Population structure of some street rabies virus strains

Brief Report

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Summary. Street rabies virus strains can contain from one to three biological (clinical) variants as it has been estimated using random bred white mice and dogs.

Wild rabies virus strains are characterized by heterogeneity of biological properties, particularly by variation in the clinical forms of the disease. This peculiarity of rabies virus has no proper explanation in spite of the significant progress of virus studies. The present paper deals with the separation of a street rabies virus strain into up to three discrete biological variants.

The following street rabies virus strains were examined: Strain Yak, isolated from a boy who died of hydrophobia after being bitten by a fox in spite of complete and timely vaccinations [1], strain BE, isolated from a badger [2], and strains F-1 and F-2, isolated from the brains of rabid foxes were used. All street rabies virus strains and derived biological variants were identified by the neutralization test in mice [3] with commercial antirabic gammaglobulin and by detection of Babes-Negri bodies in brain sections stained according to Romanovsky and Giemsa. Virus log LD₃₀ titres/0.03 ml were calculated [6]. Average durations of incubation and clinical periods of the disease was estimated [5]. White random-bred mice of both sexes weighing 6–8 g and random-bred 6 months-old dogs were used.

Three different clinical forms of rabies were obtained after intracerebral inoculation at the first passage of street virus Yak strain in random bred white mice.

1. The acute paralytic form was characterized by a short incubation and clinical period. The disease started with a sluggish movement of the animals,
ruffled hair, rapid development of flaccid paralysis, prostration followed by death. Infectious virus was isolated from the brain of a mouse with the clinical form of acute paralytic rabies. This virus in the following 59 series of intracerebral passages caused only the acute paralytic form of rabies and was designated as the paralytic rabies variant (PRV). The incubation period for mice, inoculated with PRV diluted $10^{-1.0}$ was 2.5–3 days for passages 4–59 respectively. The illness lasted not more than 6 days, usually 2–4 days. The titres of virus isolated from the brain was estimated as $10^{7.0} - 10^{8.4} \text{LD}_{50}/0.03$ ml.

2. Mice infected with the Yak strain had also a second clinical form of illness—acute convulsive rabies. This form of the disease was characterized by bouts of tonic convulsions of limbs and body with elements of agitation. A bout of tonic convulsions was expressed by tension of all the muscles: fore and hind extremities were straightened, the body was erected, the hair stood on end, and the ears stuck out. Duration of convulsions and agitation was up to 5 seconds. Death was sudden, without paralysis and long-lasting agony.

Infectious virus was isolated from the brain of a mouse with the clinical form of convulsive rabies. In 20 series of intracerebral passages this virus caused only convulsive rabies and was designated as convulsive rabies variant (CRV). The incubation period for mice inoculated with CRV diluted $10^{-1.0}$ was 3.5–4.5 days. The illness lasted for 0.5–3 days for mice infected with the virus diluted $10^{-1.0} - 10^{-6.0}$. Virus titres were $10^{5.1} - 10^{6.6} \text{LD}_{50}/0.03$ ml.

3. In some mice infected with Yak strain a chronic form of rabies developed, characterized by a slow onset (7 days). Slight paresis of one of the hind limbs and ataxia was typical at that time. Then strong continuous clinical motions of the head—tremor and agitation appeared and lasted up to 15 days. At that period and especially at the decrease of clonical convulsions animals gradually develop paresis and Landry-type paralysis. The illness lasted 55, but more usually 15–30 days. Death was in 100% of the cases, as a rule after long agony. The infectious virus was isolated from the brain of a mouse typical chronic-type rabies. In the following 27 series of intracerebral passages this virus caused a chronic form of rabies in 10–43% of inoculated mice and was marked as chronic rabies variant (ChRV).

The incubation period and virus titres correlated with the period of illness for the mouse whose brain was taken for the inoculation. Thus, a 6–8 day incubation period and virus titres of $10^{4.2} - 10^{5.5} \text{LD}_{50}/0.03$ ml observed for mice inoculated with a $10^{-1}$ dilution of ChRV taken from the brain of mice with the period of illness 1–7 days. When the brain of a mouse in the 8–55 days of the illness was used for inoculation the incubation period varied from 7 to 143 days and virus titres were $10^{4.2} - 10^{9.8} \text{LD}_{50}/0.03$ ml. Long incubation period and low virus titres correlated with high titre of neutralizing antibody in the brains of mice with chronic rabies [1]. The ChRV variant population up to the 27th passage caused chronic rabies in 10–45% of the cases and acute paralytic rabies in 90–55% of the case. Acute convulsive rabies can be observed in some cases up to the 4th passage. It should be stressed that no chronic rabies were registered.