1068 ACTIVATION AND MUCOSAL MIGRATION OF POLYMORPHONUCLEAR LEUKOCYTES (PMN) DURING RESPIRATORY SYNCYTIAL VIRUS (RSV) INFECTION. Howard S. Faden, Tej N. Kaul, Tzou-Yien Lin, Pearay L. Ogra. Dept. Peds., SUNY at Buffalo, and Children's Hospital, Buffalo, N.Y.

The distribution and functional role of PMN during acute infection with RSV was evaluated by characterizing the inflammatory process in nasopharyngeal secretions (NPS) and by determining oxidative metabolic activity of peripheral blood (PB) PMN in children with RSV-induced lower respiratory tract illness. PMN comprised > 70% of the cells in the NPS during active shedding of RSV. A similar cellular response was observed in secretions from the lower respiratory tract of other RSV-infected children who required tracheal intubation. The proportion of PMN (42%) in the PB was significantly lower than the proportion of PMN in the NPS of the same subjects. PMN collected from the PB of infected children manifested significant elevations of oxidative metabolism as evidenced by increased generation of resting chemiluminescence. Subsequent challenge of the PMN with opsonized zymosan failed to induce adequate chemiluminescence in the RSV-infected subjects. These data demonstrate in vivo activation of the oxidative metabolic pathway in PMN after RSV infection. Contrary to the popular notion, it is suggested that PMN constitute the predominent element of the cellular response at the mucosal site of disease following RSV infection. These observations indicate that the migration of PMN to the site of RSV replication and their interactions in the respiratory mucosa may have a role in the pathogenesis of RSV disease.

●1069 THE SYNERGISTIC RELATIONSHIP BETWEEN ANTIBIOTICS AND POLYMORPHONUCLEAR LEUKOCYTES (PMN) IN BACTERICIDAL ACTIVITY. Howard Faden, Jung J. Hong, and Pearay L. Ogra. State University of New York and Children's Hospital, Buffalo, N.Y.

The effect of clindamycin on PMN function was investigated in a group of normal adult volunteers who received 1200 mg of clindamycin daily for two days. Neutrophils obtained before and during clindamycin administration were tested for chemotaxis under agarose, phagocytosis and intracellular killing of staphylococci (S. aureus) by the acridine orange staining technique, and oxidative metabolic activity by chemiluminescence generation. Serum-free, washed PMN collected from subjects during clindamycin administration killed significantly greater proportion of S. aureus than the controls (45% vs. 38%, $p<.001$). The increased bactericidal activity was not related to the serum concentration of clindamycin (mean 1.6 µg/ml) which was well below the MIC for this particular organism. Despite the effect of clindamycin on the intracellular killing of S. aureus, chemotaxis, phagocytosis, and chemiluminescence generation did not differ significantly from the controls. These data suggest significant augmentation of PMN bactericidal activity with clindamycin. Because clindamycin is unique among antibiotics in that it readily enters PMN, it is proposed that PMN may act as vehicles for the transport of antibiotics to the site of infection and thus complement the antibacterial activity of certain antibiotics.

1070 THE DETECTION OF GROUP B STREPTOCOCCAL POLYSACCHARIDES IN URINE BY AGGLUTINATION. Neil Feld, Kenneth Bromberg, and Yi Zheng (Spon. by L. Glass). SUNY Downstate Medical Center/Kings County Hospital Center, Department of Pediatrics, Brooklyn, New York.

Prepared staphylococcal co-agglutination reagents were used for the detection of group B streptococcal polysaccharides in the concentrated and unconcentrated urine of 78 infants with presumed bacterial infection. These prepared reagents were compared to commerically available reagents (Phadebact, Wellcogen). All reagents were evaluated for their ability to identify patients infected with group B streptococci and for their ability to detect purified group B and type III polysaccharides. All three reagents were able to identify the six patients with positive group B streptococcal blood cultures when concentrated urine was tested. All three reagents had equivalent levels (62.5 ng/ml) of detection for the group B polysaccharide when 20 ul of polysaccharide solution was used. These reagents had poor levels of detection for the type III polysaccharide. Prepared reagents were as sensitive as those commercially available for the detection of infants with group B streptococcal disease and were one one-hundredth the cost.

†1071 PLACEBO-CONTROLLED TRIAL OF LIVE ATTENUATED COLD-ADAPTED (CA) INFLUENZA A VACCINES (H1N1 & H3N2) VERSUS INACTIVATED WHOLE VIRUS INFLUENZA A (H3N2) VACCINE. Sandor Feldman, Peter Wright, Jo Mahoney, Juliette Thompson and Paula Robertson. St. Jude Children's Res. Hosp., Memphis, TN., Dept. of Peds., Vanderbilt Univ., Nashville, TN.

On the basis of prevaccination serologic findings, 112 children, in Oct. of 1982, were randomized to receive intranasal (IN) live CA H1N1 (Group A), IN live CA H3N2 (B), intramuscular killed whole virus H3N2 vaccine (C), or an IN saline placebo (D) for the purposes of comparing vaccine immunogenicity, safety and efficacy. All but one child completed the first 6 mo of the study. At 6 wk, followup, 16/28 (57%) subjects in Group A showed seroconversion, compared with 21/25 (84%) in B ($p=<.025$), 22/22 in C, and 0/30 in placebo controls. Mean reciprocal HAI titers (log 2) were 3.9 in Group A, 5.0 in B and 5.8 in C, with 6 mo titers of 5.0 and 4.2 for B and C (significantly greater change for C vs B, $p=.02$). The mean clinical reaction scores for A and B, obtained on days 3 and 6, did not differ significantly from placebo recipients. Complications were limited to febrile reactions (38.2-40.9°C) in C: 32% at 6 hr, 36% at 12 hr and 9% at 24 hr (1 febrile seizure). Adverse reactions to killed whole virus H3N2 vaccine were unacceptable, whereas live CA vaccines were well tolerated. From Feb.-Apr. 1983, during an H1N1 outbreak, 52 cultures were obtained from 42 children and H1N1 virus was isolated from 8 (none in A). Of the 12 non-responders to the H1N1 vaccine, 6 had serologic evidence of infection compared with 4 of 16 responders. H3N2 or placebo recipients seronegative to H1N1 had a 37% (19/52) attack rate (antibody increase or viral isolate), compared with 7% (2/29) with a titer of ≥16. Comparison of influenza-related illness (illness with 4-fold antibody rise and/or viral isolate) for H1N1 seroresponsive vaccinees (0/16), with those of other children in the trial without natural antibodies to H1N1 (9/53), suggests vaccine efficacy in prevention of clinical illness.

1072 **HUMORAL IMMUNE RESPONSE IN INFECTIOUS MONONUCLEOSIS: LATE EMERGENCE OF ANTI-EA(R) AND EFFECTS OF STEROID THERAPY.** **Gary Fleisher, Marjeanne Collins, Samuel Fager. (Spon. by Stuart Starr). The Children's Hospital of Phila., Infectious Diseases Division, Phila., PA.**

Primary infection with the Epstein-Barr virus (EBV) evokes a characteristic virus-specific humoral immune response which relates to the clinical type and duration of infection. Following infectious mononucleosis (IM) patients (pts) produce IgM and IgG antibodies (Abs) to viral capsid antigen (VCA), IgG Abs to diffuse (D) early antigen (EA) in 70% of cases, and subsequently IgG Abs to EBV nuclear antigen (EBNA). Occasional pts with IM have been reported to develop Abs to restricted (R) EA, usually in association with severe disease.

We studied the Ab response to EBV in pts with IM in order to ascertain the appearance of Abs to EA (R) and to determine the effects of steroids on all aspects of the humoral immune response. 60 college students with heterophil (+) IM, confirmed by EBV-specific serology, were followed for 4-26 wks; half received prednisone for 6 days and the remainder took no steroids. Irrespective of therapy, 48% of the patients developed anti-EA(R). The response to other antigens was similar in both groups with the exception that Abs to EBNA developed later during convalescense and at lower titers in the steroid-treated group. We conclude: (1) anti-EA(R) Abs develop with considerable frequency following IM and are not a marker, as proposed previously, of unusual severity and (2) steroid therapy may retard the formation of anti-EBNA Abs but does not otherwise effect the humoral immune response to EBV.

●1073 ADENOVIRUS INFECTIONS AN IMPORTANT CAUSE OF MORBIDITY IN HOSPITALIZED CHILDREN FOLLOWING GASTROINTESTINAL SURGERY. Cynthia C. Franklin, Robert H. Yolken, Johns Hopkins Medical School, Baltimore, Maryland 21205

We investigated the effect of enteric adenovirus infections (ETAD) on children who had undergone abdominal surgical procedures resulting in intestinal stomas (ostomies). We studied 58 such children hospitalized in our institution over a 27 month period, 28 of whom had an underlying diagnosis of NEC. A total of 13 (22.4%) of the patients had at least one episode of ETAD during hospitalization documented by enzyme immunoassay during the study period. Ten of the 28 (35.7%) children with NEC had ETAD infections. Four of these children had evidence of two distinct infections with ETAD.

All of the children infected with ETAD had evidence of a substantially increased gastrointestinal output associated with their infections. In addition, infection with ETAD was associated with a substantial increase in length of hospitalization, (mean stay 163.8 +/- 42.4 days vs 46.1 +/- 11.8 days in uninfected controls, $p < .01$). Gastrointestinal infection with adenovirus was not associated with respiratory symptoms or conjunctivitis. Other causes of gastroenteritis such as rotavirus, bacteria, or toxins were not found to be significant gastrointestinal pathogens in this population and increased stool output was only infrequently noted in patients without evidence of ETAD. Adenoviruses appear to be important causes of morbidity in hospitalized patients with ostomies, especially in those with a history of NEC.