763 ERADICATION OF SERRATIA MARCESCENS FROM AN NICU. Drusin, L.M., Krauss, A.N., Auld, P.A.M. N.Y. Hosp.-Cornell Med. Cent. Perinatology Center and Div. of Epidemiology, New York, N. Y. 10021.

In a 10 month period 29 infants in an NICU were infected with a highly resistant non-pigmented strain of Serratia marcescens. The organism was cultured from stool, umbilicus, catheter tip, eye, groin, blood, and at autopsy from CSF, lung, and spleen. After one death from Serratia sepsis (+ blood culture) 5 infants in the same room developed positive cultures in nose, urine, stool and cord. Positive cultures for the same strain were found retrospectively in 8 infants during the preceding 6 months, none of whom demonstrated clinical illness. As Serratia is commonly water-borne, environmental survey was carried out. Blood pressure machines, EKG paste, and urine refractometers were negative. The epidemic strain was recovered from an incubator humidifier. An aseptic protocol for cleaning incubators was adhered to and the room with infected infants was closed and cleaned. Despite these measures 7 additional infants became infected. The Serratia was finally eradicated by placing all infants on gown and glove precautions, admitting new infants to clean rooms only, and by segregating older infants. Although 10 older infants in the isolated group developed Serratia infections in the following month, no new infant became colonized or infected. Two weeks following the discharge of the last infected infant Serratia was recovered from the umbilicus of a transported infant. This infant's strain differed in antibiotic sensitivities from the previous strain. Adherence to environmental control prevented its spread.

764 SEVERAL SEROTYPES OF ENTEROTOXIGENIC E. COLI AND REOVIRUS-LIKE AGENTS IN PEDIATRIC DIARRHEA. P. Echeverria, N. Blacklow, C. Ulyangco, and L. DuPont (Spon. by A.L. Smith). U.S. Naval Medical Research Unit No. 2, Taiwan, University of Massachusetts Medical School, Worcester, MA., San Lazaro Hospital, Manila, The Philippines, and the University of Texas Health Science Center, Houston, TX.

We investigated the etiology of pediatric diarrhea in Manila from January-June, 1976. Heat-labile and heat-stable enterotoxigenic (tox^+) E. coli were found in 11 percent (9/82) of children hospitalized with diarrhea and 8 percent (4/49) of healthy controls. None of the tox^+ E. coli were of classical enteropathogenic serotypes, however serotypes O6:H16, O8:H9 and O78:H12, found to be tox^+ in other areas of the world, were isolated from 6/9 of children with diarrhea, compared to 1/4 of well controls. Thirty-eight percent (16/42) of tox^+ E. coli isolated from children with diarrhea as compared to 6% (1/17) isolated from well controls belonged to these serotypes ($p < 0.01$). Seventeen percent of children had reovirus-like agent (RVLA) infections (27% < 3 yrs of age) as determined by detection of viral particles in stools by electronmicroscopy (15%, 12/82) and/or by an antibody rise to the serologically related Nebraska calf diarrhea virus (20%, 8/39). We conclude that RVLA's are a frequent cause of diarrhea in children less than three years of age in Manila, and tox^+ E. coli infections account for a small proportion of disease. Special serotypes of E. coli may be more virulent with certain O & H antigens appearing as markers for undefined virulence factors.

765 ASSOCIATION OF PLASMID-MEDIATED ANTIBIOTIC RESISTANCE AND ENTEROTOXIN PRODUCTION IN AN ESCHERICHIA COLI ENTEROPATHOGENIC FOR CHILDREN. Peter Echeverria and Jack Murphy (Spon. by Arnold L. Smith). Children's Hospital Medical Center and Harvard Medical School, Depts. of Med. and Microbiology and Molecular Genetics, Boston, Massachusetts

Enterotoxigenic Escherichia coli (tox^+ E. coli) isolated from children in the Philippines are frequently resistant to multiple antibiotics. In bacterial conjugation experiments a heat-labile (LT) tox^+ E. coli 719B5 (O18 ab:H27) transfered resistance to streptomycin and tetracycline to an E. coli K12 recipient; all of the transconjugants selected for streptomycin resistance were also resistant to tetracycline and LT^+; ninety percent of transconjugants selected for tetracycline resistance were LT^+ but only 20 percent were resistant to streptomycin. Resistant and LT^+ transconjugants contained plasmids of approximately 120 and 60 megadaltons whereas resistant but LT^- transconjugants contained only one plasmid of 60 megadaltons as determined by agrose gel electrophoresis. In P1 transduction experiments resistance to streptomycin and tetracycline and the ability to produce LT were transduced together, suggesting that the genes coding for antibiotic resistance and enterotoxigenicity are on the same plasmid. Resistant streptomycin and tetracycline and LT^+ transductants contain plasmids of 60 megadaltons. These experiments suggest that the genes coding for LT production, and resistance to streptomycin and tetracycline are located on a 60 megadalton segment of DNA which may associate with a larger plasmid during bacterial conjugation.

766 DEFECTIVE OPSONIZATION OF SALMONELLA LIPOPOLYSACCHARIDE IN SICKLE CELL DISEASE. Ronald Field, Gary D. Overturf, University of Southern California, Department of Pediatrics, Los Angeles, California.

Children with sickle cell disease (SCD) have an increased incidence of S. pneumoniae infections which have been associated with defective opsonization of these organisms. Also, these patients appear to have an increased incidence of infections due to Salmonella species. We have evaluated the ability of SCD sera to support opsonization of S. enteritidis lipopolysaccharide (LPS) utilizing a method that employs emulsified LPS in paraffin oil containing oil red O dye. The amount of opsonized material can be quantitated spectrophotometrically and expressed as mg of paraffin oil ingested per 10^7 phagocytes.

Fourteen children with SCD and 9 age matched controls have been studied; the mean (range) of mg paraffin oil ingested per 10^7 phagocytes was .135 (.087-.174) and .162 (.136-.184) respectively. SCD patients had opsonization values of 57-104% of normal compared to control subjects which ranged from 89-113% of normal. Values >25% below normal, occurred only among SCD children; further, these children were all >4 years of age. No SCD child <4 years had values <82% of normal.

These studies suggest a subpopulation of SCD children with an age related impairment of opsonic activity for Salmonella LPS. This defect may contribute to susceptibility to infection with these organisms, and in conjunction with previously reported defects in opsonization for S. pneumoniae, suggests that in some patients with SCD, a generalized opsonic defect exists.

767 TYPE 14 PNEUMOCOCCAL ANTISERA IS OPSONIC IN VITRO AND PROTECTIVE IN VIVO FOR GROUP B STREPTOCOCCUS TYPE III Gerald W. Fischer, George H. Lowell, Martin H. Crumrine and James W. Bass. Department of Pediatrics, Uniformed Services University School of Medicine, Bethesda and Walter Reed Army Medical Center, Washington, DC.

The absence of type specific opsonic antibody in maternal and cord sera has been shown to be a major determinant for the development of invasive neonatal group B streptococcal (GBS) disease. Using immunoelectrophoresis we recently showed that antibody to Streptococcus pneumonia type 14 (SP14) reacted with the hot HCl extracted polysaccharide antigen of GBS type III (GBS III). The present studies were designed to determine if SP14 antibody is opsonic for GBS III in vitro and protective in vivo. Heat inactivated rabbit antisera against GBS III, SP14 and S. pneumonia type 3 (SP3) were opsonic for homologous organisms in a bacteriocidal assay using human neutrophils and complement. In addition, SP14 antisera was opsonic for GBS III. Preimmunization sera and antisera against SP3 were not opsonic for GBS III. Furthermore in an experimental rat model of neonatal GBS III sepsis, antisera to either SP14 or GBS III significantly increased survival when compared to controls. Naturally acquired immunity to GBS III may be related to the development of antibodies secondary to childhood pneumococcal infections. Since currently available pneumococcal vaccines have been shown to induce anti-SP14 antibodies, protection against neonatal GBS III disease may be possible by maternal immunization using these vaccines.

768 HEMOPHILUS INFLUENZAE TYPE b (HIB) INFECTION IN A DAY CARE CENTER (DCC): ERADICATION OF CARRIER STATE BY RIFAMPIN. Janet Gilsdorf, Dan Granoff, Charles Gessert, Millie Basden and Richard Rockwell. (Spon by M Grumbach). UCSF and Valley Medical Center of Fresno, California.

Two cases of invasive Hib disease (meningitis and cellulitis) occurred among 66 children attending a DCC (median age 20 mos). Nasopharyngeal (NP) colonization rates for Hib were 49% in the children, 8.8% in family contacts of culture-positive children, and 0% in staff. NP cultures were also obtained in two control populations: 62 children (median age 33 mos) attending a second DCC, and 58 children (median age 18 mos) attending Health Supervision Clinic (HSC). None of the children attending DCC-2 (0%) and 4 of the children attending HSC (6.9%) were positive for Hib. The isolates from DCC-1 were sensitive in vitro to trimethoprim-sulfamethoxazole. Nevertheless, therapy with this combination (8 and 40 mg/kg/day, respectively) for 7-10 days was ineffective in eliminating Hib carriage among 19 of 26 children (73%) attending DCC-1. In contrast, in a second clinical trial, treatment for 4 days with rifampin (10-20 mg/kg/day) eradicated Hib carriage in 19 of 22 culture-positive persons (86%). All 3 of the rifampin failures were associated with poor compliance. Rifampin was well tolerated with no serious side effects reported. When systemic Hib infection occurs in an enclosed population of young children, the risk of serious illness among contacts may be appreciably higher than in similar age children in the general population. Our data indicate that rifampin is effective in this clinical setting, and support the need for a controlled clinical trial.