Subacute myelo-optico-neuropathy (SMON) virus, a new human herpesvirus, has been isolated from human cases of SMON and found to be a variant of avian infectious laryngotracheitis virus, but considerably depending upon the individual litter of mice (2). The reason for different results in studies of SMON virus-infected C57BL/6 newborn mice has continuously been investigated. The purpose of the present communication is to report the fact that the onset of disease in SMON virus-infected mice is influenced by the kinds of feed given to the dams. SMON herpesvirus of the Watanabe strain, which had been propagated in human diploid cells, was used in this work. The virus was inoculated intracerebrally or intraperitoneally into C57BL/6 newborn mice at birth. Only one kind of pellet feed, "MF," as indicated in Table 1 summarizing the compositions of three kinds of pellet feed, was supplied to pregnant mice in a previous study (2). However, it was demonstrated in the present study that a specially prepared feed to affect reproduction of mice "CMF," given to the dams had an influence on the onset of disease; namely, the incidence of disease in SMON virus-infected newborn mice whose dams consistently received CMF before delivery was compared with that in SMON virus-infected newborn mice whose dams were fed "LF," instead of CMF, after delivery. The incidence of disease was also examined in SMON virus-infected newborn mice whose dams were given only LF throughout the pre- and postpartum periods.

The results (Table 2) revealed that the inci-
dence of disease in the CMF-CMF group was extremely low, and no mouse showed development of disease after intraperitoneal inoculation of the virus, whereas the incidence in the CMF-LF group was nearly the same as that in mice used for the previous study and disease developed in some mice after intraperitoneal inoculation. The LF-LF group was likely to be most susceptible to the virus. After 2 to 3 weeks or more of incubation, in the animals who showed the onset of disease, a loss of weight, piloerection, and characteristic paralysis of hind limbs were manifested, resulting in either death or recovery (Fig. 1). These symptoms were transient in some mice, so that mice should be observed every day, starting 10 days after inoculation of the virus, for a period of 2 months. The main pathological findings were symmetrical axonal degeneration and demyelination of the spinal cord, without inflammatory change (2).

It is reasonable to presume that feed can possibly influence the immune response of hosts, which may regulate the onset of disease, since it was shown previously that mice that showed no signs of infection had a lower virus titer in the brain and a higher antibody titer (2). This finding concerning diet is of practical significance because SMON virus infection in humans has recently been shown to exist also in countries other than Japan (unpublished data).

LITERATURE CITED