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experimental rats demonstrate mumps virus antibody and delayed hypersensitivity.

Gestational viral infection may alter growth by a direct replicative effect on fetal tissues or perhaps indirectly via dysfunction of the placental unit.

99 Cytomegalovirus Infection of the Female Genital Tract.
Donald N. Medearis, Jr., Robert Montgomery and Leona Youngblood, Children's Hosp. of Pittsburgh and Dept. of Ped., Univ. of Pittsburgh Sch. of Med., Pittsburgh, Pa.

Infections of the female genital tract may play an important role in intrauterine or neonatal cytomegalovirus (CMV) infections. Serum, urine (U) and cervical swab specimens (CSS) were obtained from 71 unselected pregnant Navajo women in Fort Defiance Indian Hospital, and 125 women of the prenatal clinic of Magee-Women's Hospital of Pittsburgh. Cord blood and urine specimens were obtained from 81 newborn infants delivered at Fort Defiance during the same period that the mothers were studied. CMV was recovered from 9/64 CSS of Navajo women (14%) and from 3 of 68 (4.4%) U. Virus was recovered from 5 of 112 (4.5%) CSS women in Pittsburgh and from 3 of 104 (2.9%) U. Recovery of CMV was more frequent from primigravidas (7/62 CSS; 4/57 U); 43 CSS and 41 U from women who had had more than 4 pregnancies were negative. CMV was recovered from 2% of CSS taken in the first trimester, 7% of those taken in the second trimester and 12% of the third trimester specimens from the combined population. None of the 66 urine specimens obtained from newborn infants was positive. Herpes virus was not recovered. These data revealed a surprisingly high frequency of cytomegalovirus infection of the cervix and although the differences were not statistically significant (presumably due to the small numbers studied) they suggest the possibility that infection was more frequent in young women, in primigravidas, and late in pregnancy. (Supported in part by NIH grant HD 02135.)

100 Transmission of Toxoplasmosis by Leukocyte (WBC)
Transfusion. STUART E.SIEGEL, MILFORD N.
LUNDE, ARTHUR S.LEVINE and ROBERT G.
GRAW, Jr., NCI and NIAID, NIH, Bethesda,
Md. (introduced by Robert W. Miller.)

Disseminated toxoplasmosis infections without an obvious source have been documented frequently in cancer patients. Two children with acute leukemia given WBC transfusions for granulocytopenia from a donor with chronic myelogenous leukemia (CML) developed toxoplasma gondii infection. The CML donor, with no history of toxoplasma infection had received no anti-leukemic therapy prior to leukaphe-resis and remained clinically well. The first child was given 0.35×10^{11} CML leukocytes. Three weeks later she developed skin rash, pneumonia, congestive heart failure, hepatitis, and seizures, accompanied by a rising toxoplasma dye titer to 1:8,000 (IgM = 1:160). She expired 3 months later with disseminated toxoplasmosis. The second child received 2.5×10^{11} leukocytes over a 7-day period four weeks prior to splenectomy for an E. coli splenic abscess. Concomitantly, her dye titer rose to 1:128 (IgM = 1:40). Toxoplasma gondii was isolated from the spleen and a single toxoplasma cyst was found on histologic examination. She died 1 month following surgery with toxoplasma cysts found in the lung and heart at necropsy. Retrospective examination of stored serum samples from the CML donor

revealed that at the time of WBC donation she had a toxoplasma dye titer of 1:8,000 (IgM = 1:10, CF = 1:16). Leukocytes from the same donor were given to other leukemic patients. Both died within one week of transfusion without pathologic evidence of toxoplasma infection. Toxoplasma infection in two patients receiving leukocyte transfusions from a CML donor with serologic evidence of toxoplasmosis suggests the role of WBCs in the transmission of this disease.

101 Splenectomy and Impaired Resistance to Pneumococcal Infection. ROBERT H. DRACHMAN, LAI-SUNG LEUNG and GREGORY SZAL, Johns Hopkins Univ. Sch. of Med., Dept. of Ped., Baltimore, MD. Efforts to devise an animal model in which splenec-

tomy decreases the effectiveness of host defences against acute bacterial infection have been infrequently successful. It has been difficult to provide a challenge which sharply differentiates controls from the operated animals. A model is described in which all splenectomized rats died after intravenous (i.v.) challenge with an LD₅₀ dose of pneumococcus Type 25. Normal rats immunized with pneumococcus Type 25 formalized bacteria and then challenged i.v. 3 to 6 days afterwards, all survived, while previously splenectomized rats, similarly immunized, all died. As the interval between immunizations and subsequent splenectomy lengthened in a stepwise fashion, the mortality following i.v. challenge fell from 80% to zero. Clearance of pneumococci from the blood stream reflected these results, more prompt disappearance of bacteria becoming evident at I to 3 hours after i.v. challenge in those most likely to survive. During this period of increasing antibacterial resistance both early antibody and the unique ability of the spleen to clear minimally opsonized bacteria were critical for effective antibacterial host defence. Early but delayed antibody could be detected in the immunized splenectomized rat, but was not adequate for protection in the absence of the spleen.

102 Antibody Response to a Polyvalent Pseudomonas Vaccine in Children with Leukemia. Mahroo Haghbin, Lowell S.Young, Donald Armstrong and M. Lois Murphy, Memorial Hosp. and Cornell Univ. Coll., New York, NY.

Pseudomonas aeruginosa infection accounts for 30% of gram-negative bacteremias in children with acute leukemia at Memorial Hospital. There is a mortality rate of 87% despite antimicrobial therapy. Previous studies in animals and man following severe burns suggest that P. aeruginosa infection may be subjected to immunological control. Active immunization of 20 leukemic children in bone marrow remission was attempted with a lipopolysaccharide antigen derived from 7 strains of *P. aeruginosa* (Fisher-Devlin immunotypes). Vaccination consisted of 4 intramuscular injections at weekly intervals. Febrile and local reactions occurred in all, but were not severe enough to discontinue the procedure. The appearance of antibody was demonstrated by 1 to 5 precipitin bands using the Oucterlony immunodiffusion technique. This was correlated with a rise in hemagglutinating antibody titers. Sixteen children demonstrated antibody response within one month, despite the immunosupressive antileukemic therapy that they were receiving. If this antibody proves to be protective against P. aeruginosa infection by further control studies in progress, vaccination of patients early in the course of leukemia would be indicated.