the brain increases to about 2 μmole/g. The animals do not awaken for at least 5 hours. There is no significant change in the content of γ-hydroxybutyric acid in any tissue except brain. In the brain, the free acid concentration increases at 2 hours to 0.3 μmole/g; at 4 hours it is 0.37 μmole, about equal to the lactone which remains. In the lactone-treated animals the sleeping interval is parallel to the concentration of the lactone rather than anion in the brain, for in the 1st hour after lactone administration there is no significant rise in anion, yet the animals are asleep. On this basis it appears that the sleep-inducing properties of γ-hydroxybutyrate are related to its conversion to the lactone form in the brain. If sleep were related to the amount of γ-hydroxybutyrate in the brain, the animals given γ-hydroxybutyrate should have been asleep for more than two hours since this was the time of the highest measured values for free acid. The data, after administration either of lactone or free acid, are consistent in indicating the lactone form as the active intermediate.

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Cerebellar Ataxia in Hamsters Inoculated with Rat Virus

Abstract. *Chronic ataxia associated with hypoplasia of the cerebellum was induced by intracerebral inoculation of newborn hamsters with various strains of rat virus. The condition usually became recognizable within 3 weeks of inoculation and remained static as the animals matured. Preliminary studies suggest that hypoplasia is induced by the rat virus acting on the outer germinal layer of the cerebellum at a critical time in its ontogenic development.*

Rat virus has produced a wide spectrum of disease states in the Syrian hamster. Rat 12 strain (*1*), adapted by previous animal passages, has produced an acute, fulminant, rapidly fatal disease (*2*) in neonates, a mongoloid dwarfism (*3*) associated with periodontal disease (*4*) in older sucklings, and infections of the uterus, placenta, and fetus (*5*) in pregnant hamsters. In none of these earlier studies were infections of the central nervous system apparent.

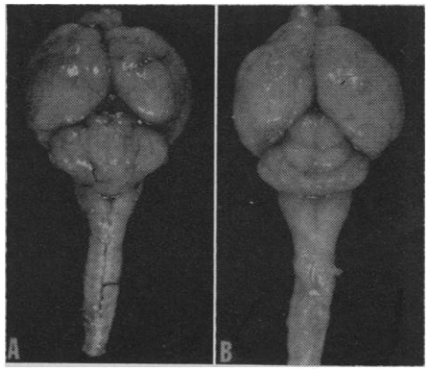

Fig. 1. The cerebellum of a normal (*A*) and an ataxic (*B*) hamster.

In more recent experiments with freshly isolated strains of rat virus inoculated intracerebrally in newborn hamsters, cerebellar ataxia has been regularly encountered. Ataxia was first observed in studies with two new strains, 171 and 312, isolated from two different lots of the Moloney leukemia virus (*6*).

Initially, neither of these strains appeared to be pathogenic for hamsters, since they produced no overt disease during the suckling period after combined intraperitoneal and intracerebral inoculation of newborn animals. It was then decided to keep the animals under observation for prolonged periods to determine whether neoplasms would develop. While no tumors appeared during periods up to 8 months, ataxia developed within 4 to 5 weeks of inoculation, and the animals remained smaller than littermates given control intracerebral injections of tissue culture fluid at birth. The ataxia was characterized by an unsteadiness of gait, quick oscillatory movements of the body and, particularly, by instability of balance. The affected animals frequently and spontaneously fell over on their backs and righted themselves with variable difficulty as they progressed around their cages. This ataxic state generally affected all inoculated littermates to the same degree, and persisted without significant change in severity as the animal matured.

The anatomic basis for the ataxia was a selective action of the rat virus upon the outer germinal layer of the cerebellum. Pathologic studies (*7*) demonstrated a lysis of these cells late in the 1st week after inoculation and, subsequently, failure of the mature cerebellar granular layer to develop. Consequently, the cerebellum presented an overall picture of severe hypoplasia, in which the neonatal appearance and relationships were retained. Characteristically, the cerebellum of the ataxic hamster appeared as a small arciform structure, above which the corpora quadrigemina were completely uncovered (Fig. 1).

During five continuous passages in rat embryo tissue culture, the virulence of the 171 and 312 strains of rat virus remained stable and continued to produce ataxia upon intracerebral inoculation. The age of the hamsters at the time of injection was a major determinant of the ability of the virus to induce the cerebellar lesion. When the inoculation was made within 24 hours of birth, virtually all animals of a litter developed ataxia. When the virus was administered on the 2nd day after birth or later, no ataxia or other manifestations of disease appeared during the observation periods which extended over 2 months. Both the 171 and 312 strains were similar to the original rat virus strain (*1*) in being resistant to exposure to 20-percent ether overnight and to heating at 60°C for 30 minutes (*6*).

After three passages in rat embryo tissue culture, and three continuous passages in hamsters (*2*), the 171 and 312

Table 1. Results of neutralization tests in which the capacities of three different strains of rat virus (RV) to induce cerebellar ataxia were tested against serum from an RV-immune hamster, after intracerebral inoculation of newborn hamsters.

Hamster serum HI titers*	RV-strain inoculated		
	171	312	13
Normal (HI = 0)	4/4†	3/3	4/4
RV-immune (HI = 1:320)	0/3	0/5	0/4

* Inhibition of hemagglutination. † Number developing ataxia/number inoculated.

strains showed a striking increase in virulence, and produced a variety of disease states depending upon the method in which they were administered. Cell suspensions from pools of infected liver and kidneys, passed by intraperitoneal inoculation, produced in neonates an acute, fulminant illness (*2*) characterized by a hemorrhagic enteritis and severe wasting, terminating fatally within 7 to 10 days. When infected brain tissue was administered to neonates by intracerebral injection, a severe form of ataxia appeared within 12 days. These animals were stunted, emaciated, exhibited teeth deformities, and died within 3 to 4 weeks, probably from systemic spread of rat virus. With the enhancement of virulence, it became almost impossible to induce a chronic ataxia because of the failure of the affected animals to survive.

Subsequent studies have demonstrated the production of cerebellar ataxia by five strains of rat virus of diverse origins. These include (i) rat 12 and rat 13 strains isolated from Fisher rats bearing spontaneous hepatic sarcomas (*1*), (ii) the 171 and 312 strains isolated, respectively, from Osborne-Mendel and Sprague-Dawley rats infected with the Moloney leukemia virus (*6*), and (iii) the L.S. strain from Wistar rats bearing a transplantable chloroleukemia (*8*).

Evidence for the serologic relationship of the various strains of rat virus to the original strain (rat 12), has been shown (*6, 8*). The neutralization tests performed on suckling hamsters by intracerebral inoculation show (Table 1) that rat-virus-immune serum, from an inoculated hamster, neutralized the capacity of three different strains of rat virus to induce ataxia (*2*).

Failure in the past to recognize cerebellar ataxia as a manifestation of experimental rat virus infection can be accounted for by the fact that the restricted host-parasite relationship necessary for the production of this disease state had not previously been met. For ataxia to develop, the following are prerequisite: (i) the use of virus freshly isolated or maintained by passage in tissue culture rather than virus with virulence enhanced by previous animal passages; (ii) the intracerebral route of inoculation; and (iii) the use of newborn hamsters, in which there is a receptive bed of germinal tissue exposed to the virus.

The ability of rat virus to attack selectively the actively proliferating germinal layer of the neonatal cerebellum is in keeping with the known property of this virus to grow preferentially in tumor-bearing rats and pregnant hamsters (*1, 4, 6, 8*). Just as with irradiation (*9*) and antimitotic agents (*10*), it appears that the target of the rat virus is the dividing cell. This predilection for growing tissues may furnish a clue to the pathogenesis of the mongoloid dwarfism (*3*) and the tooth dysplasia (*4*) produced in hamsters by rat virus.

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Inquiline Roach Responds to Trail-Marking Substance of Leaf-Cutting Ants

Abstract. *Nymphs and females of the roach inquiline,* Attaphila fungicola *W. M. Wheeler, respond to odor-trail substances of* Atta texana *(Buckley) and* Trachymyrmex septentrionalis *(McCook). The ants are more sensitive than roaches to the pheromone.*

Attaphila fungicola W. M. Wheeler is a small, wingless roach that inhabits the fungus gardens of most nests of the Texas leaf-cutting ant or town ant, *Atta texana* (Buckley). It is one of the few inquilines that live intimately with the ant.

Table 1. Response to odor-trail substances of *Atta texana* and *Trachymyrmex septentrionalis* by their workers and females of *Attaphila fungicola.* The response is the number of insects following circular trail 15 cm or more, and is the sum of two replications of ten insects each.

Test insects	Dilution of contents from one poison sac in 1 ml of CCl_4			
	1	10	10^2	10^3
Major worker *Atta texana**				
A. texana (mw)†	20	19	12	0
A. fungicola (♀)	20	18	0	0
Minor worker *Atta texana*‡				
A. texana (mw)	20	12	0	0
A. fungicola (♀)	16	0	0	0
Worker *Trachymyrmex septentrionalis*§				
T. septentrionalis	20	14	0	0
A. texana (mw)	16	10	0	0
A. fungicola (♀)	16	2	0	0

*Body length, 10.0 mm; poison sac, 0.9 by 0.65 mm. †Minor workers, mw; ‡Body length, 3.5 mm; poison sac, 0.2 by 0.2 mm. §Body length, 3.0 mm; poison sac, 0.2 by 0.2 mm.

Numerous publications describe inquilines on ant trails, but in no case is it clear that they follow the ant scent.

The bioassay technique described by Moser and Blum (*1*) was used to measure the response of the roach to trail-marking substances from the poison sac of *Atta texana* and a related ant, *Trachymyrmex septentrionalis* (McCook). Each poison sac was crushed in 1 ml of carbon tetrachloride; the solution was shaken thoroughly and 0.1 ml was applied to a sheet of paper in a narrow line, describing a circle 15 cm in diameter. Roaches were then placed inside the circle and records made of the number that followed the line for 15 cm or more. To determine the lowest concentration that roaches and ants could detect, similar trials were made with serial dilutions. Female roaches only were used since the male roach has never been found in central Louisiana. The roach specimens were taken from nests of town ants.

The trail-marking substances from the two species of ants were less attractive to the roach than to the ants, but the response of the roach to contents of sacs of equal size from ants