

1009 THE BACTERIOLOGY AND CYTOLOGY OF CHRONIC MIDDLE EAR EFFUSION, G.S. Glebink, C.T. Le, S.K. Juhn, M.L. Weber
Depts. of Pediatrics, Biometry, Otolaryngology and
Laboratory Medicine, University of Minnesota, Minneapolis.

Middle ear effusions often persist in children with serous and mucoid otitis media despite medical therapy. Residual bacterial colonization of effusion may contribute to the pathogenesis of these conditions and was investigated by examining middle ear effusion for bacteria by culture and gram stain, and for cellularity by Papanicolaou stain. Effusion was obtained at the time of myringotomy and tympanostomy tube insertion from 509 ears of 317 children age 1 to 11 years; 362 effusions were mucoid, 79 were serous, and 60 were purulent. Mucoid otitis media was found most frequently in younger children, while serous otitis media predominated in older children. Bacteria were cultured from 30% of ears and included *H. influenzae* (15%), *S. epidermidis* (6%), *N. catarrhalis* (6%), *S. pneumoniae* (4%) and others (3%). An equal percentage of mucoid, serous and purulent effusions yielded bacteria by culture or gram stain. Bacteria were observed by gram stain in 16% of the sterile effusions, and the majority of the organisms were gram positive cocci. Phagocytes were noted on Papanicolaou stained smears in 37% of culture positive effusions, but were less frequent (23%) in sterile effusions. These results suggest that bacteria which are known pathogens in acute otitis media, persist in many chronic serous, mucoid and purulent middle ear effusions. The presence of bacteria as well as phagocytes in these effusions indicates chronic inflammatory stimulation which may contribute to the pathogenesis of chronic otitis media with effusion.

1010 REOVIRUS TYPE 3 ANTIBODIES IN THE SERA OF INFANTS WITH BILIARY ATRESIA. J. Glaser, S. Cho, R. Morecki & M. Horwitz. Albert Einstein College of Medicine, Bronx, New York.

Although a viral etiology for biliary atresia (B.A.) has been suspected, no viral isolates or antibody responses have been consistently correlated with the disease. Because our studies on the hepatobiliary injury in mice inoculated with reovirus type 3 (Reo 3) have shown that many stages of the human and rodent microscopic pathology are similar (Bangaru, et al, Lab. Invest. 43, 456, 1980), we have studied Reo 3 antibody responses in 15 infants with B.A. When neutralization tests were used, the sera from 2 babies demonstrated rising antibody titers to Reo 3. When antibodies were measured by indirect immunofluorescence using Reo 3 infected L cells as antigen in a coded protocol, 8 of 15 (53%) of babies with B.A. had Reo 3 antibodies. Some of these positive sera were shown to contain viral specific IgM. Only 1 of 18 (6%) of the controls had similar antibody ($P < .001$). Neutralization tests measure response to only 1 of 10 Reo 3 proteins; however, fluorescent antibodies probably recognize additional proteins. None of these children had antibody responses to cytomegalovirus, toxoplasma, rubella or herpesvirus nor had HB_eAg. We have been unable to isolate Reo 3 from stool, throat or liver specimens taken from babies with B.A.; however, at a corresponding stage of the murine Reo 3 disease, no virus can be recovered. These observations are consistent either with the persistence of defective Reo 3 or continuing immunologic mediated tissue damage. In contrast to the common, asymptomatic acquisition of Reo 3 antibodies later in childhood, our results suggest that neonatal infections with Reo 3 are related to hepatobiliary disease.

1011 RISK OF RESPIRATORY SYNCYTIAL (RS) AND PARAINFLUENZA TYPE 3 (PARA3) INFECTION IN YOUNG CHILDREN. W. Paul Glezen, Arthur L. Frank and Larry H. Taber. Baylor Col. of Med., Depts. of Micro. and Ped., Houston.

Children in the Houston Family Study were followed from birth to determine the occurrence, consequences and immune correlates of infection and reinfection with RS and Para3 viruses. This was a unique study of children living at home; 119 were followed 1 to 5 years—a total of 339 child-years. The RS infection rate was 63 per 100 child-years ranging from about 75/100 during each of the first 2 years of life to about 40/100 for ages 3-5. One-third of infections were clinically apparent. The illness rate was highest during year 2 at 27/100 children, but lower respiratory disease (LRD) was more frequent and severe in the 1st year of life. The LRD rate was 11/100 during the 1st year—2 infants were hospitalized. With primary infection the risk of LRD was 8.6/100 child-years. The overall risk of reinfection LRD was 6.4/100 child-years; however, the rate of reinfection LRD, clinically less severe, was 13/100 during the 2nd year and then dropped sharply for older children. No child had LRD more than 1 time.

Infection and illness rates were similar for Para3 but LRD was less frequent. LRD rate was 6.9/100 with primary infection vs. 1.1/100 with reinfection.

Risk for both viruses dropped after 2 infections. Thus, multiple live virus immunizations of children > 1 year could provide substantial protection to older children and protect infants by limiting exposure. Boosting maternal immunity may aid protection for early months of life.

1012 INHIBITION OF HUMAN BACTERICIDAL (BC) ANTIBODY AGAINST NON-TYPABLE (NT) H. INFLUENZAE BY OUTER MEMBRANE PROTEIN (OMP). Hanspeter E. Gnehm, Stephen I. Pelton, Sunita Gulati, and Peter A. Rice (Spon. by J. O. Klein). Boston University School of Medicine, Boston City Hospital, Departments of Pediatrics and Medicine, Boston.

Complement dependent BC antibody directed against NT-HI develops in convalescent sera of children with otitis media caused by NT-HI. These antibodies are present in sera of most healthy adults and we have found that they are represented in both IgG and IgM fractions. OMP and lipopolysaccharide (LPS) antigens were prepared from isolates of NT-HI from an adult with pneumonia (S1) and a child with otitis media (S2). These antigens were employed to absorb BC antibody activity from adult sera and the convalescent sera of the two patients; they were used at concentrations shown not to be anti-complementary.

Absorption with 9 µg of OMP-S1 of diluted sera (.025 ml) from 2 healthy adults reduced BC activity against S1 by $\geq 90\%$. OMP-S1 produced comparable reduction in BC activity against S1 using diluted convalescent sera of the adult infected with S1. An 80% reduction in BC activity against S2 was seen when convalescent sera from the child infected with S2 was absorbed with 90 µg OMP-S2. Sera absorbed with OMP-S2 produced comparable reduction in BC activity against S2 in 1 of 2 healthy adults. In amounts employed (≥ 127 µg) LPS did not produce a significant reduction ($\geq 50\%$) in BC activity in convalescent sera. Hypogammaglobulinemic serum was used as a complement source. These data support OMP as a target of BC antibody against NT-HI.

1013 CHEMOPROPHYLAXIS FOR CONTACTS OF CHILDREN WITH INVASIVE HAEMOPHILUS INFLUENZAE TYPE B (HIB) DISEASE. M. Glode, R. Daum, D. Goldmann, D. Ambrosino, N. Halsey, R. Russell, F. Mather, B. Sullivan, M. Murray, T. Johansen, J. Kamon. (Spon. by James K. Todd). Depts. of Pediatrics, Univ. of Colorado Health Sciences Center, Denver; Tulane University School of Medicine, New Orleans; Harvard Medical School, Boston.

We conducted a multicentered prospective double-blind placebo controlled trial of rifampin (R) chemoprophylaxis (10 mg/kg bid x2 days). 396 contacts of 79 patients with HIB disease participated. HIB carriage eradication rates were 63.6% and 23.9% for R and placebo, respectively, when all carriers were analyzed. However, in carriers < age 5, R was not significantly better than placebo for HIB eradication. We have recently completed a second chemoprophylaxis trial comparing eradication rates of R (10 mg/kg bid x2 days) and an investigational antibiotic trimethoprim/rifampin (TR), (2.6 mg/kg T and 10 mg/kg R bid x2 days). 139 index patients were identified with invasive HIB disease. 142 of 618 contacts were carriers of HIB and received either R or TR. Carriage eradication rates were 76.8% and 76.3% for R and TR respectively. In children <5, eradication rates were 70% and 68.4% for R and TR. Carriers who remained positive after completing 2 days of TR were offered a 4 day course of R (20 mg/kg/day qd x4 days). 4 of 13 carriers who participated in the cross-over trial remained positive after 4 days of R. We conclude that neither R nor TR in these doses and schedules reliably eradicates HIB carriage in culture positive contacts. In addition, a 4 day course of R was not uniformly successful in those individuals who failed TR therapy.

1014 BACTERICIDAL EFFECT IN VITRO OF IRON DEPRIVATION AND HIGH TEMPERATURE ON A CLASSIC PSEUDOMONAS AERUGINOSA RECOVERED FROM A PATIENT WITH CYSTIC FIBROSIS.

Charles T. Gordon, James M. Chamberlain, David R. Cross, Westminster College, Department of Chemistry, Westminster, MD; Robert C. Stern, Case Western Reserve University, Rainbow Babies and Childrens Hospital, Department of Pediatrics, Cleveland, OH.

Recent studies suggest that fever acts in concert with nutritional deprivation to aid the host in coping with certain bacterial infections (See Kluger: Pediatrics 66:720, 1980).

We studied the effects of high temperature and iron deprivation on the growth of a classic *Pseudomonas aeruginosa* isolated from the sputum of a patient with cystic fibrosis. Organisms grew well at both 37 C and 41 C in glucose minimal medium containing 1000 µg iron/ml medium. When desferrioxamine was added, however, growth ceased at 41 C, but was virtually unaffected at 37 C. After 34 hours of incubation at 41 C in medium containing desferrioxamine, the organisms were then unable to grow at 37 C. The inability of *Pseudomonas aeruginosa* to survive at 41 C in the presence of desferrioxamine may be due to inability to synthesize iron transport compounds at this temperature. This finding of increased need for easily obtained iron at high temperature may have clinical importance in the management of *Pseudomonas aeruginosa* pulmonary infection in patients with cystic fibrosis.