	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