to be due to a deficiency of a serum component(s) rather than to the presence of an inhibitor.

Thus, activation of the terminal C' components in newborns is defective when tested with some, but not all, strains of E coli. However, the defective activation is not related to the presence of the K1 antigen.