congenital malformations at Michael Reese Hospital; 3. a similar clustering of chromosome abnormalities in newborns in Denver, Colorado (PUCK and ROBINSON, Science [1965]) and in spontaneous abortions in London, Ontario (CARR, personal communication). For the second cluster there was a corresponding increase in the abortion population in Canada. There was no statistically significant increase in the reported incidence of 9 infectious diseases including rubella or infectious hepatitis, relative to the three clusterings defined in the newborn population. Prospective studies on the distribution of chromosome aberration in time have been conducted since November, 1964, and reveals that when the spontaneous abortion and newborn populations are considered as one conceptual population, the distribution of chromosome aberrations in time appears random. These findings indicate that either the factors responsible for nonrandom distribution of chromosome aberrations were not operating during the time period covered or that onrandom distribution of chromosome aberrations does not occur in the human population. (APS)

30 *Chromosomes of Couples with Repeated Spontaneous Abortions.* R.J. MCKAY, Jr., W.E. HODGKIN* and E.H. WITTE*, University of Vermont College of Medicine, Burlington, Vt.

Because a 20–25 % incidence of chromosomal abnormalities has been reported among spontaneously aborted fetuses, it was decided to study the chromosomes of couples who have had 3 or more spontaneous abortions. Using a commercial kit method for culturing peripheral blood leukocytes, approximately 750–1000 metaphases were scanned in each patient in order to obtain 50 satisfactory for counting. For each patient, all metaphases with abnormal counts and 5 with normal counts were photographed and karyotypes made. Among 4 couples with repeated abortions and one or more children with multiple anomalies, 2 husbands showed a balanced translocation in all metaphases studied. Among the other 38 couples studied a number showed abnormalities in a few cells (trisomy, partial trisomy, balanced and unbalanced translocations, fragments, dicentrics, endoreduplication, tetraploidy, XO, long-armed Y, quadriradials and triradials). Partially trisomic cells were seen approximately 5 times as frequently among the women as among the men, and 6 patients were observed to have a single metaphase with a quadriradial chromosome. (APS)

31 *The 'Impotent Neutrophil' Syndrome.* L.L. KAHLE.* H- MORENO*, and E. KAUDER*, Dept. of Pediat. Univ. of Cincinnati, Cincinnati, O. (introduced by A.M. Mauer).

Leukocyte function was studied in a 2–2,5-year-old Negro male with the history of repeated abscesses, cervical and inguinal adenitis, recurrent pneumonia, seborrheic eczema of the scalp, hepatosplenomegaly and anemia. An appropriate leukocytosis with neutrophilia occurred with each infection, absolute neutrophil counts ranging from 3,900 to 24,500/mm^3. IgG, IgA and IgM globulins were present in increased amounts. Lymph node biopsy contained caseating granuloma. Skin tests and cultures for all types of acid fast bacilli and fungi were repeatedly negative. These findings are characteristic of the syndrome of chronic granulomatous disease of childhood. There was a normal flux of leukocytes into Rebuck skin windows and exudate fluid. Leukocytes appeared normal with alkaline phosphatase, peroxidase and Wright's stain and were normally vacuolated after bacterial phagocytosis. Normal leukocyte motility and phagocytosis followed by degranulation were seen with phase microscopy. Decreased bactericidal activity of the patient's leukocytes during in vitro incubation with Staphylococcus aureus and Aerobacter aerogenes was repeatedly demonstrated in the presence of the patient's serum or normal serum. To determine the nature of this defect a citric acid extract of the patient's leukocyte granules was compared with control specimens and decreased bactericidal activity was found. The deficient or abnormal phagocytin activity of the patient's leukocytes, demonstrated by this study, could account for his clinical syndrome. (SPR)

32 *Defective Lymphocyte Response to PHA in Congenital Rubella.* J.R. MONTGOMERY*, M.A. SOUTH*, W.E. RAWLS* and J.L. MELNICK*, Depts. of Ped., Med., and Virology, Baylor Univ. College of Med.; and G.B. OLSEN*, P.B. DENT*, and R.A. GOOD: Ped. Res. Labs., Variety Club Heart Hospital, U. of Minn., Minneapolis, Minn. (introduced by Martha D. Yow).

Persistent viral carrier state in congenital rubella remains an enigma. Defective cellular immune mechanisms may play a role in this persistence. To investigate this possibility the response of leukocytes to phytohemagglutinin (PHA) was studied. 2×10^6 peripheral leukocytes were cultured in routine media. PHA was added to achieve a concentration of 0.025 ml/ml of media. Quadruplicate cultures were incubated at 37° C for 72 h and treated with C^{14} labeled thymidine for 5 h prior to termination. Cellular response was estimated by measuring the cellular content of C^{14} in a standard liquid scintillation system. A decreased PHA responsiveness of leukocytes was demonstrated in 8 of 14 congenital rubella patients studied, despite evidence in these patients of normal delayed hypersensitivity. PHA response returned to normal later in the course of each patient. To test whether this lack of responsiveness is due to an intrinsic defect in lymphoid cells from these patients or to direct effects of rubella virus, a normal adult's leukocytes were cultured with rubella virus. PHA response was dramatically reduced in every experiment; this effect could be eliminated by pretreatment of the virus preparation with rubella neutralizing antibody. Inhibition was also produced when Newcastle disease virus was substituted for rubella virus. These studies indicate that leukocytes from some congenital rubella patients show a defective response to PHA. The defective response can be reproduced in normal leukocytes by the addition of rubella virus or Newcastle disease virus *in vitro*. The prolonged persistence of virus and the defective reactivity to PHA in babies with congenital rubella may be interrelated. (SPR)

33 *A Mitotic Inhibitor Produced by Rubella Virus Infection of Human Fibroblasts.* STANLEY A. PLOTKIN, Wistar Institute and Department of Pediatrics, University of Pennsylvania, Philadelphia, Pa.

When infected with rubella virus, many human diploid fibroblast cell strains show mild to marked degrees of mitotic inhibition (PLOTKIN *et al.*: Amer. J. Epidem. *81:* 71 [1965]); cell strains derived from infected fetuses divide more slowly than normal (RAWLS and MELNICK: J. exp. Med. *123:* 795 [1966]); and infants congenitally infected with rubella show mitotic