**●1117** *STAPHYLOCOCCUS EPIDERMIDIS* SLIME INTERFERES WITH NEUTROPHIL PHAGOCYTOSIS AND OXIDATIVE KILLING MECHANISMS. GM Johnson[1], DA Lee[1], WE Regelmann[1], ED Gray[1], G Peters[2], and PG Quie[1]. University of Minnesota, Department of Pediatrics, Minneapolis[1], Hygiene Institute, University of Cologne, West Germany[2].

*Staph. epidermidis* is a common foreign body pathogen, particularly of plastic intravenous catheters. Many strains isolated from infected IV catheters produce a loosely adherent slime material, which we have previously demonstrated interferes with human PMN chemotaxis. A surface phagocytosis assay was used to closely simulate catheter infections; slime effects on chemiluminescence (CL) and superoxide ($O_2^-$) production were examined.

Human PMN were added to layers of *S. epi* incubated 18 hours on plastic plates. Control *S. epi* were washed to remove slime and adhered to plastic plates for 2 hours. $^3$H-labeled bacteria were present at approximately 10:1 bacteria/PMN ratio. At 15 and 60 minutes PMN phagocytosis of *S. epi* with slime was less than that of washed *S. epi* ($p<.05$). Opsonization (10% human sera) did not significantly increase uptake of *S. epi* with slime in contrast to washed bacteria (7% versus 42% change).

Purified *S. epi* slime modestly decreased PMN CL and $O_2^-$ production in response to zymosan and phorbol myristate acetate. Slime stimulated PMN specific granule release (lactoferrin).

*S. epi* slime inhibition of PMN chemotaxis, phagocytosis and oxidative metabolic response may contribute to the persistence of these bacteria on plastic catheters.

**1118** CHRONIC-REACTIVATION EBSTEIN-BAR VIRUS (EBV) INFECTION PRESENTING AS RECURRENT ASEPTIC MENINGITIS. Anand G. Kantak, Patrick G. Brosnan, Tasnee Chonmaitree, Tamara A. Rakusan, James F. Jones, and Armond S. Goldman. The University of Texas Medical Branch, Department of Pediatrics, Galveston, and the National Jewish Hospital, Denver.

A wide spectrum of chronic-reactivation infections due to EBV has been documented in patients with or without primary immunodeficiencies. Recently, we found evidence suggesting EBV as the cause of recurrent aseptic meningitis. This male child presented several years beforehand with recurrent urticaria, arthralgias, chronic papillitis, neurosensory hearing loss, and 5 episodes of aseptic meningitis (last episode at 13.2 yr.). CSF during acute episodes contained up to $1X10^4$ leukocytes/mm$^3$ (principally neutrophils), elevated proteins (70-250mg/dl), but no microorganisms. B and T cell functions appeared normal. Serum antibodies to EBV were:

| Type of Antibody | 11 yr. | 13.2 yr. | 13.3 yr. |
|---|---|---|---|
| Capsid Antigen | | | |
| IgM | <1:10 | <1:10 | <1:10 |
| IgG | 1:640 | 1:640 | 1:2560 |
| Early Antigen (IgG) | | | |
| Diffuse | 1:40 | 1:10 | 1:20 |
| Restrictive | <1:10 | 1:80 | 1:160 |
| Nuclear Antigen (IgG) | 1:20 | 1:10 | 1:20 |

In addition, CSF contained IgG antibodies (1:5) to the capsid antigen. Thus, the chronic-recurrent central nervous system abnormalities may be due to EBV. EBV genes and antigens in the child's cells and immune response of the patient to EBV are being investigated.

**1119** INFECTION WITH MULTIPLE EB VIRUS (EBV) GENOTYPES IN AN INFANT WITH AIDS AND HIS MOTHER. Ben Z. Katz, Warren A. Andiman, George Miller. Yale University School of Medicine, Department of Pediatrics, New Haven, CT.

Children develop AIDS if a parent or household member falls into one of the high-risk groups. Infections with EBV are common in these patients and are responsible for some of the severe complications of the disease. We have studied a child with AIDS and his mother; both harbor EBV. We wished to determine whether they were infected with the same strain. We examined cellular DNA from lymphoblastoid cell lines (LCL's) derived from the child and his mother for different EBV isolates using Southern blot analysis with probes prepared from cloned segments of the EBV genome. A LCL derived from lymph node biopsy tissue and one from the patient's peripheral blood contained two different genotypes of EBV. A LCL that arose spontaneously from mother's blood contained the same two EBV genotypes. A single cell subclone of the mother's LCL contained only one of these strains; thus the mother's LCL was shown to be polyclonal. After one year of observation the infant developed a CNS lymphoma which was shown to contain yet a third EBV genotype. These results indicate that, at least in AIDS patients, simultaneous infections with more than one EBV strain is possible. Whether this is also true of immunocompetent hosts is presently unknown. These data also raise the possibility of vertical transmission of EBV from mother to infant.

**●1120** PREVALENCE OF GENITAL HERPES AND/OR HERPES SIMPLEX VIRUS TYPE 2 (HSV-2) ANTIBODIES IN TWO OBSTETRIC POPULATIONS. Harry Keyserling, Sumner Thompson, Michael Robinowitz, Francis Lee, Lenore Pereira, R. Marie Coleman, Raymond Bain, and André Nahmias. Emory Univ. School of Medicine, Atlanta, GA and Calif. Health Dept., Berkeley, CA

Since the pregnant woman's HSV infected genital tract is the source of virus in most cases of neonatal herpes, monitoring in the last trimester for genital virus in a woman with a history in herself or her partner is currently recommended as a method of possible prevention by C-section. We have examined the prevalence of a history of genital herpes, as well as measured HSV-2 antibodies (with an immunodot ELISA on nitrocellulose paper using a purified HSV-2 typespecific glycoprotein, gG-2) in two obstetric populations: a low socioeconomic (LS) group of 300 women and a middle class HMO group of 187 women. The prevalence of genital herpes in pregnant women by history in LS was 3.5% and in HMO 10%; including a history of genital herpes in the male partner, the rates were 4% and 13% respectively. The rates of HSV-2 antibodies in the pregnant women were 49% (LS) and 33% (HMO). Variability in history and antibody rates was noted according to age, race and awareness of the clinical entity of genital herpes. We conclude: (a) that a high proportion of obstetric patients are at some risk for transmitting HSV to their infants; (b) the current monitoring policy needs to be modified.

The changing patterns of neonatal herpes as a consequence of the above, as well as other current findings to be discussed, will affect any new approaches to management of this severe neonatal problem.

**1121** IN VITRO AND IN VIVO STUDIES OF HUMAN IMMUNE SERUM GLOBULIN (ISG) MODIFIED FOR INTRAVENOUS USE AGAINST TYPE III GROUP B STREPTOCOCCUS (GBS). Kwang Sik Kim, Carol Wass and Bascom F. Anthony. UCLA School of Medicine, Harbor-UCLA Medical Center, Department of Pediatrics, Torrance, CA.

The mortality and morbidity of neonatal GBS disease remain significant, even with optimal antibiotic therapy. Recently, with the availability of several ISG preparations modified for iv administration, use of this material as therapeutic adjunct has been suggested. However, it is not known whether ISG modified by different methods will have similar biological activity. We compared functional activities in vitro and in vivo of two ISG modified for iv use against a type III GBS, reduced and alkylated (RA) and native (N) ISGs. Both preparations (5%) contained similar amounts of type III (4.0 vs 4.8 µg/ml) and group B (49.5 and 49.8 µg/ml) streptococcal antibodies of IgG class measured by the ELISA. IgM and IgA antibodies were undectable.

In vivo, we used the newborn rat model of GBS bacteremia and meningitis. Five-day-old rats received ip 10-fold diluted RA or N ISG in a dose of 50 µl/10 gm and then sc $LD_{100}$ of the GBS strain. Mortality was recorded for 5 days and blood cultures done in dead animals. The 50% protective dose (dilution) was 1:4 for RA ISG vs 1:16 for N ISG. In vitro studies measured phagocytosis and killing of the GBS strain by rat PMNs in the presence of ISG and rat complement. Efficient opsonophagocytosis ($\geq$90% killing) occurred with $\leq 10^{-3}$ dilution of RA ISG vs $\leq 10^{-4}$ dilution of N ISG.

The findings suggest that RA ISG is less active than N ISG against type III GBS. Further studies are needed to understand the mechanisms responsible for this apparent discrepancy in functional activity of RA and N ISGs.

**†1122** TREATMENT OF EXPERIMENTAL E. COLI (EC) INFECTION WITH THE COMBINATION OF CHEMOTHERAPY AND IMMUNOTHERAPY. Kwang Sik Kim, Carol Wass, Elizabeth Ziegler, Alan Cross, Bennett Kaufman and Jerald Sadoff (Sponsored by B.F. Anthony). UCLA School of Medicine, Harbor-UCLA Medical Ctr, Dept. of Pediatrics, Torrance, CA; UCSD School of Medicine, Dept. of Medicine, San Diego; Walter Reed Army Medical Center, Washington, D.C.

The morbidity and mortality associated with neonatal EC infection have remained significant despite advances in antimicrobial chemotherapy. In a search for more effective therapy, we evaluated combination therapy against a $K_1$ EC with cefotaxime (Ct) and two different antibody (Ab) preparations, rabbit J5 antiserum (J5 Ab) and murine IgM monoclonal antibody to the lipopolysaccharide of the infecting EC serotype (LPS Ab). EC bacteremia and meningitis were induced in 5-day-old rats by sc inoculation. At 6 days of age, each litter was divided into 3 groups to receive Ct (50 mg/kg sc twice daily), one of the Ab preparations (50 µl/10 gm ip) or combination therapy. A control rat from each litter received saline. Bacterial counts in blood and CSF were determined daily before and during therapy and mortality recorded. All animals receiving saline died. Bacterial clearance was inversely related to the mortality, which is summarized below:

| Exp 1. Rx | Mortality(%) | Exp. 2. Rx | Mortality(%) |
|---|---|---|---|
| Ct | 5/11 ( 45%) | Ct | 13/24 (54%) |
| J5 Ab | 10/10 (100%) | LPS Ab | 15/17 (88%) |
| Ct+J5 Ab | 7/11 ( 64%) | Ct+LPS Ab | 6/24 (25%) |

As noted, LPS Ab enhanced the efficacy of Ct while J5 Ab was not beneficial. These findings suggest that the combination of antibiotics and LPS Ab have enhanced efficacy in this model.