tation of motion, mild tenderness or swelling, and in two cases, synovial thickening. Two other children had reactive tests for rheumatoid factor. The mean rubella HI titer 6–9 months after vaccination was 1:66 among the 11 children with recurrent symptoms. This was lower but not significantly different from the mean HI titer (1:127) of sera collected at the same time from 18 children who had joint symptoms only during the first 45 days following vaccination.

Recurrent joint symptoms may rarely be observed following administration of HPV-77DK12 rubella vaccine. Serologic studies failed to distinguish children with recurrent joint symptoms from controls.

Protective effect of antirubella human immunoglobulin. LOUIS Z. COOPER, JOAN P. GILES, ALFRED L. FLORMAN, PHILIP R. ZIRING, and SAUL KRUGMAN. *New York Univ. Sch. of Med., N.Y., N.Y.*

Previous experience with immune serum globulin (ISG) indicated 1) that it did not prevent viremia in children with rubella infection, and 2) that it did not prevent congenital rubella. This report describes the protective effect of an experimental lot of high titer antirubella human immunoglobulin (RIG) in 33 children exposed to the Brown strain of rubella virus (RV).

RIG was given to 22 of the susceptible children 24 or 96 hours after intranasal exposure to RV. Six other children received ISG. The dose of RV was either 10^4TCInD$_{50}$ or 10^1TCInD$_{50}$, and the dose of immunoglobulin was 0.3 ml/kg of body weight. In the group that received RIG: 1) passively acquired rubella antibody was detected transiently after inoculation in 15 children; 2) no detectable viremia was observed; 3) pharyngeal shedding of RV was decreased and 4) the rubella specific IgM response was depressed. RIG was more effective when given at 24 hours against low dose virus challenge, preventing or delaying seroconversion; it was less effective in modifying infection when given at 96 hours after high dose challenge. These data suggest that RIG may be useful for the prevention of congenital rubella.

Serologic responses to live further attenuated rubeola vaccine among term and low birth weight infants. J. WILKINS, P. F. WEHRLE, and B. PORTNOY. *Hastings Found. Univ. Southern Calif.-Los Angeles County Med. Ctr., Los Angeles, Calif.*

Protective antibodies against rubeola virus have been demonstrated in IgG and passive transfer of maternal antibodies against rubeola has been known to protect infants during the first several months of life. It has also been shown that the presence of this passively acquired antibody interferes with effective immunization against rubeola with live attentuated virus vaccines. For all infants there is a strong correlation between initial level of passively acquired antibody of maternal origin and its persistence. The concentration of IgG at birth is lower in low birth weight (LBW) than in term infants. This suggests that early curtailment of interuterine life is likely to result in lower IgG levels in the infant. From August 1, 1965 through March, 1968 251 infants (114 terms and 151 LBW) were inoculated with further attenuated rubeola vaccine at varying ages between six months and two years. No detectable rubeola HAI antibody was present at $<1:8$ prior to inoculation. The data indicate that the serologic responses of the two groups are better than previously recognized. Thus, the effectiveness of rubeola immunization of infants <12 months of age is such that use of this vaccine may be considered in younger infants during epidemic situations or in immunization programs in developing countries.

The acquisition of antibodies against adeno-associated satellite viruses. MARTHA D. YOW, LARRY H. TABER, JOSEPH L. MELNICK, and D. WARK BOUCHER. *Baylor Coll. of Med., Houston, Tex.*

The adeno-associated satellite viruses were first recognized in 1965. In spite of significant investigations since that time, their role in human disease remains obscure. In an attempt to define this role a longitudinal study of 42 infants (birth–2 yrs.) from a low socioeconomic group was initiated in 1968. The infants were examined at regular intervals and during illnesses. Specimens for viral isolation were collected on each occasion. Sera for antibody titers were obtained at birth and every 3 months thereafter. The purpose of this paper is to report the results of the serologic portion of this study. Neutralization tests revealed that at birth 50% of the infants had antibody to Type 1 satellite virus and 71% to the Type 2-3 complex. These percentages declined to 9% and 18%, respectively, at 6 months, remained low from 6 to 12 months, then rose sharply at 15 months. At 2 years 66% of the infants had antibody against Type 1 and 73% against Type 2-3. Mean geometric titers were high at birth, low from 6–12 months, and highest at 24 months. These data indicate the passive transfer of maternal antibody, and its decline. They also indicate infection due to adeno-associated satellite viruses early in life.

Parotitis in previous recipients of mumps vaccine. PHILIP A. BRUNELL, ANTHONY BRICKMAN, SHARON STEINBERG, and ELAINE ALLEN. *New York Univ. Sch. of Med., N. Y., N. Y.*

Between 1967 and 1970, twenty children with parotitis who had previously received mumps vaccine were studied. These children were immunized in four different Pediatric offices over a four year period so that no single lot of vaccine or immunizing procedure could be implicated as the cause of these apparent "vaccine failures." Mumps soluble complement fixing (MSCF) antibody determinations revealed that only eight of the seventeen children for whom appropriate serum specimens were available had evidence of mumps infection. Children without a detectable antibody response could not be differentiated clinically from those who developed MSCF antibody. Parotitis occurred in two siblings without a MSCF antibody response suggesting that these illnesses might have an infectious etiology. Neither mumps nor any other virus could be recovered from five children without an antibody response or from three children who could not be classified serologically. These findings confirm the multiple etiology of parotitis and indicate that serologic studies are required to identify true mumps vaccine failures.

Host resistance to virus infection in the fetus: I. Interferon (IF) production. J. C. OVERALL, JR. and L. A. GLASGOW. *Univ. of Utah Coll. of Med., Salt Lake City, Utah.*

The mechanisms underlying impaired host resistance of the fetus and newborn infant to virus infections are poorly understood. Immaturity of the IF system has been suggested as one major determinant of this enhanced susceptibility. Our previous studies demonstrated that the third trimester (140–117 days) fetal lamb (normal gestation 150 days) produced markedly higher levels of serum IF (27,000–250,000 units/ml) than adult sheep (180–250 μ/ml) following intravenous (IV) inoculation with Chikungunya virus (CV). The present report extends these studies to fetuses in the second and first trimester, the period during which greatest damage from virus infection occurs. Second and third trimester fetuses were inoculated by the IV, and first trimester by the intraperitoneal route. Highest IF titers (μ/ml of serum or gram of tissue) were present 2–4 hours following inoculation:

	Gest. Age							
	111 day	109 day	97 day	84 day	68 day	43 day	30 day	30 day
IF Titer	80,000	200,000	400,000	200,000	150,000	1,000	800	2,000

During the second trimester, fetal animals (68–97 days) continue to produce high levels of IF. Although fetal immaturity is the likely explanation for the significantly lower levels of IF observed in the first trimester animals, the route of inoculation could be a factor. The data clearly indicate, however, that the capacity to produce interferon is present during early stages of fetal development.

The effect of early folic acid deprivation on later growth and susceptibility to Shigella infection. JOHN D. NELSON and KENNETH C. HALTALIN. *Univ. of Tex. (Southwestern) Med. Sch. at Dallas, Dallas, Texas.*

Early, brief environmental factors may have lasting effects on growth rate, adult size and responses to stress. Nutritional deprivation is one of these events. Newborn guinea pigs fed a folic acid-deficient diet for 14 days are susceptible to fatal infection with *Shigella flexneri* while optimally-fed control animals are resistant. (J. Infect. Dis. 121:275, 1970). To test the hypothesis that animals subjected to a brief period of folic-acid deficiency as newborns would show enhanced susceptibility to Shigella challenge later in life after resuming a normal diet, newborn guinea pigs randomized for sex and litter were assigned to a control group receiving optimal diet or to a test group fed the folate deficient diet for 14 days and subsequently given optimal diet. 27% of animals (9/33) receiving deficient diet died unexpectedly and without obvious cause shortly after being offered standard diet. Possibly metabolic readjustments led to fatal biochemical derangements. At 45, 75, 105 or 135 days of age when selected animals were challenged with Shigella, there was only transient fecal shedding and no differences existed between the test and control groups. There was a profound and lasting effect of early folic acid deprivation on subsequent growth. Between 125–149 days of age the average weight of the control group was 856g and of the early-deprivation group 649g. Aside from smaller body size the early-deprivation animals appeared healthy. Although previous studies have shown similar growth retardation from early periods of protein or caloric restriction and from incomplete protein feeding, it has not been noted previously with a specific dietary deficiency such as folic acid.

The effects of corticosteroids on experimental herpes simplex virus encephalitis. LAURIE N. ECKMAN and JOHN F. GRIFFITH (Intr. by Samuel L. Katz). *Duke Univ. Med. Ctr., Durham, N.C.*

This study examines the effects of corticosteroids on the ultimate survival, virus concentration and neuropathologic changes in mice with herpes simplex virus (HSV) encephalitis.

Swiss white mice were inoculated intracerebrally with 0.01 ml of a 5×10^{-4} dilution of stock Rodanis strain HSV. Virus was injected directly into the right cerebral cortex. Treatment was initiated 60 hours after inoculation of virus with either hydrocortisone, corticosterone or placebo. One group of animals was inoculated with virus and left untreated and another group was retained as an uninfected control population. Both preparations were given as 5 mg intramuscular injections every 24 hours for 5 days.

Fifty percent of the infected, untreated animals and an identical proportion of those treated with placebo, died with encephalitis. In the steroid treated group sixty percent of those receiving hydrocortisone died as compared with fifty percent receiving corticosterone. The differences were not significant.

The study relating virus concentration in brain and neuropathology to serum adrenal corticosteroid levels was done using an identical population of animals with the same inoculum. Groups of 8 animals were sacrificed by decapitation at intervals before and after institution of therapy. The blood was pooled and the plasma separated and stored at −70°C. Steroid levels were measured using the fluorimetric method. Preliminary results revealed a level of 90 μgm/100 ml for hydrocortisone treated animals and 13 μgm/100 ml for untreated animals. Two brains from each of these groups were aseptically removed and made into a 10% suspension for plaque assay. The results will be presented and discussed, stressing the survival data and the interrelationship between steroid concentration and prognosis in this illness.

Intrinsic defect of the polymorphonuclear leukocyte resulting in impaired chemotaxis and phagocytosis. RUTH L. STEERMAN, RALPH SNYDERMAN, SANFORD L. LEIKIN, and HARVEY R. COLTEN. *Children's Hosp., Washington, D.C., NIDR, Bethesda, Md., NCI, Bethesda, Md.*

Abnormalities of polymorphonuclear leukocyte (PMN) chemotaxis and phagocytosis have been described. In each instance the combined chemotactic and phagocytic defects were a consequence of an abnormality in humoral, not cellular, function. We now describe a 3½ year-old white male with recurrent pneumonia and skin infections associated with a *cellular defect* of PMN chemotaxis and phagocytosis, as well as a sex-linked form of congenital agammaglobulinemia. He had normal delayed hypersensitivity and normal levels of hemolytically active whole complement and C1 thru C5. The impairments of PMN function were demonstrated *in vitro* by an inability of his PMNS to respond to chemotactic factors, to phagocytize *S. aureus*, and by abnormal NBT dye tests. These abnormalities were not corrected by the addition of normal serum to the patient's PMNs. No evidence was obtained for a humoral inhibitor of PMN function since normal PMNs were capable of normal chemotaxis and phagocytosis in the presence of his serum. In addition to the intrinsic granulocytic defect, the patient's serum was deficient in generating chemotactic activity for normal PMNs. The parents' PMNs were capable of normal chemotaxis and phagocytosis. It is not certain whether the patient's PMN abnormalities are the result of an acquired or an inherited defect. These studies demonstrate a previously unrecognized abnormality of PMN function, namely, an intrinsic defect of the PMN resulting in impaired chemotaxis and phagocytosis.

Hemorrhagic chickenpox associated with disseminated intravascular coagulation. JAMES J. CORRIGAN, JR. and W. LORRAINE WATKINS. *Univ. of Arizona, Coll. of Med., Tucson, Ariz. and Emory Univ. Sch. of Med., Atlanta, Ga.*

Disseminated intravascular coagulation (DIC) or consumption coagulopathy has been well documented in children with bacterial septicemia but infrequently reported in viral diseases. Fibrin thrombi have been noted with the malignant form of varicella-zoster virus infections and there is laboratory evidence that DIC may be present in hemorrhagic smallpox. It is the purpose of