Demonstration of Herpes Simplex Virus in Fetal Rabbits[1]

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Winsser, *et al* (1957) demonstrated a viremia stage in a newborn whose mother suffered poliomyelitis shortly before childbirth and Schaeffer, *et al* (1954) isolated the poliovirus from the aborted fetus of an infected mother. In a study of the dissemination of herpes simplex virus Biegeleisen, *et al* (1957) demonstrated the virus in specimens of blood

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from rabbits following inoculation of this infectious agent on the scarified cornea.

This report deals with the isolation of herpes simplex virus from the fetuses of rabbits which had been experimentally infected with the virus on the scarified cornea.

Fourteen days after breeding, six white female rabbits were infected with the HF strain of the virus by the corneal scarification method. Following inoculation, one ml. of blood was removed aseptically from the marginal ear vein of each rabbit at 24, 48 and 54 hours and every 12 hours thereafter for 180 hours. The presence of the virus in the specimens of blood and tissue homogenates (described below) was determined by the production of lesions on the chorioallantoic membranes (CAM) of developing chick embryos.

Fifty-four hours following inoculation of the rabbits, one gravid horn of the bifurcate uterus was surgically removed. The other horn was left intact so that each rabbit might deliver. The fetuses from each rabbit were dissected from the excised horn and washed thoroughly. A homogenate of fetuses of each rabbit was made with buffered gelatin saline and 0.1 ml. was inoculated onto each of eight CAM. After incubation, the membranes were harvested and examined for herpetic lesions.

Typical herpetic lesions were observed on the CAM inoculated with specimens of blood and those with fetal homogenates from each of the six rabbits. The isolated virus was identified as herpes simplex by comparative titrations using specific immune serum. The infectivity of the virus for CAM was decreased in the presence of the antiserum.

One of the infected mothers delivered three offsprings and at this time developed a severe recurrent herpetic conjunctivitis in the previously infected eye. She died within 48 hours following a central nervous system involvement. Exudate from the infected eye was inoculated onto CAM and typical herpetic plaques were observed. Organs from this rabbit and the newborn, all of which died, are being investigated. It is possible that the natural birth process may have played a triggering role in the production of the recurrent infection.

The demonstration of herpes simplex virus in the blood of these animals confirmed previous results of Biegeleisen, *et al* (1957) and indicates that the virus was probably transported to the placenta by the blood. Zuelzer and Stulberg (1952) reported a viremia in infants who succumbed to overwhelming herpetic infections. Thus, the role of a viremia in the infecton of fetuses of herpes simplex infected rabbits could prove valuable in discovering another possible method of neonatal infection. In view of the report of Gregg, *et al* (1945) concerning congenital malformations in fetuses due to rubella infections of expectant mothers, studies with herpes simplex virus infections of the fetuses may be revealing.

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