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# NONLETHAL INFECTION OF LABORATORY MICE INDUCED WITH "MOUSE" RABIES STRAINS

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*Summary.* — Nonlethal infection was induced in laboratory mice with sublethal doses of rabies strains isolated from small wild rodents. Three types of nonlethal infection were observed: persistent — with virus irregularly reisolable for at least 56 days after inoculation; latent — with negative virus reisolation but presence of infection demonstrable by transmission of virus neutralization activity to other animals; and abortive — with only a primary immune response, nontransmissible to other animals. A hyperreactive state called "early death" was observed in some mice. All surviving animals recovered without sequelae.

*Key words*: *rabies*; *nonlethal infection*; *mice*

## *Introduction*

Nonlethal infection with rabies virus has been described by numerous authors and different explanations for the phenomenon have been offered (see Discussion). A harmonization of the differing views is hampered by the diversity of the strains and animals employed.

For the present study, virus isolates from small wild rodents, displaying a tendency to induce nonlethal or chronic disease in their natural host animals (Sodja *et al.*, 1971), were selected. It is a continuation of a previous report concerned with the early events following infection of laboratory mice with sublethal doses of these strains (Sodja, 1979).

## *Materials and Methods*

*Virus.* CVS and mouse strains 297 BF, 703 B, 598 SG and 301 B were used. For their passage histories and origin see Sodja (1979). The strains were stored in portions at −70° C and the same batches were used throughout. The "mean time of death" of all the strains was 7 ± 1 days.

*White mice* (Velaz, Prague) weighing 9–10 g were used.

*Protection tests. Protection* (P): groups of 10 animals each were challenged intracerebrally with approximately 100 $LD_{50}$ of CVS. The number of survivors expressed the protection values. *Protection index* (PI) was assayed and calculated according to Habel (1973).

*Virus reisolation* was carried out by the mouse inoculation test (Koprowski, 1973). The specificity of death was checked by immunofluorescence and, in some cases, by the VN test. Groups of five mice were inoculated.

**Table 1. Protection of mice after i.c. inoculation of virus and reisolation attempts**

| Strain and dose of inoculum | Day after inoculation 1 | 2 | 3 | 4 | 5 | 6 | 7 | 10 | 14 | 21 | 28 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 297 BF, 1.7 $LD_{50}$ | | | | | | | | | | | |
| PI | −1.3 | 0,5 | 1.6 | −1.8 | 0.1 | −2.1 | −0.6 | 0.7 | 0.7 | ND | ND |
| P/300 | 0 | 3 | 3 | 0 | 0 | 0 | 0 | 0 | 3 | ND | ND |
| P/30 | 2 | 6 | 10 | 0 | 0 | 0 | 6 | 0 | 3 | ND | ND |
| RI-B | | | I+ | | | | | I+ | II+ | neg. | ND |
| RI-BF | | | II+ | | | | | I+ | II+ | neg. | ND |
| 297 BF, 0.3 $LD_{50}$ | | | | | | | | | | | |
| PI | −0.4 | 1.1 | −0.7 | −1.7 | 0.0 | −1.2 | −0.9 | 1.4 | 2.0 | 0.2 | 0.7 |
| P/300 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 5 | 2 | 0 |
| P/30 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 3 | 8 | 3 | 0 |
| RI-B | | | II+ | | | | | neg. | neg. | neg. | neg. |
| RI-BF | | | neg. | | | | | neg. | neg. | neg. | neg. |
| 703 B, 0.3 $LD_{50}$ | | | | | | | | | | | |
| PI | 0.3 | −0.7 | −1.5 | 1.9 | −1.2 | 0.6 | 1.6 | 2.0 | 2.2 | 2.7 | 1.0 |
| P/300 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 5 | 6 | 6 | 3 |
| P/30 | 0 | 3 | 0 | 6 | 0 | 0 | 6 | 10 | 10 | 10 | 6 |
| RI-B | | | neg. | | | | | neg. | neg. | neg. | neg. |
| RJ-BF | | | neg. | | | | | neg. | neg. | neg. | neg. |
| 598 SG, 0.4 $LD_{50}$ | | | | | | | | | | | |
| PI | −0.1 | 0.8 | 0.0 | −0.3 | −0.3 | 0.0 | −0.1 | 0.3 | 0.8 | 1.0 | 0.1 |
| P/300 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 3 | 0 |
| P/30 | 0 | 10 | 10 | 6 | 0 | 0 | 0 | 6 | 10 | 3 | 0 |
| RI-B | | | neg. | | | | | Ia | Ia | neg. | II+ |
| RI-BF | | | neg. | | | | | Ia | neg. | neg. | neg. |

PI = protection index ($\log_{10}$).
P/300, P/30 = No. of mice surviving the i.c. challenge dose indicated (out of 10 mice inoculated).
RI-B, RI-BF = reisolation of virus from the brain (brown fat) in the passage indicated (+) or positive immunofluorescence only (a).
ND = not done.

*Neutralization tests* (Atanasiu, 1973). In the virus-serum neutralization (VN) test, serial dilutions of virus and constant dilutions of a reference rabies-positive and a rabies-negative serum (1 : 10) were used. In the serum (brain suspension)-virus neutralization (SN) test, serial dilutions (1 : 10, 1 : 40, 1 : 160) of serum or 10% brain suspension were mixed with an equal volume (30−50 $LD_{50}$) of CVS strain. The mixtures were incubated at 4° C for 16 hr. The sera and brain suspensions were used inactivated at 56° C for 30 min.

*The direct immunofluorescence test* was performed by the method of Dean and Abelseth (1973). Commercial hamster antirabies virus conjugate (Bioveta Ivanovice, Czechoslovakia) was employed.

*Outline of experiments. Experiment 1.* Groups of mice were inoculated intracerebrally (i.c.) with sublethal doses of virus. At the intervals indicated in Table 1, P and PI were determined. Reisolation attempts from the brain and brown fat were carried out on days 3, 10, 14, 21 and 28 post infection (p.i.). From brains of mice surviving in the reisolation test, another blind passage was performed on the 10th day.

*Experiment 2.* Strain 297 BF in a dose of 0.1 or 0.01 $LD_{50}$ was inoculated i.c. or intramuscularly (i.m.) into mice. Protection against 100 $LD_{50}$ of CVS strain given i.c. and VN activity in sera and brain suspensions were tested at intervals indicated in Table 2.

*Experiment 3.* Strains 297 BF and 703 B in doses of 0.1, 0.01 or 0.001 $LD_{50}$ were inoculated i.c. into mice. Virus reisolation was attempted and serum and brain VN activities were deter-

**Table 2. Immune responses after inoculation of mice with strain 297 BF**

| Inoculation dose ($LD_{50}$) | Day p.i. | Route | | | | | |
|---|---|---|---|---|---|---|---|
| | | i.c. | | | i.m. | | |
| | | P | SVN | BVN | P | SVN | BVN |
| 0.1 | 2 | 3 | 2.0 | 1.0 | 6 | 2.0 | 1.5 |
| | 4 | 0 | 1.6 | 0.6 | 0 | 2.0 | 2.1 |
| | 6 | 0 | 1.5 | 1.0 | 0 | 2.0 | 0.6 |
| | 8 | 10 | 2.2 | 0 | 9 | 2.2 | 0.5 |
| | 14 | 7 | 1.3 | 1.4 | 3 | 1.1 | 1.4 |
| | 28 | 10 | 2.0 | 1.6 | 10 | 0.6 | 1.6 |
| | 56 | 7 | 1.6 | 1.6 | 10 | 2.0 | 1.0 |
| 0.01 | 2 | 6 | 1.1 | 1.3 | 8 | 2.0 | 0.9 |
| | 4 | 0 | 2.1 | 0.5 | 0 | 1.9 | 0.5 |
| | 6 | 0 | 2.2 | 1.3 | 0 | 1.6 | 0.6 |
| | 8 | 10 | 2.0 | 0 | 10 | 2.1 | 2.1 |

P: protection; No. of mice surviving i.c. challenge with 100 $LD_{50}$, out of 10 inoculated.
SVN, BVN: VN titre of sera and brain suspensions ($log_{10}$).

mined in three blind passages performed at 10-day intervals (if virus was isolated, subsequent blind passages were not performed). The reisolation attempt intervals are indicated in Fig. 1.

*Experiment 4.* Mice were inoculated i.m. or intranasally (i.n.) with 0.1 $LD_{50}$ of strain 297 BF or 703 B. After 14, 28 and 56 days groups of five mice each were killed and reisolation attempts were performed from their brains, brown fat, salivary glands, lungs and kidneys. Blind passages were not performed. The organ suspensions collected 14 days p.i. were heated at 56° C for 30 min and used for intraperitoneal (i.p.) immunization of 10 mice (see Fig. 2). Immunization scheme: 6 i.p. doses of 0.5 ml each during 14 days. On day 21 the animals were exsanguinated. The sera and brain suspensions from mice used for virus reisolation were furthermore assayed for VN activity.

*Experiment 5.* Groups of mice were given sublethal i.m. or i.n. doses of strain 297 BF or 703 B (see Table 3). The PI was calculated and reisolation attempts (three blind passages) were performed after 14, 28 and 56 days. VN activity of sera and brain suspensions was assayed in the 1st and 2nd blind-passage animals 10 days after inoculation.

## *Results*

Evidence of nonlethal infection was based on virus reisolation or development of specific immune reactions in experimental animals. In exp. 1 (Table 1), in which strains isolated from different organs of wild mice were used, a negative PI was frequently obtained in the first week p.i. This phenomenon of "immune" mice dying 1—2 days earlier than "control" animals and after a lower virus dose is called "early death" (Sikes *et al.*, 1971). Virus was irregularly reisolated throughout the period of observation of 28 days. Attempts to reisolate strain 598 SG on days 10 and 14 p.i. failed in spite of evident specific immunofluorescence in the brains.

In exp. 2 (Table 2), only strain 297 BF was inoculated i.c. and i.m. Protection was evident on day 2 (and 3), disappeared on days 4 to 6, and was pronounced thereafter. The route of inoculation or the dose of virus did not essentially affect the value of P and VN activity, though the VN titres rose more slowly after the lower virus dose, especially if administered i.c. During

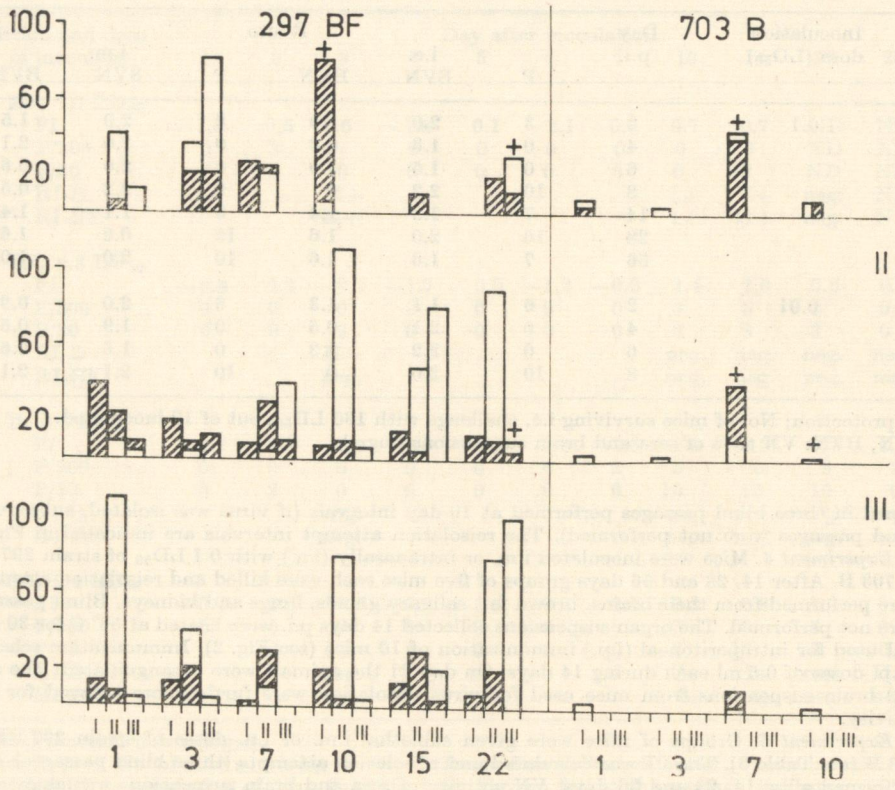


**Fig. 1.**

Results of i.c. inoculation of mice with 0.1 (I), 0.01 (II) and 0.01 (III) $LD_{50}$ of 297 BF or 703 B virus

Abscissa: passage number (I—III) and day p.i. (1—22)

Ordinate: titre of VN activity in sera (empty columns) and brain suspensions (shaded columns)

\+ Reisolation positive

the early phase p.i., brain VN activity was lower than serum VN activity, but both more or less evened up by day 14. The P value was in no pronounced correlation with VN activity.

Fig. 1 illustrates the results of exp. 3, in which strain 297 BF was reisolated in the first blind passage on day 10 after an inoculum of 0.1 $LD_{50}$ and in the third blind passage on day 22 after inocula of 0.1 and 0.01 $LD_{50}$. Strain 703 B was recovered in the first passage on day 7 after both inocula (0.1 and 0.01 $LD_{50}$) and on day 10 p.i. in the 3rd passage, but only after a dose of 0.01 $LD_{50}$. Evidence of the presence of strain 287 BF in terms of VN activity was obtained in animal sera and brain suspensions in individual blind passages, but again the serum and brain VN activity levels

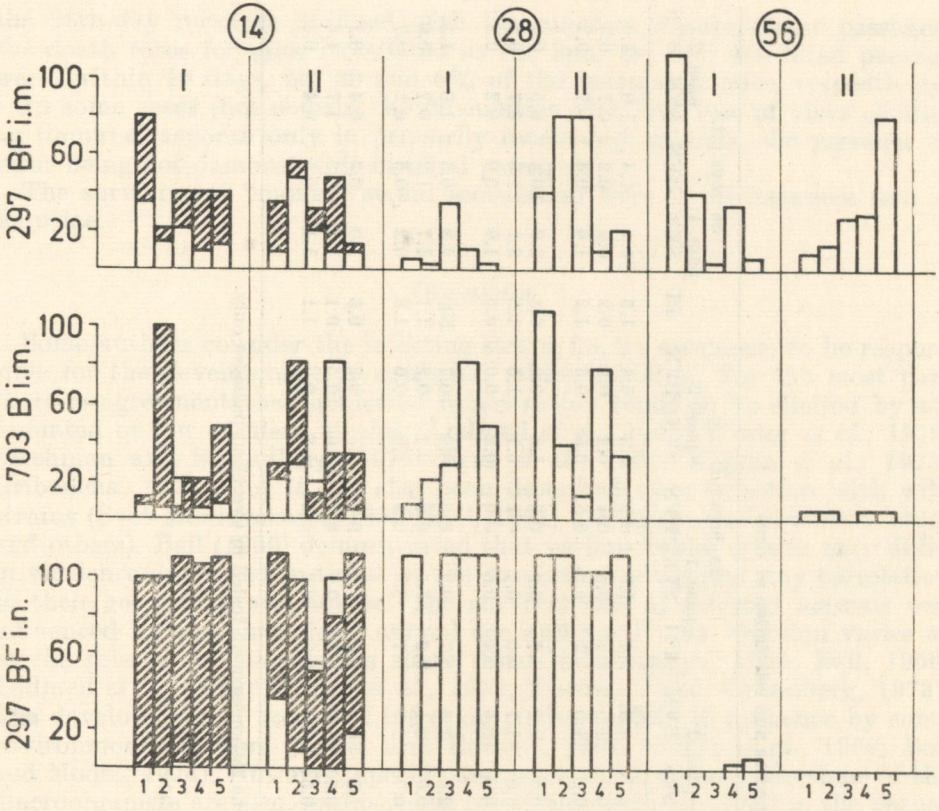


**Fig. 2.**

VN activities in sera (I) and brain suspensions (II) from mice immunized with organ suspensions (shaded columns) and from mice used for reisolation of virus (empty columns)
Figures in circles: days p.i.
Abscissa: 1 — brain; 2 — brown fat; 3 — salivary glands; 4 — lungs; 5 — kidneys
Ordinate: VN activity titre

were not in mutual correlation. With strain 703 B, both activities were negative as from day 10.

The wild rodent strains had been isolated from different organs. It thus could not be precluded that the virus tended to persist in some other organ than the brain. Reisolation attempts in exp. 4 (Fig. 2) failed, but specific VN activity was demonstrated both in animals employed for the reisolation and in animals immunized with suspensions of the individual tissues. As a rule, the values of VN activity were higher in immunized mice than in animals used for reisolation of virus.

Positive virus reisolation at a protracted interval p.i. is documented in Table 3. After i.n. inoculation of 0.02 $LD_{50}$ of strain 703 B, the virulence of

**Table 3. Immune responses and reisolation of virus**

| Strain | Route | Reisolation* | Inoculum ($LD_{50}$) | 14 days p.i. | | | | | 28 days p.i. | | | | | 56 days p.i. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | pass. 1 | | pass. 2 | | | pass. 1 | | pass. 2 | | | pass. 1 | | pass. 2 | |
| | | | | PI | SVN | BVN | SVN | BVN | PI | SVN | BVN | SVN | BVN | PI | SVN | BVN | SVN | BVN |
| 297 BF | i.m. | 3/56 | 0.27 | 1.2 | 2.0 | 0.0 | 0.7 | 0.0 | 3.4 | 1.0 | 0.0 | 0.3 | 0.4 | 1.5 | 1.1 | 1.4 | 1.0 | 1.0 |
| | | | 0.02 | 1.2 | 1.2 | 0.5 | 0.5 | 1.0 | 4.2 | 0.5 | 0.1 | 0.2 | 0.5 | 0.2 | 0.9 | 0.2 | 0.7 | 0.2 |
| | | | 0.002 | 2.5 | 1.7 | 0.5 | 0.2 | 1.0 | 3.3 | 0.2 | 0.5 | 0.5 | 0.5 | 1.2 | 1.6 | 0.4 | 0.7 | 0.2 |
| | i.n. | | 0.42 | 2.2 | 1.0 | 2.0 | 0.7 | 0.2 | 2.2 | 0.0 | 0.5 | 1.2 | 0.0 | 0.8 | 0.9 | 0.4 | 0.5 | 0.2 |
| | | | 0.04 | 2.0 | 0.5 | 2.0 | 0.0 | 1.7 | 0.6 | 0.1 | 0.1 | 0.1 | 1.5 | 1.5 | 2.2 | 1.6 | 0.7 | 0.5 |
| | | | 0.004 | 2.2 | 0.5 | 2.0 | 0.0 | 0.5 | 0.4 | 0.5 | 0.0 | 0.2 | 0.0 | 0.6 | 1.9 | 0.9 | 0.2 | 0.2 |
| 703 B | i.m. | | 1.22 | 0.3 | 0.6 | 0.7 | 2.0 | 1.5 | 0.7 | 0.2 | 0.5 | 0.2 | 0.7 | ND | 2.3 | 0.5 | 1.0 | 1.3 |
| | | 3/14 | 0.22 | 2.6 | 0.0 | 0.0 | 0.0 | 2.1 | 0.2 | 3.5 | 0.2 | 0.2 | 0.2 | 1.1 | 1.7 | 1.2 | 0.7 | 0.0 |
| | | 3/14 | 0.02 | 2.6 | 0.0 | 0.5 | 0.7 | 2.7 | 0.7 | 2.5 | 0.5 | 1.0 | 0.0 | 1.8 | 0.2 | 0.5 | 0.2 | 0.7 |
| | i.n. | 3/56 | 0.25 | 0.5 | 1.1 | 0.9 | 1.2 | 0.7 | 0.0 | 1.2 | 0.2 | 0.5 | 0.2 | 0.5 | 1.0 | 0.7 | 0.4 | 0.1 |
| | | 3/56 | 0.02 | 0.2 | 1.2 | 0.2 | 0.7 | 1.0 | 0.7 | 0.5 | 0.2 | 1.0 | 0.5 | 0.1 | 1.0 | 0.2 | 1.7 | 1.6 |
| | | 2/14 3/56 | 0.002 | 0.2 | 1.0 | 0.0 | 0.5 | 0.7 | 0.9 | 0.2 | 0.2 | 1.2 | 0.9 | 1.1 | 1.1 | 0.5 | 0.4 | 0.4 |

Three blind passages were used for reisolation. Immune responses were tested in two blind passages only.
* No./day of positive blind passage.
PI: protection index ($log_{10}$).
SVN, BVN: serum and brain suspension VN titre ($log_{10}$).

the 28th-day reisolate declined with the number of subsequent passages: the death rates for mice inoculated as the 2nd, 3rd and 4th blind passage were, within 10 days, 60, 30 and 0% of the inoculated mice, respectively.

In some cases (not shown) the inoculation of a low dose of virus elicited an immune response only in primarily inoculated animals, the presence of virus being not demonstrable in blind passages.

The survivors of "mouse" strain inoculation were in all instances free of sequelae.

## *Discussion*

Some authors consider the infecting strain, i.e. its virulence, to be responsible for the development of nonlethal rabies infection. For the most part there is agreement that nonlethal rabies rather tends to be elicited by attenuated or low virulent strains (Lodmell *et al.*, 1969; Wiktor *et al.*, 1972; Fischman and Strandberg, 1973; Bear *et al.*, 1975; Kaplan *et al.*, 1975; Gribencha, 1976), but it has also been described after infection with wild strains (Svet-Moldavskaya, 1958; Bell, 1964; Gribencha and Selimov, 1974; and others). Bell (1966) demonstrated that various rabies strains may differ in virulence expressed in terms of the survival rate without any correlation to their geography and source. The survival rate of infected animals was influenced by the virus dose, animal age and partly sex. Opinion varies as to the role of the inoculation route (Svet-Moldavskaya, 1958; Bell, 1966; Lodmell *et al.*, 1969; Wiktor *et al.*, 1972; Fischman and Strandberg, 1973). The development of nonlethal infection is also subject to influence by some environmental factors (Sadler and Enright, 1959; Sulkin *et al.*, 1960; Bell and Moore, 1974). However, specific and nonspecific defense reactions of the macroorganism are also, and perhaps preponderantly, involved in the pathogenesis of rabies (Campbell *et al.*, 1968; Lodmell *et al.*, 1969; Wiktor *et al.*, 1972).

For the present studies virus isolates from small wild rodents were selected in view of some of their biological properties (e.g. high neurotropism, viscerotropism, fixed incubation period, ability to induce chronic disease in the natural host — Sodja and Matouch, 1972, 1973). Except where indicated otherwise, the experiments were performed with identical batches of viruses throughout. No relationship between the source of the strain and its reisolability or the development of a particular form of nonlethal infection was found. After inoculation of sublethal doses (at least 0.01 $LD_{50}$), sometimes a transitory replication of virus was found. Gribencha and Ovsyannikova (1976) demonstrated that the intensity of virus replication in "abortive" infection is slower than in lethal infection. I observed (unpublished results) relatively low virus titres in the first days after inoculation of low lethal doses, but at the time of death they were practically equal to those after higher infective doses.

In the present experiments, sublethal doses of "mouse" strains induced the following types of nonlethal infection: (*a*) persistent — with reisolable

virus; (*b*) latent — with negative reisolation but positive immune response in blind animal passages; (*c*) abortive — with negative reisolation and negative immune reactions in blind passage animals, but with positive VN activity in primarily inoculated animals; and (*d*) "early death" with the inoculated (immunized) animals dying earlier and after a lower dose than controls.

The virus reisolation rate was in no correlation with the dose of inoculum. Negative reisolation can, at least in some instances, be explained by prolongation of the incubation period, lower virulence of the persisting virus, or its binding to antibody. A higher virus affinity for brown fat or other organs than for the central nervous system (CNS) was not observed.

The experiments did not elucidate the role of antibodies in the pathogenesis of nonlethal rabies. Relatively high and comparable titres of VN activity demonstrated in cases both with and without reisolable virus. There was no correlation between VN activity titres in sera and brain suspensions, nor between these values and protection. Similar findings were reported by Wiktor *et al.*,(1976).

Gribencha (1976) and Coe and Bell (1977) suggested the formation of specific antibodies in the CNS.

The manner of virus inoculation, taking rabies pathogenesis into consideration (Miller and Nathanson, 1977), has some influence on animal survival after infection with strains of relatively low invasiveness (Baer *et al.*, 1977) rather than on the development of one or an other form of nonlethal infection. The "early death" phenomenon is usually attributed to autoimmune reaction (Sikes *et al.*, 1971).

One may also suspect, especially at early stages after infection, the assembly or activation of "incomplete" virus in the broad sense of the word. Assay for interferon was, in the present study, performed only in some instances but the results were invariably negative, like in previous experiments (Sodja, 1975). However, the presence of interferon cannot be excluded by the evidence obtained and it possibly might be detectable by more sensitive methods.

The present results were obtained in several groups of experiments. They imply that none of the suggested factors probably plays a crucial role in the development of the individual forms of nonlethal infection by itself, but that this is a result of interaction between the micro- and macroorganism at different stages of virus development. Experiments on reactivation of persisting virus might help to clarify some of these problems.

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