principal intermediate hosts. Other snails and slugs mentioned above seem to be of minor importance. The development of the third stage larvae is described and subsequent infections of cats and kittens are recorded. Mice fed with first stage larvae of the lungworm escaped infections. The intermediate hosts of the lungworm *Aelurostrongylus abstrusus* are not, as Cameron states, mice, but certain mollusks.

8211 P

Route of Transmission of St. Louis Encephalitis Virus in Mice.*

MAURICE BRODIE.

From the Department of Bacteriology, New York University Medical School and the Bureau of Laboratories, Department of Health, New York City.

Histopathological studies by Webster and Fite[1] suggest that the virus isolated from the St. Louis outbreak of encephalitis, travels by way of the central nervous system in mice. The present report confirms and extends these findings.

In order to determine the portal by which mice could be infected, they were given virus by the nasal and gastro-intestinal routes. Three series of mice each consisting of 4 to 6 animals, were given a concentrated suspension of the virus in their food over 2 to 3 days. Not one of these mice became infected. Three animals, upon whom a laparotomy was performed, were injected intrastomachally with 0.2 cc. of a thick virus suspension. None of these showed evidence of the disease.

Mice could be infected regularly with the intracerebral virus, within an incubation period of 6-8 days, by placing 0.03 cc. of a 1% suspension into one nostril, while 0.1% failed to give regular infection. The virus was transmitted serially by the intranasal route for 28 passages, using a 10% suspension, but its infectivity, by this route, did not increase.

After 28 passages the virus was infective by the intranasal route, at approximately a 1 :250 dilution.

In order to trace the route of infection from the nasopharynx,

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[1]Webster, L. T., and Fite, S. L., *J. Immun.*, 1934, **26**, 344; *Am. J. Path.*, 1934, **10**, 666.

the blood, parenchymatous tissues and various areas of the cerebrospinal axis were tested for the presence of virus after intranasal instillation of virus at various stages of the incubation period and of the disease. Two experiments were carried on simultaneously; one in which various organs and areas of the cerebrospinal axis were suspended in distilled water and one in which 10% normal monkey serum, which enhances the infectivity of the virus, was employed as diluent. In testing any area, a suspension made of a pooling of 2 to 3 organs in that area was injected intracerebrally and intraperitoneally Control animals receiving intranasal inoculations of the same virus were included in east test and always showed symptoms.

On only 2 occasions at 3 hours and 48 hours was virus demonstrated in the blood. It was demonstrable in the parenchymatous organs only occasionally and then usually early or late in the disease. On the other hand, it was present in the olfactory bulbs as early as 2 hours after inoculation. The next location of the virus was the lobus pyriformis, while late in the incubation period or early in the disease, it had spread throughout the whole central nervous system.

The findings indicate that the virus enters by the olfactory rather than the gastrointestinal tract. Moreover, inasmuch as the virus is rarely found in the blood stream, as indicated by these experiments and those of Webster and Fite,[1] and since we were unable to infect regularly by the skin route with less than 0.5 cc. of a 10% suspension, insect transmission is unlikely. The virus then appears to travel along the nerve fibers of the olfactory nerve to the bulb and thence to the lobus pyriformis, after which it spreads to all parts of the central nervous system. These experiments confirm the histopathological findings of Webster and Fite, who found that the lesions appeared in the above mentioned sequence after intranasal instillation of virus. It is interesting to note that the virus appears earlier than do the lesions, for these authors did not find lesions in the olfactory bulbs until the third day after intranasal inoculation, whereas they found virus present 2 days after nasal instillation and in the present work it has been reported as early as 2 hours after the introduction of the virus. Likewise, the virus is present in the lobus pyriformis prior to the presence of cellular infiltration, indicating that the lesions are the result of damage by the virus.

The early, occasional demonstrable virus in the parenchymatous organs or blood stream may be due to absorption by the nasal mucosa. Its more frequent later appearance may be due to an over-

flow of the virus from the perivascular spaces of the central nervous system, into the blood and thence to the organs.

8212 P

"Mottling of Enamel" Effected by a Single Fluorine Dose.

S. LOEWE AND H. SALFELD. (Introduced by H. Sobotka.)
From the Laboratories of the Mount Sinai Hospital, New York.

In a study of the minimal effective dosage of fluorine, as determined by various biological criteria, one of the authors[1] observed "fluorine-rickets" as a significant response to very small amounts of fluorine. The biological effectiveness of even smaller amounts of this element has been established by the discovery of the "mottling of enamel."[2] Until now, mottling of enamel has been studied only on frequently repeated fluorine administration. The question arose whether it can also be produced by a short "fluorine shock."

The following is a report on a preliminary series of experiments on 20 young rats. In the first experiment (Table I) doses from 9 to

TABLE I.
Effect of Fluorine Administration on Incisors of Recently Weaned Rats.

No.	Wt at start gm.	Wt on 11th day gm.	Administered mg. F per kg. b. w.	Enamel Spot Day of Appearance	Disappearance
1	31	25	CaF_2 37	—	—
2	29	30	NaF 30	—	—
3	23	32	CaF_2 19	—	—
4	23	34	CaF_2 19	—	—
5	35	38	NaF 17	12	24
6	30	38	NaF 15	—	—
7	36	39	CaF_2 10	—	—
8	28	29	CaF_2 9	9	21
9	35	36	CaF_2 9	9	24
10	31	37	NaF 7	—	—

37 mg. fluorine per kilo of b.w. were administered through a stomach tube to just weaned albino rats weighing from 21 to 35 gm. (solutions of 0.625% or less NaF and a finely dispersed sol of 0.375% or less CaF_2[3]). Such a single dose was able to produce a definite alteration on the enamel. In 3 out of 10 animals, a sharply

[1] Loewe, S., *Schweizer med. Ws.*, 1934, **64**, 1177.

[2] Smith, M. C., and Leverton, R. M., *Ind. Eng. Chem.*, 1934, **26**, 792; De Eds, F., *Medicine*, 1933, **12**, 1.

[3] Bachmann, W., D. R. P., 485,052.