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SEVERE AMEBIASIS IN EARLY INFANCY: A REPORT OF 3 CASES ASSOCIATED WITH HIGH PREVALENCE RATES AMONG FAMILY MEMBERS. Aubert C. Dykes, Trenton K. Ruebush, James D. Cherry, UCLA School of Med., Dept. of Peds., Los Angeles and The Center for Disease Control (CDC), Atlanta.

Two infants died with extraintestinal amebiasis, one on March 23, 1976 in Ga. and the other on July 1, 1976 in S.C. One infant in Ca. recovered after a long illness. Case 1: A 7-wk-old male infant in suburban Atlanta died of amebic peritonitis and a liver abscess. Stool samples from 17 of 19 family members revealed that 4 (24%) were infected with *Entamoeba histolytica*. Serospecimens from all 19 family members revealed that 9 (47%) had elevated indirect hemagglutination (IHA) titers to *E. histolytica*. Case 2: An 8-wk-old male infant in S.C. died with amebic colitis, peritonitis and liver abscess. Of 34 extended family members, 10 (32%) had elevated titers by IHA and 6 (23%) of 26 had *E. histolytica* organisms on stool examination. Case 3: A 12-wk-old female infant in suburban Los Angeles developed amebic colitis, peritonitis and a hepatic abscess, but recovered following surgery and chemotherapy. Two (40%) of 5 family members were infected with amoebae on stool examination. No history of travel or job exposure to sources of amoebae could be elicited from the families and environmental studies revealed no infective sources. Thus, the source and route of spread of *E. histolytica* within these 3 families could not be traced; however, person-to-person transmission was suspected. Of significance is that all 3 mothers were passing *E. histolytica* organisms and that the infection in these otherwise healthy infants was severe.

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BRONCHIOLITIS: A STUDY OF 1900 CASES IN A PRIVATE PEDIATRIC PRACTICE. F. W. Henderson, W. A. Clyde, A. M. Collier, F. W. Denny. University of North Carolina, Department of Pediatrics, Chapel Hill, North Carolina.

Data from an 11 year study of acute lower respiratory disease (LRD) were analyzed to develop a more complete understanding of the etiology and epidemiology of bronchiolitis. Cultures for viruses and mycoplasmas were obtained from all patients. The incidence of bronchiolitis was 11.4 cases/100 children/yr. in the first and second six months of life, declined 47% in the second year, and more gradually thereafter. Males were affected 1.32 times more frequently than females. Although cases occurred in most months of the study, the pattern of occurrence was distinctly epidemic. 22 epidemics were observed over 11 years; 60% of all cases occurred during epidemic months. RSV virus accounted for 11 outbreaks; parainfluenza viruses and *M. pneumoniae* were implicated in an additional 7. The impact of RSV infections on the occurrence of bronchiolitis varied with age; in children less than 2 years old 37% of all cases occurred in months when RSV was epidemic. In children age 5-10 years, *M. pneumoniae* was the single most frequent pathogen; the parainfluenza viruses as a group were recovered from an equal number of patients. The seasonal occurrence reflected the shifting etiology; cases peaked in mid-winter in children < 5 years old and in the fall in older ages. These data confirm the significance of RSV and delineate the roles of parainfluenza viruses and *M. pneumoniae* as causes of bronchiolitis, and have implications in the control of LRD. They also provide a foundation for studying the relationship of bronchiolitis to chronic airways disease, including asthma.

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NEONATAL RESPIRATORY SYNCYTIAL VIRAL (RSV) INFECTIONS. Caroline B. Hall, Arthur E. Konecman, R. Gordon Douglas, Jr. U. of Rochester Medical School, Pediatrics/Medicine, NY.

RSV infection appears to be uncommon in the first 4 weeks of life. If this is due to host immunity, environment or atypical illness is unknown. Nursery outbreaks have been described, but have mostly involved infants aged > 1 month. During a community RSV outbreak, nosocomial RSV infection was prospectively studied in our special care nursery to examine possible transmission to and infection in infants < 1 month old. Viral cultures were obtained q3d on all neonates and staff who were examined daily without knowledge of viral results. Of 82 neonates, 23 (29%) shed RSV for a mean 9d. Median age was 15d (range 6-77d); 20 were < 1mo. Signs tended to be atypical and nonspecific in younger infants < 3 wks (14) vs. those > 3 wks (9) who had more typical respiratory signs. Four (17%) died while shedding RSV, 3 unexpectedly, 2 after discharge. Of 53 staff, 18 (34%) contacted RSV and 83% were symptomatic. Infants in rooms with no infected babies, and in all types of beds, got RSV. These findings suggest RSV readily infects neonates, and staff may play a role in its spread. RSV infection in neonates may be missed due to atypical presentation, but may contribute to the morbidity and unexpected mortality in premature and other infants in special care nurseries. The reason for atypical illness in younger infants is unknown, but may be related to maternal antibody or other host immune factors.

Age in Weeks:	< 3	> 3
Pneumonia	7%	44%
Cough	14%	22%
URI	43%	55%
Apnea	14%	33%
Poor Feeding	29%	11%
Lethargy/ Irritability	43%	22%

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GENETIC AND EPIDEMIOLOGIC STUDIES IN FAMILIES WITH MULTIPLE DIABETIC OFFSPRING. M. Jaworski, E. Colle, R. Guttman, M. Belmonte, R. Poirier and J. Wilkins, Dept. Ped. & Med., McGill Univ., Dept. Ped., U. de Montréal, Montreal

Families with 2 or more insulin-dependent juvenile onset diabetics (JOD) were ascertained from an ongoing epidemiologic study. Patients and offspring in 15 families were HLA typed at the A and B loci. In 10/15 families, the diabetic offspring share 2 identical haplotypes. In about half of these families there is no previous history of diabetes and the possibility exists that the disease may be due to an autosomal recessive HLA-linked gene. In 4/15 families the diabetic offspring share only 1 HLA haplotype. This group includes a family in whom three members (a mother and two sons sharing haplotype A2B40) became diabetic within the same year, and another family in whom three siblings became diabetics by the age of 5 years. Diabetic siblings share no HLA haplotypes in only 1 family. This distribution differs from that predicted by the null hypothesis of random assortment of HLA-haplotypes. The data suggest genetic heterogeneity of JOD as well as a linkage between the HLA region and gene(s) predisposing to the development of some types of JOD. No A/B recombinants were found among the 31 diabetics and 12 unaffected siblings, nor among the 7 offspring tested at the D locus by mixed lymphocyte culture. Comparison of the familial diabetics with the total JOD patients ascertained since 1971 showed in both a higher incidence in the winter months and a clustering of cases at around age 11 years. However, in the families there is an increased number of children in whom the diagnosis was made before the age of 6 years.

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MECONIUM SCREENING FOR CYSTIC FIBROSIS. Frances L. Harley, Beth Marshall and Ernest E. McCoy. Dept. Ped. Univ. of Alberta Sch. Med., Edmonton, Alberta.

Meconium Screening for CF based on increased albumin content in the meconium of CF newborns is used in Europe for early detection. A recent collaborative study in U.S. centres did not find BMC-test meconium screening, done at the bedside as currently marketed, wholly satisfactory. We wish to report results of a 2 year "centralized lab" study involving 30,000 infants born up to 400 miles distant from the laboratory in 20 rural and urban hospitals. The project was managed by a nurse who visited each hospital, gave instructions on collection, freezing and shipment of meconium samples. A central lab tested all samples first with BMC strips. All BMC positive samples with an adequate quantity of meconium had albumin quantitated by radial immunodiffusion (RID) technique. Sweat chlorides were requested on all babies with > 10 mgm albumin/gm wet weight meconium and on those 30 whose BMC was positive but meconium insufficient for quantitation. Results to 10/30/1977: Infants tested 30,851. Samples lost or not collected = 251 (0.81%). BMC pos (+) = 124 (0.4%). BMC +, RID + or RID nsq = 85 (0.27%), study pos group, BMC +, RID + = 49/94, BMC +, RID - = 45. Sweats incomplete = 7. Infants "study +", sweat + = 7. Infant "study -" sweat + = 1. Meconium collected 48 hrs post op from meconium ileus pt. nsq for test was sweat +. We conclude that BMC-test meconium performed in a central lab with albumin quantitation for positive strips is a suitable CF screen for both urban and rural populations, acceptably specific and selective, and can be done at reasonable cost.

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INFLUENZA A AND B VIRUS INFECTION IN INFANTS AND YOUNG CHILDREN DURING THE YEARS 1957-1976. Hyun W. Kim, Carl D. Brandt, Robert M. Chanock, Robert H. Parrott. Children's Hospital National Medical Center, George Washington Un. Washington, D.C., NIAID, Bethesda, Md.

Influenza A virus activity was demonstrated in infants and young children from metropolitan Washington, D. C. during each of 19 successive August-July respiratory disease years, and during 17 of these years at least two per cent of hospitalized respiratory disease patients yielded an influenza A or B virus and/or showed an influenza A or B serum complement fixing (CF) antibody response. Between October 1957 and July 1976, 14.3 per cent of 860 croup patients and 5.3 per cent of 5,655 hospitalized respiratory patients showed evidence of influenza A or B virus infection. Serious illness with influenza A virus was 4.5 times more common than with influenza B virus. Influenza A virus infections were seen more than twice as frequently during the era of the H3N2 virus subtype as during the H2N2 era. Patients with serious influenza A virus infections were especially likely to have croup (particularly during the H3N2 era), to be seen during December through February, and to be black male infants. During the peak month of a composite of 13 consecutive influenza A virus outbreaks, influenza A virus infection was demonstrated in 67.6 per cent of croup patients and 35.6 per cent of all hospitalized respiratory patients. During the peak month of a composite of 6 consecutive influenza B virus outbreaks, influenza B virus infection was demonstrated in 36.0 per cent of croup patients and in 10.8 per cent of all hospitalized respiratory disease patients.