

The inoculums used in test and control tubes were the same in any given set of experiments. Population counts and pH measurements were determined simultaneously. Routine bacteriologic cultures were made from all tubes and no data are included from contaminated specimens.

Under the conditions of these experiments glucose, and its polymers, maltose, soluble starch, glycogen, and dextrin alone were utilized to any considerable extent. Fructose and galactose are in a borderline group as evidenced by a slight stimulation of growth but no appreciable pH shift.

These results differ somewhat from those recorded by Cailleau<sup>6</sup> for *T. foetus* and *T. columbae* and offer one more argument against the identity of *T. vaginalis* and the lower animal trichomonads.

It is interesting clinically that growth of the strain of *T. vaginalis* tested is not stimulated by lactose. This is also true of the common vaginal fungi ("monilia"). Hesseltine<sup>7</sup> contends that lactose should be used in the therapy of vaginitis to prevent the growth stimulation of the fungi which may occur if dextrose is employed. The data here recorded indicate that a similar situation prevails among the trichomonads, although it is obvious that other strains must be studied before such conclusions are warranted.

## 13077

### Susceptibility of Syrian Hamster (*Cricetus auratus*) to Viruses of St. Louis and Japanese B Encephalitis.

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Because of the lack of other susceptible animal hosts, experimental studies on the virus of St. Louis encephalitis have been confined entirely to the use of the mouse and the monkey. Similarly, although the host-range for the Japanese B encephalitis virus appears to be larger,<sup>1</sup> only the mouse and the monkey have proved suitable for laboratory purposes. In view of the desirability, therefore, of having available a fairly large, inexpensive, and readily procurable

<sup>6</sup> Cailleau, R., *Ann. Inst. Pasteur*, 1937, **59**, 137, 293.

<sup>7</sup> Adair, F. L., and Hesseltine, H. C., *Am. J. Obst. and Gynec.*, 1936, **32**, 1.

<sup>1</sup> Kasahara, S., *et al.*, *Kitasato Arch. Exp. Med.*, 1936, **13**, 248.

animal suitable for the study of these viruses, several animal species were examined for their susceptibility to infection. The present report deals with the susceptibility of the Syrian (golden) hamster, *Cricetus auratus*.

*Material.* The Broun\* and Hubbard strains of the St. Louis virus and the Nakayama strain of the Japanese virus were employed. Mouse-passage virus was used to initiate the hamster-passages; virus-infected mouse brains were ground with 10% normal horse-serum broth to make a 10% suspension. After centrifugation at 500 rpm for 2 to 5 minutes, each supernate was injected intracerebrally in 0.1 cc amounts into groups of 2 hamsters. Hamster-brains removed for passage were similarly prepared. All brains were cultured immediately after removal, and only bacteriologically sterile specimens were used.

*St. Louis Encephalitis Virus.* The Broun strain had been passaged serially through 7, and the Hubbard strain through 9 hamsters. With each strain one hamster-brain from the 5th passage was titrated in mice by the cerebral route. The Broun strain was found to be infectious through a dilution of  $10^{-6}$ , the Hubbard strain through a dilution of  $10^{-7}$  (Table I). A portion of each hamster-brain suspension from this passage was also injected into mice to obtain virus for neutralization-tests. The mice were killed when in convulsions; the brains of 4 were pooled, ground, and made into a 20% suspension in 20% horse-serum broth. The supernate obtained after light centrifugation was used to make serial tenfold dilutions in 10% horse-serum broth. To 0.2 cc of undiluted serum was added 0.2 cc of virus-dilution. The mixtures were incubated for 4 hours at  $37^{\circ}\text{C}$  and each was inoculated in 0.03 cc amounts into groups of 4 Swiss mice. Both strains were neutralized specifically by rabbit antisera to St. Louis virus but not by superimmune lymphocytic-choriomeningitis guinea pig serum.<sup>†</sup> Sera from normal rabbits and guinea pigs failed to neutralize the virus.

The incubationary period with both strains was 3 to 4 days. Then the fur became ruffled, the animal was apathetic, and the eyes were closed. Salivation was common. The back might become arched, but in the few instances in which this was observed the animal became cachectic and died without showing further symptoms. A fine or a gross body-tremor was frequently discernible

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\* This strain was obtained through the courtesy of Dr. L. T. Webster of the Rockefeller Institute.

† The lymphocytic-choriomeningitis antiserum was kindly supplied by Dr. J. E. Smadel of the Hospital of the Rockefeller Institute.

TABLE I.  
Titration in Mice of Virus-Content of 5th-passage Hamster-brain.

Hamster	Virus and strain	Dilution of hamster-brain				
		10 <sup>-4</sup>	10 <sup>-5</sup>	10 <sup>-6</sup>	10 <sup>-7</sup>	10 <sup>-8</sup>
H447	St. Louis, Broun	4/4*	4/4	3/4	0/4	0/4
H460	'' '' Hubbard	4/4	4/4	4/4	2/4	1/4
H463	Japanese B, Nakayama	4/4	4/4	4/4	4/4	0/4

\*The numerator represents the number of mice that died; the denominator, the number of mice inoculated.

and appeared with an ataxia of variable degree. This was followed by paralyzes of the limbs, usually the forelegs and spastic in type; flaccidity might occur but was associated chiefly with paralysis of the hind legs. Death supervened within 1 to 2 days after onset of symptoms, although the entire course of the disease might be telescoped into a period of several hours.

The susceptibility of the hamster to the St. Louis virus approximated that of the mouse. Serial tenfold dilutions in 10% horse-serum broth were made from a 10% suspension of mouse-brains infected with the Hubbard strain and inoculated intracerebrally into hamsters (0.1 cc) and mice (0.03 cc). As shown in Table II, the virus was lethal for mice in a dilution of 10<sup>-7</sup> and for hamsters in a dilution of 10<sup>-6</sup>. Furthermore, both species appeared to react in a similar way to intranasal, intraabdominal, or subcutaneous inoculation of large amounts of virus. Thus, of 3 hamsters inoculated intranasally with 0.5 cc of 10% mouse-virus suspension, all died of encephalitis on the 6th day. In another group of 3 animals inoculated intraabdominally with 0.5 cc of a 1% viral suspension, 2 died of encephalitis and one survived without exhibiting any evidence of infection. None of 3 hamsters inoculated subcutaneously with 0.25 cc of 1% virus showed any signs of infection.

*Japanese B Encephalitis Virus.* The Nakayama strain was passed serially, brain to brain, through 7 hamsters and its propa-

TABLE II.  
Comparison of Infectivity of St. Louis and Japanese B Encephalitic Viruses for Mice and Hamsters.

Virus and strain	Animal inoculated	Dilution of mouse-passage virus				
		10 <sup>-4</sup>	10 <sup>-5</sup>	10 <sup>-6</sup>	10 <sup>-7</sup>	10 <sup>-8</sup>
St. Louis, Hubbard	Mouse	4/4	4/4	4/4	4/4	0/2*
	Hamster	2/2	2/2	2/2	0/2	
Japanese B, Nakayama	Mouse	4/4	4/4	4/4	3/4	2/4
	Hamster	2/2	2/2	2/2	2/2	

\*Two mice died of nonspecific causes within 3 days after inoculation.

gation in this environment is demonstrated by comparing Tables I and II, which show the infectivity for mice of mouse-brain virus (31st mouse-passage) and hamster-brain virus (5th hamster-passage), respectively. Proof was adduced from neutralization-tests that the infectious agent being passed was the Japanese virus and not another latent virus in the host. The agent was neutralized specifically by rabbit antisera to Japanese virus. Normal rabbit or guinea pig sera as well as the sera of guinea pigs immunized against lymphocytic choriomeningitis failed to inactivate the virus.

The incubationary period for the Nakayama strain after cerebral inoculation was 5 to 7 days, somewhat longer than for the strains of St. Louis virus, and the animals exhibited the same clinical picture described above for the St. Louis virus. Like the St. Louis virus, the Japanese virus, by the cerebral route, also possessed about the same degree of infectiousness for the hamster as for the mouse (Table II). The hamster also was susceptible to infection by the nasal route; failure of infection after intraabdominal or subcutaneous inoculation (Table III), however, was unexpected. While no emphasis is placed on the findings of a single experiment employing only a few animals, it should be pointed out that the latter results are at variance with what occurs when mice are inoculated by the intraabdominal or subcutaneous routes with the Japanese virus.

*Conclusions.* The Syrian hamster (*Cricetus auratus*) is highly susceptible to cerebral infection with the viruses of St. Louis and of Japanese B encephalitis and should prove valuable in certain types of investigation where the use of mice or monkeys is not feasible. Both viruses easily propagate in the brain of this host and attain a titer comparable to that achieved in mouse-brain.

TABLE III.  
Infectivity for the Hamster of St. Louis and Japanese B Viruses Administered by Various Routes.

Virus and strain	Inoculum		Encephalitis
	Amount	Route	
St. Louis, Hubbard*	cc		
	.5	10-1 intranasal	3/3†
	.5	10-2 inträabdominal	2/3
	.25	10-2 subcutaneous	0/3
Japanese, Nakayama‡	.5	10-2 intranasal	2/3
	.5	10-2 inträabdominal	0/3
	.25	10-2 subcutaneous	0/3

\*Lethal dilution for mice = 10-7.

†Numerator denotes number of animals succumbing with encephalitis, denominator the number of animals inoculated.

‡Lethal dilution for mice = 10-8.