907 SPECIES DIFFERENTIATION AND BIOCHEMICAL MARKERS FOR TOXIGENICITY IN FECAL AEROMONAS ISOLATES FROM CHILDREN. B.J. Freij, S. Shelton, A.A.M. Lima, R.L. Guerrant, and J.D. Nelson. Univ. of Texas Health Science Center, Dallas, TX, and the Univ. of Virginia, Charlottesville, VA.

The role of Aeromonas (Aer) in the etiology of pediatric diarrhea is incompletely defined. It is unclear whether identification of isolates at the species level or performance of toxigenicity studies help distinguish pathogenic from non-pathogenic strains. We isolated Aer from 33 of 704 children whose fecal specimens were submitted to the Microbiology Laboratory of Children's Medical Center, Dallas, over a 1-year period. Isolates were identified as Aer using the API 20E system. Additional tests performed on 29 strains included gas from glucose, salicin fermentation, growth in KCN, esculin hydrolysis, and elastase production. A. caviae was the predominant species (n = 19, 66%). A. sobria and A. hydrophila accounted for 3 each and 4 (14%) strains could not be speciated. Toxin production by Aer was studied using CHO cell, adrenal Y1 cell, suckling mouse, and rabbit ileal loop assays. Twelve (39%) of 31 strains were positive in at least one of these assays including 3 (17%) of 18 A. caviae and all 9 non-caviae strains tested. Two toxigenic strains were from asymptomatic older children. Toxigenic strains were significantly more likely to be positive for arginine dihydrolase ($p < 0.05$), lysine decarboxylase ($p < 0.001$), and Voges-Proskauer reaction ($p < 0.01$), produce gas from glucose ($p < 0.001$), and not ferment arabinose ($p < 0.005$) compared with non-toxigenic strains. We conclude that Aer differentiation at the species level is of limited clinical value and that several biochemical reactions can serve as markers for toxin production.

●908 A PROSPECTIVE STUDY OF PREGNANT WOMEN TO DETERMINE PREVALENCE AND RISK FACTORS OF ASYMPTOMATIC INFECTION WITH HERPES SIMPLEX VIRUS (HSV) TYPE 2 AND THE RISK OF SHEDDING VIRUS AT DELIVERY. LM Frenkel, JP Shen, E Garratty, YJ Bryson. UCLA Sch. Med., Los Angeles, Ca.

In spite of a greater awareness and current obstetrical practices, the incidence of neonatal HSV is increasing. The mothers of 50-70% of these infants have no hx of genital HSV, yet serology usually implies asymptomatic (ASX) HSV2 infection. We prospectively studied 231 pregnant middle & upper SES women with no hx of genital HSV to determine the prevalence and risk factors of ASX HSV2 and the rate of genital tract shedding of HSV at delivery. We assessed Ab to HSV1 and 2 in prenatal sera by western blot, and risk factors for HSV2 infection by questionnaires. HSV2 Ab was detected in 79/231 (34%) of the women. Seropositivity was associated with a hx of ≥ 6 sexual partners, TABs, other STDs and yeast vaginitis ($p < .05$); while age, age at first sexual intercourse, a hx or present sexual partner with genital HSV was not significantly associated with HSV2 Ab. A first episode of clinical HSV developed during pregnancy in 7/43 ASX HSV2 seropositive women who have delivered; 5 occurred close to term and were delivered by C/S. ASX shedding of HSV2 was detected in an additional woman 4 wks prior to delivery with negative weekly cultures subsequently. Among the remaining 35 ASX seropositive women, 178 viral cultures during the last 6 wks of pregnancy or at delivery were negative. The high prevalence of HSV2 Ab in this ASX pregnant population stresses the importance of further assessing the risk of viral shedding and potential transmission of HSV to the infants.

●909 LIVE ATTENUATED VARICELLA VACCINE: USE IN HEALTHY ADULTS. Anne Gershon, Sharon Steinberg, Philip LaRussa, Philip Oh, Lawrence Gelb & the NIAID Varicella Vaccine Collaborative Study Group. Dept. Pediatrics, Columbia University College of Physicians & Surgeons, NY, NY, & Dept. Medicine Wash U., St. Louis, MO.

We administered live attenuated varicella vaccine to 187 healthy adults seronegative to varicella-zoster virus (VZV); 176 have been followed at least 2 mos. 121/176 (69%) received 2 doses about 3 months apart. Side effects after 1 dose were: local reactions in 18 (10%), rash in 12 (7%), & fever in 4 (2%). Side effects were less frequent after the 2nd dose. Vaccine associated rash was mild (mean 14 lesions, range 1-100). Vaccine type virus was isolated from 1 adult with a total of 8 lesions, 1 mo. after vaccination; there was no spread of vaccine virus to others. Seroconversions to VZV, measured by fluorescent antibody to membrane antigen (FAMA) were: after 1 dose 136/169 (80%), & after 2 doses 114/121 (94%). 22 seroconverted only after the 2nd dose; 7 (6%) failed to seroconvert after 2 doses. Positive antibody responses have been detected after 1 yr in 44/67 (66%), after 2-3 yr.in 19/28 (68%), & after 4-6 yr in 10/13 (77%). Ten have had a household exposure to varicella after as long as 7 years (mean 3 yrs); 6 (60%) developed at least some evidence of clinical varicella with 1-40 lesions (mean 16) but no toxicity. Seropositives (5/10) rarely developed clinical symptoms; 1 had 1 vesicle. There were a total of 10 cases of mild varicella in adult vaccinees with 1-58 lesions, (mean 10), 4-72 months after immunization. Wild type virus was isolated from 2 MDs, but there was no spread. Clinical infection was likely to occur in those seronegative at exposure (7/8, 88%) but the illness was still very mild. Varicella vaccine has the potential to control but not prevent nosocomial varicella. Varicella vaccine is less protective for healthy adults than for healthy children, but it modifies the illness even if VZV antibodies are no longer detectable.

910 DOES LIVE VARICELLA VACCINE (LVV) PROTECT AGAINST OTHER HERPESVIRUSES? Alice K. Gong, Susan V. Lipton, Philip A. Brunell, The University of Texas Health Science Center, Dept. of Pediatrics, San Antonio, TX.

It has been shown that Herpes Simplex Virus (HSV) and Varicella-zoster Virus (VZV) share antigenic determinants and that the VZV genome contains homologous regions to HSV-1 and HSV-2 genomes. More recently Edson, et al, found that the envelope glycoproteins HSV gB and VZV gp63 share common antigens and that cross reactive anti-HSV polyclonal and monoclonal antibody can neutralize VZV infection. Because of these common antigens, we sought to determine if administration of LVV in children can cause cross-reactive immune responses which could confer partial immunity to the heterologous viruses. Blood was obtained from 43 children at the time of LVV vaccine and three months later. A control group of 36 age matched children were tested to determine seroconversion rates in the absence of vaccine. Antibody response was determined by enzyme-linked immunosorbent assay. A sero-negative range for HSV and for CMV was established by studying sera obtained from 200 individuals of varying ages including newborns, infants, and adults. Seroconversion rate for the immunized population was 0 of 43 infants for HSV and 1 of 40 for CMV; respective rates for the control population were 2 of 35 and 1 of 36. (NS) We conclude that immunization with LVV vaccine offers no heterologous antibody protection to HSV and CMV in children.

●911 IGG RESPONSES TO H. INFLUENZAE TYPE B (HIB) AND PNEUMOCOCCAL (PN) TYPES 3 (P-3) AND 23 (P-23) POLYSACCHARIDES (PS) IN CHILDREN DEVELOPING HIB DISEASE DESPITE VACCINATION WITH HIB PS VACCINE. Dan M. Granoff and Penelope G. Shackelford, Wash. Univ. Sch. of Med., & Children's Hosp., St. Louis.

Hib PS-vaccinated patients (VP) who develop Hib disease have deficient serum antibody (AB) responses to Hib PS despite normal concentrations of Ig and tetanus AB (NEJM 1986;315). We nov report the IgG subclass composition of Hib PS AB in VP who recovered from infection, and their IgG responses to Pn vaccine. 14/41 (34%) VP were considered to be AB responders to Hib disease (>1 µg/ml of Hib PS Ab in convalescent sera as measured by a Farr assay) compared with 14/25 (56%) unvaccinated patients with Hib disease of similar ages ($P=.08$). In 10 responder VP (mean age=34 mo), the geo. mean convalescent IgG, G1 and G2 Hib PS AB as measured by ELISA, were 0.97, 0.69 and 0.09 µg/ml, respectively. The IgG and G1 values were 10-fold lower than those in convalescent sera from 14 unvaccinated responder patients (mean age = 37 mo) (9.24, 7.37 and 0.2 µg/ml, respectively; $P<0.01$). 18 of 41 Hib VP (mean age of 40 mo) were vaccinated with 23-valent Pn PS. They showed significant increases in geo. mean recip. serum IgG titers to P-3 (107 to 1936, $P<0.001$) and to P-23 (52 to 120, $P<0.02$). However, the geo. mean Pn titers in post-immune serum of the Hib VP group were lower than those of 15 immunized healthy children (mean age=41 mo) (P-3: 3350, $P<0.05$; P-23: 279, $P=0.10$). Thus, VP have deficient IgG responses to Hib PS following recovery from Hib disease, and the subclass most affected is G1. Hib VP can respond to Pn PS vaccine but their IgG responses are lower than those of healthy children.

912 DERMAL REACTIVITY TO HUMAN CYTOMEGALOVIRUS (CMV). Renu Gupta, Eva Gonczol, John Ianacone, Karen Connor, Stanley A. Plotkin, Division of Infectious Diseases, The Children's Hospital of Philadelphia, and the Wistar Institute, Philadelphia, PA.

A skin test for immunity to human CMV is described in which skin induration is measured after intradermal injection of heat-inactivated Towne strain CMV antigen. Randomly selected healthy young adult males and non-pregnant females were prescreened for evidence of past infection with CMV using a latex agglutination test. Each individual was inoculated with 100 ul of test antigen (50 ug) prepared from serum-free supernatants of CMV-infected MRC-5 cells (inactivated at 56°C for 6 hrs), Candida extract (1:1000) and non-infected MRC-5 cell lysates. CMV seropositive individuals elicited positive skin reactions to both the Candida extract and the test antigen. No response was observed at the MRC-5 cell lysate inoculation site. Seronegative individuals who were negative to the test antigen at the start of the study, developed a positive response 8 days after intramuscular immunization with live attenuated Towne strain CMV. This response also correlated with in vitro CMV- induced lymphocyte proliferation of peripheral blood lymphocytes from the immunized individuals and persisted for at least 93 days.

In guinea pig experiments with human CMV, those animals immunized with purified CMV, a virus envelope preparation, or a 130-55K glycoprotein complex (isolated by immunoadsorbent column chromatography), developed strong skin reactions to intradermal injection of 5 ug of each of these antigens. A weak reaction was also observed to viral capsid antigen. No reactions were observed in non-immune animals.