α-fetoprotein concentrations in the rat normally declined abruptly after birth to approximately half of the prenatal level by 2 to 3 days of age, in accord with the loss of fetal membranes at delivery; the α-fetoprotein level then remained relatively constant until the rat was 6 to 8 days of age, after which synthesis of the protein was increasingly suppressed. Marked suppression of α-fetoprotein synthesis in the rat could be induced in the first week of life either by cortisone or by sham operations; epinephrine, corticosterone, testosterone, progesterone and estradiol had no observable effect on synthesis. Participation of the adrenal in the suppression noted to follow surgery was indicated by the observation that adrenalectomy did not inhibit α-fetoprotein synthesis. Subcutaneous injection of cortisone into the pregnant rat suppressed α-fetoprotein synthesis in the fetus *in utero* as did sham operations on the pregnant rat. (APS)

65 *Cord Blood Gamma M as a Screening Test for Congenital Viral Infections.* JOHN L. SEVER and HEINZ W. BERENDES*, NIH, Bethesda, Md.

Elevated levels of gamma M have been found in newborns with a number of congenital infections including syphillis, toxoplasmosis, rubella, and cytomegalic inclusion disease (CID). The frequency of this finding in normal and infected children was studied with specimens from the Collaborative Perinatal Research Study. Cord sera from 1000 children at 10 collaborating institutions were tested. 29 had elevated gamma M; 14 of these children had abnormalities including unexplained jaundice with hyperbilirubinemia; mental and motor retardation; hepatosplenomegaly; skeletal malformations; cataracts and strabismus with nystagmus; failure to thrive; and other significant findings. One of these children had congenital toxoplasmosis. Tests of children with congenital infections showed high cord blood gamma M for rubella (6 of 9), CID (2 of 3), toxoplasmosis (2) and generalized herpes (1). Maternal infections also were associated with high gamma M in the cord of children for rubella in the first trimester (9 of 37,6 of these were abnormal), and serological evidence for maternal toxoplasmosis (4 of 5,1 child abnormal). Other maternal infections during pregnancy did not result in significant elevation of cord gamma M including varicella (6), mumps (14) and rubeola (17). There was no elevation of gamma M in children with erythroblastosis (6), congenital leukemia (3) and mongolism (19 of 20). One mongoloid child had high gamma M and bronchial pneumonia and peritonitis. Only 1 of 36 children with congenital heart disease had high gamma M, and this child had congenital rubella. The simple gel diffusion determination of gamma M in cord blood and in the newborn should be useful as an initial screening test when considering congenital viral infections. (SPR)

66 *Immunologic Consequences of Congenital Rubella.* LOUIS Z. COOPER*, STEBBINS B. CHANDOR*, ALBERT B. OCKERSE*, DONALD FEINSTEIN* and SAUL KRUGMAN, New York Univ. Sch. of Med., New York, N.Y.

The immunologic consequences of maternal rubella have been correlated with the clinical and virologic data accumulated on 350 children followed since the 1964 epidemic. Mothers and their infants with rubella-associated defects have had persistence of rubella serum neutralizing and hemagglutination-inhibition antibodies. Antibody titers among these children have remained at levels ≥ to those in their mothers. In contrast, most children who are clinically normal, despite maternal rubella, have not produced rubella antibody. This relationship of fetal infection to congenital defects and persistent antibody production is supported by a study of 3 sets of twins; 5 of the children have anomalies and antibody, 1 child is normal and has no antibody. Alterations in serum immunoglobulin levels most commonly elevations of IgM and in one instance production of a small molecular weight (approximately 7S) IgM, and a decreased incidence of positive skin reactions to oidiomycin indicate that congenital rubella produces a spectrum of immunologic abnormalities similar to that which it produces in other organs. (SPR)

67 *Cells of Human Colostrum: In Vitro Studies.* CLIFTON W. SMITH* and ARMOUND S. GOLDMAN*, Univ. Tex. Med. Br., Galveston, Tex. (introduced Warren F. Dodge).

The types and behavior of human colostral cells both *in vivo* and *in vitro* were studied. Samples obtained from thirty individuals consistently revealed neutrophils, lymphocytes and macrophages. The relative frequency of these cells varied with time following delivery. The most abundant cells were macrophages.

Cultures in Leighton tubes without phytohemagglutinin (PHA) revealed many macrophages and vacuolated cells resembling colostral corpuscles. These two cell types could not be clearly separated morphologically. Both types adhered to glass surfaces; however, only those typical-appearing macrophages showed ameboid motion. Cell cultures with PHA uniformly displayed all stages of lymphoblastic transformation. Synthesis of deoxyribonucleic acid by these lymphoblasts was evidenced by radioautography of cells previously exposed to thymidine-H^3. It is concluded that living lymphocytes and macrophages are constituents of normal human colostrum. Studies of the immunologic functions of these cells will be described. (Supported by NIH Grant 5 RO1 HD 00735-03) (SPR)

68 *Specific Local Antibody Defect in Chronic Mucocutaneous Candidiasis.* RICHARD A. CHILGREN*, RICHARD HONG and PAUL G. QUIE, Univ. of Minn. Sch. of Med., Minneapolis, Minn.

Immunological defense mechanisms were studied in 3 patients with chronic mucocutaneous candidiasis of at least 9 years duration. None had systemic candidiasis or increased susceptibility to other infections.

Agglutination titers of standardized suspensions of heat-killed *Candida albicans* were measured in concentrated parotid duct saliva samples. Two patients' samples contained no agglutinating antibodies and one had a titer of only 1:4, in spite of gross oral infection. In contrast, parotid fluid from 6 patients who had recovered from *C. albicans* oral infection had titers ranging from 1:16 to 1:400. Despite the absence of agglutinating antibodies to *C. albicans*, isohemagglutinins and sheep cell agglutinins were present, and a normal immunoglobulin pattern was found (IgA present in normal amounts, IgG and IgM not detected).

The patients' sera, however, contained levels of agglutinating antibody ranging from 1:64 to 1:256 and had normal amounts of immunoglobulins. These findings are consistent with the known lack of correlation between local and circulating antibody levels and further suggest a specific deficiency in local antibody response to Candida.