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A HISTORY OF CHIROPTERAN RABIES WITH SPECIAL REFERENCE TO OCCURRENCE AND IMPORTANCE IN THE UNITED STATES[1]

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INTRODUCTION

The term "rabies" denotes a pattern of characteristic and definitive changes in the central nervous system of certain animals, brought about by a specific agent, a virus. These changes in the nerve tissue do not always produce characteristic observable symptoms, but the agent in atypical forms of the disease may produce characteristic symptoms of the typical form when introduced into other animals.

The two general forms of the virus are "street" virus and "fixed" virus. Street virus is the virus present in animals developing the disease under natural conditions. Fixed virus is the virus after undergoing many serial passages through the brain tissue of experimental animals. The fixed virus has marked permanent changes in length of incubation period, infectivity, type of reaction by inoculated animals, and loss of or change in Negri bodies.

Diagnosis of the disease is accomplished by means of the clinical symptoms, by demonstrating the presence of Negri bodies in the brain tissue accompanied by other tissue changes, and by testing living animals with inoculations of suspected infected tissues. If a virus is isolated, it may then be subjected to various testing procedures to establish whether or not it is really rabies virus.

The Negri bodies mentioned here are inclusions in the tissue of the central nervous system which are specific to rabies. They are found exclusively in the cytoplasm of certain nerve cells, especially in the pyramidal and ganglion cells of Ammon's horn of the hippocampus, the Gasserian ganglion (Lapi. et al., 1952), and other areas of the brain. They are usually found in normal-appearing cells rather than in degenerated cells. The Negri body itself is acidophilic (eosinophilic) and characteristically possesses a single basophilic granule, although large Negri bodies may contain more than one such granule. The size of the Negri body varies from 0.25 to 27.0 microns. Although the presence of Negri bodies is considered to be a positive diagnosis for rabies, there is a possibility of incorrect diagnosis of rabies due to similar cytoplasmic inclusions found by Szlachta and Habel (1953) in non-rabid cat brains. The failure to detect Negri bodies does not prove the absence of the virus or the disease in the animal, especially in the case of the virus' being present only in the hibernating fat of the animal (Sulkin, et al., 1957).

The disease produced by the rabies virus is infectious; that is, the virus is transmissible to other animals, usually by means of the introduction of saliva containing the virus into a bite-created wound.

The virus is readily transferred to experimental animals by inoculation with infected tissue, usually that of

the central nervous system or of the salivary glands. Perhaps the two most important possible sources of infection other than the saliva are the blood and the milk of infected animals. The presence of the virus in these fluids has been a controversial subject, particularly in reference to presence and infectivity of the virus in the blood. This subject will be discussed in relation to transmission of the virus from bats to wildlife.

Progression of the infection follows a general pattern that may be designated as a series of periods; introduction, incubation, and invasion. During the incubation period, some local irritation, as itching or general discomfort, may occur at the site of entry of the virus. In general, the tissues of the body show no reaction to the virus until later. During the incubation period, the virus is transmitted from the site of entry throughout the central nervous system. According to Webster (1937 and 1942), it is probable that the virus is transmitted in association with the regional nerves, rather than by the blood stream. During the invasive period, the virus progresses to the salivary glands, causing the animal to be a source of infection to others. The animal at this time exhibits abnormal behavior, characterized by hypersensitivity or excitement in the "furious" form and by gradual paralysis in the "dumb" form. In the dumb form, and in the time following the activity of the furious form, there is an ascending paralysis. Some animals exhibit intermediate

forms between these extremes, as in the "fulminating" form in guinea pigs, which is characterized by rapid and almost asymptomatic death. Fracastorious, in 1546, according to Roueche (1957), mentioned the bite of a rabid dog as being "the incurable wound." This dramatic and picturesque phrase reflects the fact that, in cases where the symptoms have developed to an observable point, death almost always follows. The possibility of natural recovery from an infection, however, is indicated by the presence of rabies-neutralizing antibodies found in the serums of animals in the wild, particularly in bats (Burns and Farinacci, 1955). A certain percentage of mammals, including man (van Rooyen and Rhodes, 1948), actively exposed to infection by the rabies virus fail to develop the disease. This may be an indication of a possible latency of infection, as suggested by Koprowski (1952), rather than recovery. Vampire bats have been observed to recover from the disease (Pawan, 1936a), a topic to be covered more fully later in this paper.

From the present evidence, it would appear that most all mammals are susceptible to infection by the rabies virus. According to Webster (1942), all mammals in nature constitute potential reservoirs for the virus. Although obviously incomplete, the following general listing of mammals found naturally infected with the rabies virus is given to illustrate this potential danger: dogs, red and grey foxes, coyotes, wolves, domestic

cats, lynxes, mountain lions, striped and spotted skunks, weasels, raccoons, rats, mice, squirrels, beaver, horses, cattle, moose, caribou, hogs, sheep, rabbits, opossum, mongooses, and bats. It should be noted that both herbivorous as well as carnivorous mammals are included in this listing.

Birds are also susceptible to the rabies virus, and some cases of rabies in birds may give clues to the overall picture of the disease in all its forms. Remlinger and Bailly, as reported by Malaga-Alba (1954), stated that rabid fowl often recover from almost complete paralysis, and that the virus in a latent state has been recovered from brain tissue of such fowl. Van Lote succeeded in infecting rabbits and guinea pigs with the brains of two eagle owls which had died without manifesting any symptoms of rabies two and one-half and nine months, respectively, after being inoculated (Malaga-Alba, 1954).

BAT-TRANSMITTED RABIES IN AREAS OTHER THAN THE UNITED STATES

Bat-transmitted rabies has been known since 1908, but has been confused with other diseases, making an exact history difficult. The disease has been incorrectly identified as botulism in cattle and a form of poliomyelitis in humans in Trinidad (Hurst and Pawan, 1932), rinderpest of cattle in Brazil (Metivier, 1935), and trypanosomiasis of cattle in Paraguay (Metivier, 1935).

The disease has occurred in such unusual forms as the bulbar paralysis first noted in cattle in Trinidad in 1925, which later changed to an ascending myelitis more like typical rabies. The disease has been given a wide variety of names: "mal de caderas" (Carini, 1911) and "peste das caderas" (Metivier, 1935) of cattle in Brazil; "enzootic paraplegia", "bovine mal de caderas" (Metivier, 1935), and "tumbi baba" (Malaga-Alba, 1954) in Paraguay; "rabia paresiante" (Malaga-Alba, 1954) in Argentina; "renguera" (Malaga-Alba, 1954) in Costa Rica; "derriengue" (Malaga-Alba, 1954) in other Central American countries; and "huila", "derrengue", "tronchado" (Malaga-Alba, 1954), or "derriengue" (Johnson, 1948) in Mexico.

A paralytic disease called "mal de caderas de bovinos" was first reported in epizootic proportions in horses, mules, and cattle in the coastal state of Santa Catharina, Brazil, in 1908 (Carini, 1911). The disease, according to Pawan (1936a), destroyed over 4,000 bovines and 1,000 equines in this and neighboring states in Brazil. It was not until 1911 that Carini identified the disease as rabies by the finding of Negri bodies and by animal inoculation. Although Carini noted that canine rabies was rare in the areas where infection was present, control of the disease was attempted by killing dogs. During a six-month period in 1912, more than 6,799 dogs were killed and canine importation was forbidden. This program had no influence on the incidence

reported. Carini came to the conclusion that the disease was being transmitted by a wild animal. Swiftly flowing rivers were no barrier to the spread of the disease; and, as ranchers had observed bats attacking and biting cattle even in daytime, Carini suggested that the transmitting agent could be the bat.

According to Pawan (1936a) Haupt and Rehaag studied the outbreak from 1913 to 1918 and were able to produce evidence that rabies was being transmitted by bats. The first infected bat discovered was a leaf-nosed bat found on a calf in a protected stall in Blumenau, Brazil. The evidence suggested that the bat had bitten the calf, which subsequently developed paralytic rabies (Pawan, 1948). Investigation by Haupt showed that the normally non-sanguivorous bats of the family Phyllostomidae may at times ingest blood, and Haupt and Rehaag were able to produce rabies in animals inoculated with the brain tissue of a leaf-nosed bat, Phyllostoma superciliatum. Sanderson (1955) stated that Phyllostoma acquires the rabies virus from vampire bats of the family Desmodontidae.

In 1925, cattle near Port-of-Spain, Trinidad, were dying from a form of bulbar paralysis incorrectly diagnosed as botulism. By 1926 the disease had spread west to Diego Martin, and by 1930 had assumed a different clinical form. The new form was that of an acute ascending spinal paralysis similar to the paralytic bovine rabies of South America, which

by this time had been recognized in Paraguay, Uruguay, Argentina, Bolivia, British Guiana, and Venezuela (van Rooyen and Rhodes, 1948; Gilyard, 1945; Metivier, 1935).

In July, 1929, a human case of acute ascending myelitis occurred in southwestern Trinidad, followed by 12 more cases in three months (Hurst and Pawan, 1931). In July and August of 1930, there were three more cases, followed by another in early 1931. Pawan (Hurst and Pawan, 1931) forwarded brain tissues to Dr. Hurst of the Lister Institute and Dr. Flexner of The Rockefeller Institute. The diagnosis of both these workers was that the disease in both man and cattle was rabies, as Pawan had suspected. No official case of human rabies had been known in Trinidad since 1914 (Hurst and Pawan, 1931), with the exception of one diagnosed by Pawan (1938) in 1919.

No cases of rabid dogs were known and destruction of dogs did not lower the number of cases. The heaviest mortality of humans and domestic animals occurred in the most thickly wooded districts. Quarantine laws of six month isolation periods for canines before admission to the island were strictly enforced. There was no history of animal bite or evidence of injury in the 17 cases reported up to 1931. Until experimental work indicated otherwise, it was thought that transmission might be accomplished by means of walking on dead frogs frequently seen lying on the roads, entrance of the virus being through wounds

on the sole of the foot (Pawan, 1936a).

Hurst and Pawan (1931) expressed the opinion that the vampire bat might be the vector. This was based on the fact that vampire bats attack at night without arousing humans from sleep and that many of the cases had numbness of the foot as an initial symptom. The 1935 records of Dr. Rankine, the Medical Inspector of Health of the Colony of Trinidad, showed that of the 87 persons bitten by bats in that year, 60 were not aware of the bites until morning (Pawan, 1936a).

In July, 1931, a woman bitten on a toe by a bat died from rabies, the incident coinciding with the appearance in the same district of bovine rabies. At this time, it was also noted that bats were biting cattle. In September of the same year, a boy was bitten by a bat and also died (Hurst and Pawan, 1932). A third case of rabies in a human with a history of bat bite occurred soon afterward. Although vampire bat transmission was not proven, the three case histories of bat bite followed by death from rabies was considered suggestive of such transmission.

During the height of the rabies epidemic, about 200 bats were caught in the city of Port-of-Spain and examined for the presence of the rabies virus. The first bat found to be infected was identified as the fruit-eating bat, Artibeus planirostris trinitatis (Pawan, 1936a). Accounts were given at

this time of bats, probably fruit-eating species, flying from fruit trees and attacking animals, including man. Negri bodies were demonstrated in the brain tissue of a total of five Artibeus bats by 1936 (Pawan, 1948). According to Reagan (1951), De Verteuil and Urich considered Artibeus to be a reservoir of the virus for Desmodus rotundus murinus, the main vector. Although De Verteuil and Urich knew that Artibeus harbored the virus, they did not believe that this species would attack man.

Of 2,059 bats examined from October, 1934 to September, 1935, 64 bats were found positive for the presence of Negri bodies. A total of 59 of these positive bats were identified as vampires, Desmodus rotundus murinus, with four Artibeus planirostris trinitatis and one Hemiderma (now Carollia) completing the number. A rare white bat, Diclidurus albus, also was found to be infected and was used in animal experimentation along with a number of other naturally-infected bats (Pawan, 1936a). The vampire bats were proven to be infected by means of histological examination, animal inoculation, cross-immunity testing, and serum neutralization tests.

Of 31 cases of human rabies in Trinidad, 23 gave evidence of bat bite on the toes or foot (Pawan, 1936a). Dalquest (1955), however, noted that the most frequent site of vampire bat bite on humans in Mexico was the cheek.

Periods of prevalence of paralytic rabies in both man and domestic animals coincided with reports of abnormal aggressiveness and daytime flight of bats in both South America and Trinidad. Man and other animals have been bitten by vampire bats on the island of Trinidad for a period of over 60 years with no cases of bat-transmitted rabies having been reported until 1925.

The source of the bat-transmitted rabies virus in Trinidad is a matter of conjecture. Van Rooyen and Rhodes (1948) suggested that the first infected bats flew or were carried by ship to Trinidad. Bats have been reported flying from the mainland of South America toward Trinidad (Pawan, 1936a). The distance from the mainland is nine miles at one point and 12 miles at another, with islands found between the mainland and the 12-mile point. The presence of vampire bats on the island of Chacachacare, midway between Venezuela and Trinidad (Pawan, 1936a), provides another possible link. Gallo and Iturbe, as quoted by Johnson (1948), stated that a paralytic disease of cattle had been known in the state of Miranda, Venezuela, since 1925.

Although an isolated incident, in 1889 a chemist engaged in dissecting vampire bats to determine the stomach contents developed a fatal acute ascending spinal paralysis (Pawan, 1936a). This led Pawan to suggest the possibility of a latent unrecognized source of the virus on the island itself.

In September, 1953, six miners of the Mazaruni District of British Guiana died from a rapid paralytic disease. Inquiry revealed that three miners had died in August and early September of the same year with similar symptoms. Negri bodies were demonstrated in the tissue of the five brains that were tested. As the disease began with a tingling sensation in the toes or fingers, and miners in this district are frequently bitten by vampire bats, bat transmission of the virus was suggested by Nehaul (1955). Paralytic rabies in cattle had been noted some time earlier in this district. These cases are the first reported human paralytic cases from continental South America.

In 1950, at La Ceiba, Honduras, serious cattle losses occurred, and rabies virus was identified from cattle brain tissue by means of mouse inoculation and the demonstration of inclusion bodies in the tissue. Cattle losses were greatest where the bat populations were highest, and other potential sources for transmission of the virus were not apparent. However, of 50 bat brains tested, all proved negative for rabies in mouse inoculation tests. Transmission of the rabies virus by bats was suggested by Schroeder (1955) in spite of this lack of demonstrably infected bats.

The disease of cattle known as derriengue has been prevalent in the Pacific coast states of Mexico since about 1910 (Johnson, 1948). In 1932 Giron isolated from cattle a

virus considered to be the specific etiological agent of derriengue, but which was not proven to be rabies virus. Dr. TenBroeck of The Rockefeller Institute for Medical Research isolated a virus from the brain tissue of a cow which died from derriengue in the State of Jalisco in 1943. The virus was found to be antigenically related to known strains of rabies virus (Johnson, 1948). Johnson conducted field investigations of the disease for The Rockefeller Foundation in 1944 in the States of Jalisco and Michoacau. Vampire bats (Desmodus rotundus murinus) were found in the immediate vicinity of each focus of the disease. Rabies virus was isolated from salivary glands and brain tissue of a paralyzed cow and vampire bats captured near by in one area. The virus was studied and compared to known strains of rabies virus by means of animal inoculations and cross-neutralization, complement fixation, and protection tests (Johnson, 1948).

In April, 1951, the first case of vampire bat rabies in man outside of Trinidad was noted by Dr. Landa in the State of Sinaloa, Mexico (Malaga-Alba, 1954). Of ten people bitten in an attack by a furious bat in the village of El Platanito, a man and four children died from rabies. All exhibited typical symptoms of paralytic rabies and died within a few days following the onset of symptoms (Campillo and Malaga-Alba, 1957). In June of the same year, seven persons in Bolanos and Chimaltitlan in the State of Jalisco were bitten by vampire bats. Three died with

clinical symptoms of paralytic rabies, as quoted from Acosta by Malaga-Alba (1954). In May, 1952, Camargo reported a case in Ixtlan del Rio, State of Nayarit, where derriengue was enzootic (Malaga-Alba, 1954). Desmodus rotundus vampire bats sent for investigation were found to be negative for rabies (Campillo and Malaga-Alba, 1957).

Malaga-Alba, in an Annual Report to the Pan American Sanitary Bureau in 1952 (Malaga-Alba, 1954), noted that bats of species Molossus nigricans and Macrotus mexicanus had been observed attacking man and other animals. No rabies tests were made as specimens sent for examination were decomposed.

In the Palo Bolero Cave in the State of Morelos, Mexico, Malaga-Alba found two dying bats which later proved positive for rabies. This occurred in September, 1952. Malaga-Alba (1953) noted that vampire bat rabies is found in the States of Sonora, Chihuahua, and Tamaulipas, all of which border the United States.

In 1954, three cases of human bat rabies deaths were reported in Jalpa, in the State of Zacatecas, Mexico. In 1955, two additional cases were reported, one in Coatzamala de Pinzon and one in Taxco, both in the State of Guerrero. The case which occurred in Taxco was the first case of human rabies transmitted by bats diagnosed in Mexico with laboratory evidence, as reported by Campillo and Malaga-Alba (1957).

Grimes, et al. (1955) reported rabies virus isolation from bats of species Chilonycteris personata in Mexico in 1954. J. E. Werler, Curator of Reptiles of the San Antonio Zoological Society, Texas, collected two bats of this species from a cave near San Andres Tuxtla, State of Veracruz, in February, 1954. The cave from which the bats were taken was also inhabited by vampire bats. Both bats died shortly after being flown to the San Antonio Zoo, and one specimen was turned over to the State Department of Health at Austin, Texas. Mouse inoculations were positive for rabies.

In contrast to the 4,000 cattle killed by vampire bat rabies in Brazil at the time of the first report (Carini, 1911), a total of 50,000 cattle were reported killed in 1956 in Rio Grande do Sul by the same disease, according to Time magazine of April 28, 1958.

A Wildlife Disease Association newsletter of August, 1956, noted that a man bitten by a bat in Germany in 1955 subsequently died from rabies. An article entitled "Rabies in bats, moles, and cattle" (1958) in the Journal of the American Veterinary Medical Association reported the isolation of rabies virus from bats in northeastern Yugoslavia. The virus was detected in four insectivorous bats of species Nyctalus noctula, but was found in brain tissue only and was considered to be a passive form. For this reason, bats were not thought to play a part in the epizootiology of rabies in that

country. Schindler and Dennig (1958) note that the disease in Europe progresses uniformly rather than sporadically as it would if the bats were responsible, as epidemiological evidence against such transmission.

Stovell (1959) has reported four cases of rabies in bats in British Columbia. In June, 1957, a big brown bat was turned over to the Vancouver City Health Department after it had bitten a boy who picked it up. As it was found to be rabid, over 200 specimens of seven species were collected and examined by the Health of Animals Division at Vancouver in pooled lots of six each. One pooled unit of little brown myotis bats from the Point Grey area was positive and two of the bats had unusual histories. In July of the next year, another big brown bat was found positive for rabies after having bitten a boy in the Okanagan Valley when he picked it up. The last case involved a silver-haired bat (Lasionycteris noctivagans) found in an old shoe at Osoyoos, where it had bitten the finder. The people bitten in these episodes were given anti-rabies treatment and remain well to date.

BAT-TRANSMITTED RABIES IN THE UNITED STATES

Although experimental transmission of rabies virus to insectivorous bats in the United States was reported by Reagan and Breuckner in 1951, no cases of naturally infected insectivorous bats were reported until 1953. Since that time,

a total of 17 infected insectivorous bat species have been reported from 19 states. Figure 1 shows the year of the first discovery of bat rabies in each state involved to date. The bat species found rabid in each state involved to date are listed in Table 1.

The first state in the United States to report the detection of rabies in bats was Florida. On June 25, 1953, a lactating female Florida yellow bat (Dasypterus floridanus) made an unprovoked attack on a young boy in Hillsborough County, Florida. The bat remained strongly attached until removed and killed. Typical Negri bodies were found in the brain tissue of the bat and mouse inoculations were positive for rabies. Complement fixation and serum neutralization tests also were positive for rabies (Scatterday, 1954). The boy became ill 28 hours following the bite and received anti-rabies treatment, after which no ill effects were reported (Venters, et al., 1954).

Of 208 apparently normal non-colonial bats subsequently shot in evening flight, six proved positive for rabies. One was a Seminole bat (Lasiurus seminolus), and five were Florida yellow bats. Five of the six had demonstrable Negri bodies in the brain tissue. Brain tissue from the remaining bat caused mouse death with demonstrable Negri bodies in the mouse brain tissues. Five additional bats possessed atypical inclusions, but mouse inoculations proved negative for rabies. Of 15 colonial bats

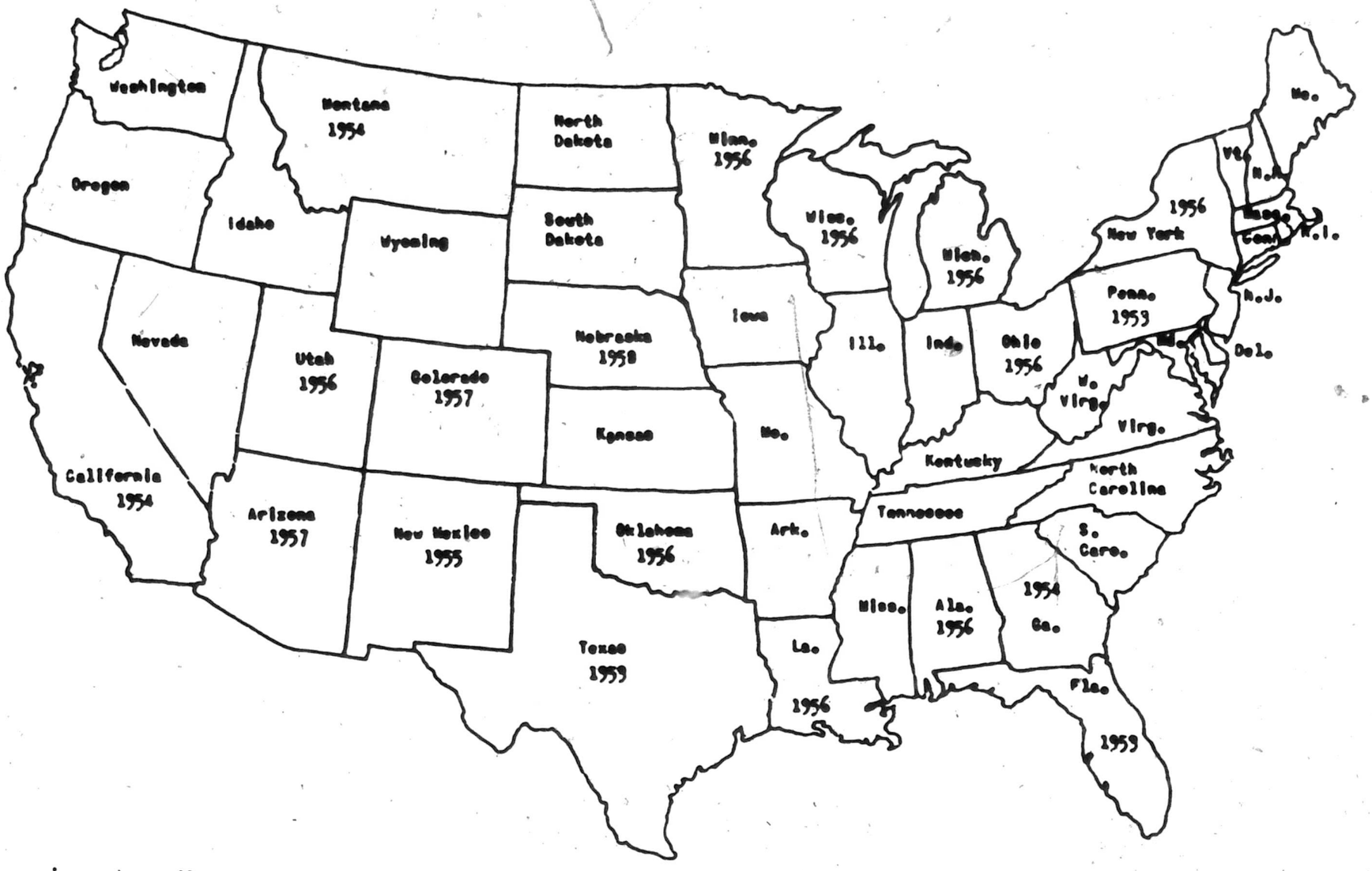

Fig. 1. Year of the United States showing year of first discovery of tsetse flies in each state involved.

Table 1. Occurrence by state of bat species found naturally infected with rabies, to January, 1959.[1]

	Alabama	Arizona	California	Colorado	Florida	Georgia	Louisiana	Michigan	Minnesota	Montana	Nebraska	New Mexico	New York	Ohio	Oklahoma	Pennsylvania	Texas	Utah	Wisconsin
Little brown myotis (Myotis lucifugus)	0	0	X	X	0	0	0	0	0	0	X	0	0	0	0	X	0	0	X
Cave myotis (Myotis velifer)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	X	0	0
California myotis (Myotis californicus)	0	0	X	0	0	0	0	0	0	X	0	0	0	0	0	0	0	0	0
Gray myotis (Myotis grisescens)	0	0	0	0	X	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Southeastern myotis (Myotis austroriparius)	0	0	0	0	X	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Long-eared myotis (Myotis evotis)	0	0	X	0	0	0	0	0	0	X	0	0	0	0	0	0	0	0	0
Long-legged myotis (Myotis volans)	0	0	0	0	0	0	0	0	0	X	0	0	0	0	0	0	0	0	0
Eastern pipistrelle (Pipistrellus subflavus)	0	0	0	0	X	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Big brown bat (Eptesicus fuscus)	0	X	0	0	0	0	0	X	0	X	0	0	X	X	0	0	0	0	X
Pallid bat (Antrozous pallidus)	0	X	0	0	0	0	0	0	0	0	0	0	0	0	0	0	X	X	0

	Alabama	Arizona	California	Colorado	Florida	Georgia	Louisiana	Michigan	Minnesota	Montana	Nebraska	New Mexico	New York	Ohio	Oklahoma	Pennsylvania	Texas	Utah	Wisconsin
Florida yellow bat (Dasypterus floridanus)	o	o	o	o	x	o	o	o	o	o	o	o	o	o	o	o	x	o	o
Hoary bat (Lasiurus cinereus)	o	x	x	o	o	o	o	o	x	x	o	x	o	o	x	x	x	o	o
Red bat (Lasiurus borealis)	x	o	o	o	x	x	o	o	o	o	o	o	o	o	o	o	x	o	o
Seminole bat (Lasiurus seminolus)	x	o	o	o	x	x	o	o	o	o	o	o	o	o	o	o	o	o	o
Mexican free-tailed bat (Tadarida brasiliensis mexicana)	o	x	x	o	o	o	o	o	o	o	o	x	o	o	x	o	x	o	o
Florida free-tailed bat (Tadarida brasiliensis cynocephala)	o	o	o	o	x	x	x	o	o	o	o	o	o	o	o	o	o	o	o
Big free-tailed bat (Tadarida molossa)	o	o	o	o	o	o	o	o	o	o	o	x	o	o	o	o	o	o	o

[1] An x indicates the record of at least one occurrence and an o indicates the absence of such a record.

shot on the wing, all were negative for rabies virus (Venters, et al., 1954). Scatterday and Galton (1954) reported that of 385 bats of eight species killed in evening flight by shotgun, six were found to be positive for rabies. Three were Florida yellow bats and three were Seminole bats. Atypical inclusions were found in one bat, but it was confirmed as positive for the rabies virus by further tests. One bat brain produced typical symptoms of rabies in mice, but no Negri bodies were detected in either the bat brain or in the mice brains through two passages. The Florida yellow bat was considered rare at the time of the first attack, but the rabies investigations which followed showed it to be the most commonly taken species in central Florida.

A search for the source of infection followed these preliminary studies. The possibility of accidental introduction of bats with imported materials was discounted by banana importing companies, who demonstrated that the possibility of stowaways was slight, and determined that stowaway bats had never been seen (Scatterday and Galton, 1954). The fact that careful observers have stated that bats are absent from the Florida Keys decreased the possibility of the presence of an inter-island route for contact between the Florida bats and Jamaican fruit bats or vampire bats (Schneider, et al., 1957).

Schneider, et al. (1957) made a two and one-half year study of bat rabies in Florida. A total of 5,503 bats of 12 spe-

cies was collected and 31 were found positive for rabies. None of the eight rabies-infected colonial bats exhibited Negri bodies, while 15 of the 25 free-living bats had detectable Negri bodies.

Colonial species found infected were: the southeastern myotis (Myotis austroriparius), the gray myotis (Myotis grisescens), the eastern pipistrelle (Pipistrellus subflavus), and the Florida free-tailed bat (Tadarida brasiliensis cynocephala). Free-living species found infected were: the red bat (Lasiurus borealis), the Seminole bat, and the Florida yellow bat.

The four methods of collecting the sample bats influenced the proportions of infected bats found as indicated by figures from the study as of December, 1956. Of 3,675 bats collected from colonies, 0.2 per cent were found infected, while of 1,222 bats shot in flight, 1.2 per cent were found infected. Bats collected from Spanish moss processing plants had 1.7 per cent of 296 infected, and of the 35 received by donation, 14.0 per cent were infected (Scatterday, 1958a). Among the colonial species the proportion infected was 0.2 for the total group, while the free-living species had 1.5 per cent of the total group infected.

Neutralizing antibodies for the rabies virus were not found in the 49 five-bat serum pools tested. The bats were apparently normal but had been taken from colonies in which infected bats had been discovered (Schneider, et al., 1957).

A Veterinary Public Health newsletter from the Communicable Disease Center at Atlanta, Georgia, reported that in August of 1957 four more cases were discovered. The bats involved were a red bat, two Seminole bats, an eastern pipistrelle, and one not identified. Another C.D.C. newsletter reported that a man in Jacksonville, Florida, was attacked by a Seminole bat while driving his car on June 3, 1958. Inclusion bodies considered to be Negri bodies were found in the brain tissue of this bat. Four more bat rabies cases were reported in another C.D.C. newsletter, covering the period of January through August 15, 1958. Scatterday (1958b) reported a rabid Florida yellow bat for the fall of 1958 in Florida.

Scatterday and Galton (1954) suggested the possibility of a connection between the presence of rabies in Florida bats and the increase in sporadic, unexplained cases of rabies in wildlife in Florida, particularly in raccoons, since 1951. Schneider and co-workers (1957) stated that the role of the bat as a reservoir for rabies in wildlife and domestic animals may be presumed, though not proved, and that the public health implications are apparent, especially with regard to man.

The second state to report a case of human attack by a rabid bat was Pennsylvania. On September 29, 1953, a woman in Boiling Springs, Pennsylvania, was bitten on her left arm in an unprovoked attack by a ba.. Typical Negri

bodies were found in the brain of the bat, and rabbit inoculations proved positive for rabies. Although the body of the bat was discarded before official identification, identification of museum skins by those people concerned and the bite marks on the woman's arm gave evidence that the bat was a hoary bat (Lasiurus cinereus), as reported by Dr. Witte of the Pennsylvania Department of Health (1954). Four little brown myotis bats (Myotis lucifugus) and three big brown bats (Eptesicus fuscus) shot by the Pennsylvania Game Commission in the vicinity of the attack were found negative for rabies (Kough, 1954).

Another rabid bat attack on a human in Pennsylvania occurred on November 28, 1953, in Harrisburg. A man inside a tavern was attacked and bitten severely on the hand, but the bat was killed and thrown out, allowing no testing for rabies or species identification (Roueche, 1957). The bat carcass was recovered later, but had been so mutilated by animals that there was insufficient brain tissue available to make an accurate diagnosis for rabies (Witte, 1959).

A newsletter from the C.D.C. at Atlanta reported an account by Dr. Witte of another case on May 18, 1958. A man was bitten by an attacking bat in a home in Bangor, Pennsylvania, and the bat brain was subsequently tested for rabies. Although Negri bodies were not detected, mouse inoculations were positive for rabies. The bat's body was

discarded before a species identification could be made, but was later identified as a little brown bat, possibly Myotis lucifugus, by the persons who saw it (Witte, 1959).

Because of the number of cases of bat rabies reported in the United States, a formerly unexplained case of human rabies in Texas was examined in retrospect by Sulkin and Greve (1954). A woman bitten by a bat picked up from a roadside in Parkland County, Texas, in October of 1951 died a month later from rabies. At the time of the death, no connection was made between the bat and the source of the woman's infection. Her death was considered by Sulkin and Greve to be the first human death from bat rabies in the United States.

In November of 1953, rabies virus was isolated from one Mexican free-tailed bat (Tadarida brasiliensis mexicana) in Austin, Texas, and from another found near San Antonio (Quist, et al., 1957).

Grimes and his associates were quoted by Sulkin and Greve (1954) as having demonstrated Negri bodies in the brain of a Mexican free-tailed bat which had bitten a young boy in Austin, Texas, on May 19, 1954.

A United States Department of Health, Education, and Welfare report (1954) noted that two of 72 insectivorous bats studied at a Texas military reservation were rabid. These bats, all Mexican free-tailed species, were collected

on several occasions, the first being in February of 1954 at Fort Sam Houston, Texas. The second collection was made in May of 1954, at the time of the appearance of a malady in the bats characterized by various stages of paresis and frequent deaths. This was believed to be the result of an intensified DDT insecticide program then in effect. Five individual bats were collected in April and May of the same year, all exhibiting forms of paralysis. Of these five, two were collected approximately two weeks before the insecticide program was started. Of the last five bats collected, two proved to be rabid, the identification being confirmed by neutralization tests after the detection of Negri bodies and positive mouse inoculations (Burns and Farinacci, 1954 and 1955).

Serum collections from apparently normal bats in the Fort Sam Houston area were made in June of 1954. Antibody studies revealed that serum from 65 per cent of 207 bats contained a rabies virus neutralizing substance. Inactivation of the bat serum did not eliminate this substance in the blood, so it appears that this substance may be specifically related to immunity (Burns and Farinacci, 1955).

Evidence that the malady previously reported in the Fort Sam Houston bats was not the result of insecticide use came when the malady continued for six months after insecticide spraying was discontinued. The malady also

occurred in a bat colony 20 miles away, where no spraying had been done (Burns, Farinacci, and Murnane, 1956). Perhaps connected to this malady was the presence of four neurotropic viruses other than rabies isolated from a colony of Mexican free-tailed bats at Fort Sam Houston. These viral isolates were later reported to be antigenically related to the virus of St. Louis encephalitis (Burns and Farinacci, 1956) and to Group B arthropod-borne encephalitis viruses, probably representing a distinct new entity (Burns, et al., 1957).

Sullivan, et al. (1954) tested 200 bats in Texas and found that two of 151 Mexican free-tailed bats were rabid. Both rabid specimens were taken in abnormal locations.

Studies of four Texas bat colonies (Burns, Farinacci, and Murnane, 1956) yielded a total of 27 out of 44 serum pools positive for rabies neutralizing antibodies. Thus approximately 61 per cent of the 283 Mexican free-tailed bats from which the serum pools were obtained were considered to have had past experience with the disease. The percentages of bats exhibiting the neutralizing antibodies varied from 16.6 to 79.0 per cent in the four colonies. Isolation of the rabies virus was made from nine bats, only five of which exhibited detectable Negri bodies. These isolates were made from members of only two of the colonies, although all four colonies were positive for the presence of neutralizing antibodies. Of 40 cave myotis bats

(Myotis velifer) tested during the study, none proved positive for either the rabies virus or antibodies.

Eads and associates (1955) collected 329 Mexican free-tailed bats from May, 1954 to April, 1955 in Bracken Cave, near San Antonio, Texas. Of 98 brain and salivary gland tissue pools made from these bats, 12 were positive for rabies. The bats inhabiting this cave were considered to have a high mortality rate, possibly from rabies.

In a study covering areas in Texas, Arkansas, New Mexico, Louisiana, and Mexico (Burns, et al., 1956), a total of 1,247 bats of nine species were collected. Rabies virus was found in nine of 166 bat brains tested and in one of 19 pools of brain tissue, all from Mexican free-tailed bats taken in Texas. Neutralizing antibodies for rabies were found in 67 of 110 serum pools from 688 Mexican free-tailed bats. Rabies virus was isolated from one of 24 salivary gland tissue pools from cave myotis bats from Fort Hood, Texas. Of two 1955 tests of cave myotis bats from Bracken, Texas, six of eight serum pools from 44 bats and one of three serum pools from 15 bats possessed rabies neutralizing antibodies. One of 10 pallid bats (Antrozous pallidus) and two of six red bats also taken in Texas were found to be rabid. Only 17 per cent of the rabid bats had detectable Negri bodies, but Negri bodies were detected in 97 per cent of the mouse passage brain tissues.

Quist and co-workers (1957) found that three per cent of red bats tested were rabid, but considered this fact of minor public health significance because of the comparatively small numbers of red bats in Texas.

Of 22 cases of human bat bite reported in Texas during 1954 through 1956, five of the 15 bats involved were rabid. Two of the bats found positive for rabies were Mexican free-tailed bats, and the other three were red bats. All cases involving humans were the result of the bats being handled rather than from unprovoked attack (Irons, et al., 1957).

On January 4, 1956, George C. Menzies of the Entomology Section of the Texas State Department of Health died from rabies. Although a member of a team conducting bat rabies investigations, he was unable to give a history of bat bite or any other animal bite. A possible method whereby Mr. Menzies acquired the infection was suggested by his co-workers as being an accidental rubbing of a chronic skin eruption on his neck with contaminated gloves. Negri bodies were not found in his brain tissue but were detected in mice inoculated with the brain tissue (Irons, et al., 1957).

Tierkel (1957) listed the hoary bat as being one of the Texas species in which rabies had been detected. In the summer of 1958 a large number of Florida yellow bats and red bats were found to be rabid, as well as Mexican free-tailed

bats, bringing the total number of bat species found infected with rabies in Texas to six (Irons, 1958).

A colony of bats was taken from Thomasville, Georgia, by Scatterday and Galton (1954) during their study of bat rabies in Florida. Although 54 little brown myotis bats were found negative for rabies, two Florida free-tailed bats from this colony were found positive for rabies. In spite of this, the Georgia State Health Department reported what was called the first case of bat rabies in the state in a C.D.C. newsletter in the summer of 1956. A moribund Seminole bat was picked up on a sidewalk in Thomasville at that time and tested for rabies with the subsequent isolation of the rabies virus.

Rabies was isolated from two red bats found in Atlanta, Georgia, in August, 1956, according to another C.D.C. newsletter. One of the two bats was a female found fighting on the ground with a non-rabid male.

Enright (1956) reported that rabies virus was isolated from horses in southern California in 1944 with no vector identified for the disease. Then, in May, 1945, an adult Masai giraffe at the San Diego Zoo in San Diego, California, died from rabies. A woven wire fence had prevented contact between the giraffe and other animals, and there was no history of rodent-transmitted rabies in the San Diego area at the time (Schroeder, 1955). Hog-nosed bats (Choeronycteris mexicana) from the mainland of

Mexico were found in San Diego at a later date (Olson, 1947; Huey, 1954), and the possibility of bat transmission of rabies to the giraffe was suggested by Schroeder (1955). A man bitten at least four times by these bats subsequently became violently ill and had a prolonged seige of disability. Relief from illness involving headache, nausea, and high temperature came after four days of hospital treatment with penicillin, morphine, ice packs on head, and intravenous injection of saline and glucose. There was no apparent source for the infection other than from the bat bites (Olson, 1947). Hammon, as noted by Enright (1955), stated that the rabies virus isolates from both horses and the giraffe behaved in experimental animals as did strains isolated from vampire bats.

According to Courter (1954a), a 1952 Pan American Sanitary Bureau survey in southern California resulted in no positive proof of presence of vampire bats in that area, but circumstantial evidence was found. A member of the United States Fish and Wildlife Service had seen a bat he identified tentatively as a vampire bat. Morning discoveries of bleeding ears of stabled horses were suggestive of bat bite as no other definite cause could be found. Two cases of paralytic rabies in cattle in San Diego County had no history of animal bite, and rabies was not known to be present in dogs or coyotes in the immediate area. Paralytic cases of a rabies-like disease which ended in recovery were re-

ported as well. Ranchers in nearby northern Baja California, Mexico, had complained that bats were damaging the teats of nursing sows, indicating the presence of vampire bats in that area. This circumstantial evidence may possibly indicate a northward movement of vampire bats, as Dalquest (1955) noted their high adaptability and wondered why they hadn't ranged into the United States. Vampire bat rabies has been found in the Mexican States of Sonora, Chihuahua, and Tamaulipas, all of which border the United States (Malaga-Alba, 1953).

In Campo, California, one bat was killed in 1952 and another in 1953 after swooping down on children playing outdoors in the daytime (Malaga-Alba, 1954). The specimens were not saved for identification or testing.

A total of 211 bats of 14 species were collected from northern California and tested, but rabies virus was isolated from only one Mexican free-tailed bat. The rabid bat, taken in July, 1954, was normal in appearance and no Negri bodies were detected in the brain tissue. Negri bodies were detected in the brain tissue of first passage mice (Enright, 1955). Rabies virus was isolated in June, 1955, from the salivary glands of a California myotis bat (Myotis californicus) found dead in a fishpond, according to a communication from Johnson to Enright (1956). In October, 1955, rabies virus was isolated from the brain and salivary glands of a bat, possibly a Mexican free-tailed

bat which had bitten a man. According to Lennette, in a communication to Enright, the brain contained atypical inclusions.

Nine cases in California have since been reported by C. D. C. newsletters. Four cases in 1956 involved two hoary bats, one Mexican free-tailed bat, and one thought to be a myotis species. In 1957, a long-eared myotis bat (Myotis evotis) and a bat thought to be a Mexican free-tailed bat were involved in the only two cases reported. In 1958, two Mexican free-tailed bats and what was thought to be a hoary bat were reported as rabid. Negri bodies were not detected in brain tissue of one of the Mexican free-tailed bats nor in the questionable hoary bat, but mouse inoculations were positive for rabies.

The first clearly established case of human rabies death from bat bite in the United States was reported in such newspapers as the Wichita (Kansas) Eagle of December 2, 1958, and the Washington (District of Columbia) Post of February 26, 1959. On August 2, 1958 a woman from Magalia, Butte County community, California, was bitten by a bat she tried to pick up. From September 2 to September 26 she received antirabies treatment, but became ill on October 26. The illness terminated in her death on November 4, 1958. The bat was proven to be rabid by the detection of inclusion bodies, but information concerning the species of bat involved is not presently available.

A rabid bat was brought to the Rocky Mountain Laboratory of the Public Health Ser-

vice at Hamilton, Montana, on August 20, 1954. Identification of rabies was accomplished by means of finding Negri bodies, by mouse inoculation, and by neutralization and cross-protection tests. The prior arrival of a canvas-covered trailer from Ohio which was parked in the neighborhood where the bat was captured brought up the possibility of the bat's being a recent arrival, but that species of bat was found to be native to Montana. The bat was identified as a big brown bat, and was the first reported isolation from this species (Bell, et al., 1955).

Although 121 apparently normal bats of six species tested for the presence of rabies in a 1955 Montana study proved negative, three of six bats exhibiting aberrant behavior or found in unusual situations were found to be rabid. One infected bat was identified as a California myotis bat, a species of which little is known in regard to occurrence and distribution in Montana. The second infected bat was a big brown bat. The third infected bat was a hoary bat taken after it had flown headfirst into a tree trunk in daytime flight. Negri bodies were detected in the brain tissue of all three bats (Bell, et al., 1957).

Only one case was reported from Montana in 1956, occurring in October. The infected bat was identified as a long-eared myotis bat. Three cases were reported in Montana in July and August, 1957, the bats being identified as a long-eared myotis bat, a big

brown bat, and a long-legged bat (Myotis volans). These four cases all occurred in Ravalli County (Bell, 1958).

In a wide-spread study of bat rabies in the southwestern United States, rabies virus was isolated from 11 of 20 brains, from two of 16 sets of salivary glands, and from one of two salivary gland tissue pools taken at Carlsbad, New Mexico, from Mexican free-tailed bats. Of 28 serum pools from 140 bats of the same species from the same location, 15 were positive for the presence of rabies neutralizing antibodies (Burns, et al., 1956).

In August, 1955, bats at Carlsbad Caverns, New Mexico, were found dying in large numbers. The epidemic lasted for 10 days and was blamed on insecticide spraying for a time, but rabies tests made on dead and dying bats collected during the epidemic revealed the presence of rabies in over 50 per cent of those tested. In addition, serum samples from live bats collected in flight after the epidemic possessed rabies neutralizing antibodies. No human cases were involved (Roueche, 1957).

Tierkel (1957) listed the hoary bat as being one of the bat species in which rabies had been detected in New Mexico. A C.D.C. newsletter reported an isolation of rabies virus from one of about 15 big free-tailed bats (Tadarida molossa) found dead or dying in close proximity to one another in New Mexico in the late summer of 1958. This species was

reported to have been observed in New Mexico only once before. Three additional dead bats picked up in the same area were reported positive for rabies on mouse inoculation.

In July, 1956, a moribund bat identified as a pallid bat was brought to the Utah State Department of Health Laboratory after having bitten a young boy. Microscopic examination of the bat brain tissue was considered inconclusive but suspicious, and Negri bodies were later detected in both bat and first passage mice. Mouse inoculation was positive for rabies (Soffe, 1958; Jenkins, 1958).

A hoary bat found paralyzed on the University of Minnesota campus, Minneapolis, was reported to be rabid by the Minnesota Department of Health on August 30, 1956. Negri bodies were found in the brain tissue of both bat and first passage mice. The virus was confirmed by the Communicable Disease Center at Atlanta as being rabies virus by neutralization tests. In 1957, two human cases of bat bite occurred with atypical inclusion bodies being found in the brain tissue of the bats involved. Mouse inoculation, however, was negative for rabies. Both cases occurred in Hennepin County. Since 1953, 16 cases of human bat bite have been recorded, including the above, and six of the cases occurred in 1958 (Fleming, 1958).

Of 25 bats examined for rabies by the Minnesota Live Stock Sanitary Board from July 1, 1954, to November 26,

1958, only two were found positive for rabies. The laboratory reports concerning these examinations merely listed the animals as bats without reference to species (Flint, 1958).

A Medical News note in the Journal of the American Medical Association (1958) reported rabies isolation from two bats taken in an Oklahoma University and Oklahoma State Department of Health laboratory research project in 1956. The note also reported a rabid bat found in 1957 by the Oklahoma State Department of Health Laboratories. The latter was a hoary bat taken in Enid, Oklahoma, in July, as reported by Hassler in a communication to Glass (1958a). The former two bats were Mexican free-tailed bats taken in a study by Glass (1958a) in which 1,323 bats from Oklahoma were collected and tested for rabies. The first of the Mexican free-tailed bats was one of two moribund bats taken from a cave in Woodward County on July 9, and the other rabid Mexican free-tailed bat was one of nine sick bats taken from a cave in Greer County on August 14. All bat brains tested in 1957 and 1958 during this study proved negative for rabies (Glass, 1958b).

A study of bat rabies in the southwestern United States (Burns, et al., 1956) revealed the presence of rabies virus in one of 19 brain tissue pools from 94 Florida free-tailed bats taken in New Orleans, Louisiana. Of 19 serum pools from these bats, 14 were positive for the presence of rabies

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neutralizing antibodies.

In 1956, a soldier on maneuvers in Louisiana was attacked and bitten by a rabid bat, according to Roueche (1957).

Of 100 bats examined by the Louisiana Department of Health, none was found positive for rabies (Hauser, 1958).

Two cases of bat rabies have been reported by the Alabama Department of Public Health. A United States Air Force man was attacked and bitten on the hand by a red bat in Montgomery in 1956. The bat brain possessed Negri bodies and virus was isolated from the salivary glands. The second case involved a red bat captured near Mobile and exhibited symptoms of encephalitis (Hosty, 1958). A C.D.C. newsletter reported that Negri bodies were detected in the bat brain tissue and that rabies virus was isolated from both the brain and salivary glands. Tierkel (1957) listed the Seminole bat as being a species from which rabies had been detected in Alabama, but a report of this was not handled by the Alabama Department of Public Health.

The New York Department of Health reported the isolation of rabies virus from a big brown bat in Rensselaer County in 1956. Negri bodies were detected in the bat brain and the virus was confirmed as being rabies by serum neutralization tests (Parker, 1956). Two more rabid big brown bats were collected in Suffern, New York, on July 15 and Septem-

ber 2 of 1958. Both bats were found in a moribund condition and rabies identification was made by mouse inoculation and serum neutralization tests (Cox, 1959). Bats tested in a mammal survey of New York State were all negative for rabies at last notice, in September 1958 (Palmer, 1958).

Only one isolation of bat rabies has been made in Ohio, being accomplished in 1956. One of 21 three-brain tissue pools from 63 big brown bats selected at random from a hibernating colony in central Ohio yielded rabies virus in mouse inoculation (Tjalma and Wentworth, 1957). Negri bodies were not detected in brain smears from the positive group, but were detected in brain tissue of first passage mice. Serum neutralization tests confirmed the diagnosis of rabies.

In November, 1956, the Michigan Department of Health reported in a C.D.C. newsletter that rabies had been isolated from a big brown bat. The bat had been found on the ground and had bitten a boy. Although no Negri bodies were detected in the bat brain, they were found in first passage mice. A second case involving a bat thought to be a big brown bat was reported by a C.D.C. newsletter in 1957. On October 27, 1957, a University of Michigan student was bitten by the bat which was lying on the floor of the basement of a house. Negri bodies were detected in the bat brain, but mouse inoculation results were not typical of ra-

bies.

Five cases involving rabid bats were reported from Arizona in 1957 (Maddy, et al., 1958). A pallid bat left at a museum in Tucson in April bit a man and died the following day. A big brown bat, taken from a colony in Willcox in which bats were found to be dying, bit a boy on May 14. A hoary bat, knocked down in daytime flight by a fisherman on the Verde River in Yavapai County, bit the man and died two days later, on May 20. Another hoary bat found on the ground in Tempe on May 28 was apparently unable to fly. On August 14, a Mexican free-tailed bat was found dead on the University of Arizona campus. Each of these bats proved positive for rabies when tested. A rabies case which could possibly have involved a rabid bat also was reported. A cat frequently seen trying to catch a solitary bat in Cornville, Arizona, in August, 1957, attacked a child on September 7 and possessed Negri bodies in the brain. The bat could not be found. In view of these findings, a Pima Indian belief took on a new significance, as they fear bats and say, "If a bat bites you, you will go crazy and die." (Maddy, et al., 1958).

Dr. Martin Baum of the Colorado State Health Department reported a case involving a sick bat which had bitten a young boy on May 28, 1957. As reported in a C.D.C. newsletter, results were inconclusive, as only one of 10 mice inoculated died. Inclusion bodies of some kind seemed to be

present in the mouse brain. Baum (1958) reported that bat rabies had been identified in Colorado on several occasions and that the little brown bat, probably Myotis lucifugus, had been incriminated.

The Wisconsin Department of Health reported two cases of bat rabies in the summer of 1957, according to a C.D.C. newsletter. A bat tentatively identified as a little brown myotis severely bit a boy in Janesville on May 2. The second case, in Eau Claire, involved a big brown bat which, in daytime flight, flew into but did not bite a judge as he was leaving the courthouse.

Although no positive cases of bat rabies have been reported in Kansas, inclusion bodies found in a brain smear from a hoary bat were tentatively identified as Negri bodies on June 29, 1957 (Kitselman, 1958). The bat was captured in the Kansas City, Kansas area and was one of about 65 bats of several species picked up at random throughout the state. The three-brain tissue pool containing the brain tissue from this bat was negative for rabies on mouse inoculation. No human exposure was involved.

Only one case of bat rabies has been reported from Nebraska. The case involved a little brown myotis bat which had scratched a girl in Lincoln on August 14, 1958. Negri bodies were detected in the brain tissue and mouse inoculation was positive for rabies (Olsen, 1958).

Bailey, in a communica-

tion to Burns, Farinacci, and Murnane (1956), reported negative results from pooled brains and salivary gland tissues from 194 Indiana myotis bats (Myotis sodalis), three big brown bats, and two little brown myotis bats collected from Carter Caves in Kentucky.

The Iowa State Department of Health reported that, of several hundred bats examined for rabies in recent years, all have proved negative (Hendricks, 1958).

The increased number of reports of bat rabies in the recent years brings up the question of the origin of the infection. There is a possibility that it has existed for many years in insectivorous bats of the United States. This is the view held by Dr. C. C. Dauer in an article entitled "Mad bats offer clues about spread of rabies" (1957) in the Science News Letter. An increased knowledge and interest with a greater alertness to abnormal bats and bat bites, together with the extensive research work now being done on bats, may have brought about the increase in reported cases of bat rabies. The unprovoked attacks on humans have made us more aware of the problem, at least.

CHARACTERISTICS OF BAT RABIES VIRUS

Rabies in the vampire bat may be manifested in six forms, according to Pawan (1936b). They are as follows: (1) a typical furious form in

which the fury is followed by paralysis and death; (2) a typical paralytic form with no fury exhibited; (3) a furious form with recovery; (4) a furious form with no paralysis preceding death; (5) sudden death with no previous evidence of illness, and (6) a subclinical or latent form with no apparent departure from normal. Because of the unusual forms exhibited by this virus, Pawan stated that the virus seemed to be of low virulence although of high infectivity. The incubation period in vampire bats was reported to vary from seven to 171 days, although this period may actually represent an example of latency of infection rather than of incubation length. An exceptionally virulent strain reported by Pawan (1938a) killed rabbits within two days after inoculation, even after six months' cold storage of the virus in glycerine saline. The vampire bat from which this strain was isolated had been kept in captivity for over 199 days before being allowed to feed upon the first experimental animal.

The incubation period of the vampire bat rabies virus of 29 human cases in Trinidad averaged slightly less than three and one-half weeks, the time between bite and the onset of symptoms varying from two weeks to 11 months (Pawan, 1936a). Hurst and Pawan (1932) reported human cases of bat rabies with death occurring seven days after the onset of symptoms. The paralytic form of rabies in humans was described by Pawan (1938b). The Trinidad cases were of a paralytic rather than a furious

form, which was considered improbable, if not impossible, up to that time. The paralytic human rabies cases in British Guiana reported by Nehaul (1955) terminated in death nine days after the onset of symptoms. This is comparable to the human paralytic rabies death of a woman in Texas, attributed by Sulkin and Greve (1954) to the bite of a rabid insectivorous bat. Death in this case also occurred nine days after the onset of symptoms.

Experiments by Pawan (1948) showed that the saliva of apparently normal fruit-eating bats may be infective. He also showed that these bats may bite other mammals, the bitten animals developing rabies. The average length of life of an infected fruit-eating bat was reported to be 130 days.

Negri bodies were not detectable in many rabid vampire bats tested by Pawan in Trinidad (1936b) and by Johnson in Mexico (1948), nor were they detected in many of the fruit-eating bats also tested by Pawan in Trinidad (1948).

Dogs are considered more susceptible to rabies than man (Malaga-Alba, 1954), but van Rooyen and Rhodes (1948) stated that paralytic rabies contracted from dogs and other animals should not be confused with paralytic rabies spread by bats in Trinidad and made a definite distinction between the two. Johnson (1948) considered the vampire bat rabies virus isolated by TenBroeck in Mexico to be avirulent for dogs, although one became paralyzed

to the point of prostration and then recovered. The blood from this particular dog exhibited rabies neutralizing antibodies after recovery, but had not exhibited the antibodies prior to inoculation. Other dogs tested in a similar manner showed no signs of illness. The marked lack of canine rabies infection in both Brazil (Carini, 1911) and Trinidad (Pawan, 1936a) supported the evidence for a lack of virulence by the vampire bat rabies virus for dogs. Courter (1954b) quoted Briceno Rossi as having stated that bat rabies virus had been isolated from dogs in Venezuela, but added that this was the only report of such an occurrence. Pawan (1936a) noted that strains of the Trinidad virus proved experimentally infective for dogs elsewhere and concluded that there might be some immunity on the part of the dogs in Trinidad and Brazil able to modify or render the bat rabies virus harmless.

Schroeder (1955) found that the bat rabies virus isolated from cattle in Honduras was immunologically indistinguishable from a New York dog salivary gland street virus strain. This similarity between the two strains allowed the use of a vaccine for immunization of thousands of cattle in Guatemala, Mexico, Honduras, Costa Rica, and Nicaragua, none of which had acquired rabies from any source at the time of the publication of Johnson's work in 1955.

In the United States, no difference was noted in clinical symptoms exhibited by

mice inoculated with virus isolated from dogs and mice inoculated with virus isolated from an insectivorous bat from Alabama (Hosty, 1958). In Texas, Quist and co-workers (1957) inoculated three 12-week-old dogs with insectivorous bat rabies virus. One died with typical rabies symptoms, but no virus was isolated from the salivary glands. Neutralizing antibodies were detected in the serums of the two surviving dogs. Mice inoculated with this virus died after convulsive seizures rather than after a lingering paralysis produced by street rabies virus.

The rabies disease found in insectivorous bats also differs from typical rabies in the frequent lack of abnormal behavior of the infected animal, the different locations of the virus in the infected animal, and the varying degrees of negrigenesis.

Rabid insectivorous bats of two species were shot in normal evening flight in Florida (Scatterday, 1954) shortly after the presence of rabies in insectivorous bats was discovered in that state. The first case of bat rabies in California involved a normal-appearing bat whose brain tissue produced symptoms in mice in five days (Enright, et al., 1955). In Montana, however, the first case of bat rabies involving a normal-appearing bat was reported by Dr. Bell in a C. D. C. newsletter in 1957, although bat rabies was first discovered in that state in 1954. The salivary glands of another rabid bat reported by Dr. Bell did not produce rabies in mice inoculated with that tissue, indi-

cating the absence of the virus in the salivary glands. Burns and co-workers (1956) reported that insectivorous bats may harbor rabies and yet show no signs of illness nor abnormality in habitat. The presence of neutralizing antibodies in bats led these workers (Burns, et al., 1956) to note the possibility that infection in an established host rather than a current invasion of a new one was involved. Thus there would also be the possibility of the virus' being of low virulence but of high infectivity, as Pawan (1936b) suggested in reference to the vampire bat rabies virus in Trinidad.

Successful experimental transmission of six different strains of rabies virus to little brown myotis bats by Reagan and associates (1954) indicated the susceptibility of these bats to rabies virus. All of 72 bats inoculated developed paralysis between three and eight days post-inoculation with the diagnosis of rabies confirmed by mouse inoculations. Little brown myotis bats and big brown bats experimentally infected by Stamm, et al. (1956) proved to be susceptible to the virus, with aggressiveness noted as a symptom. Rabies virus was repeatedly recovered from the saliva of one bat in this study during the two-week period preceding its death. These workers reported that the virus from naturally infected bats had an unusually long incubation period in mice, and an even longer one in bats.

In contrast to the lack of recovery of experimentally infected bats in the preceding two

studies. Enright of California (Burns, et al., 1956) found that insectivorous bats may recover following experimental infection with bat rabies virus. A more recent study (Burns, et al., 1958) utilizing six rabies virus strains from Mexican free-tailed bats reported that there was no recovery of Mexican free-tailed or pallid bats, nor of goats, rabbits, guinea pigs, hamsters, or mice inoculated with these strains. Bell, et al. (1955) reported that seven mice inoculated with rabies virus from a big brown bat developed the same paretic signs as others inoculated at the same time but had not succumbed to the disease at 45 days post-inoculation. Tierkel (1957) reported that two bats, clinically well during a three-week period of observation in captivity, yielded rabies virus from the salivary glands but not from the central nervous system

Rabies virus isolated from the first rabid insectivorous bat found in California was inoculated into Mexican free-tailed bats and pallid bats (Enright, et al., 1955). The Mexican free-tailed bats developed the furious form of rabies with a 15-day incubation period, while the pallid bats exhibited the paralytic form with an incubation period of 16 days. None of the bats inoculated intramuscularly in this study developed symptoms of rabies. In a Texas study (Sulkin, et al., 1957), 32 of 137 bats inoculated by the intramuscular route showed infection and few developed symptoms.

Experimentally infected Mexican free-tailed bats were examined by Sulkin and his coworkers (1957) for presence of the rabies virus in the brown hibernating fat. The virus was detected in the brown fat from 11 of the 136 bats finally tested. In four of these cases it was found only in the brown fat and not in brain or salivary gland tissue. The virus was demonstrated in the brown fat 75 days after intramuscular inoculation, in bats showing no overt signs of the disease.

According to Tierkel (1957), the ultimate test of asymptomatic carriers, that of demonstrating continuous yield of virus from saliva of normal bats observed for long periods of time, has not yet been done.

The degree of negrigenesis varies greatly in rabid insectivorous bats. Of rabid bats examined in Florida by Scatterday and Galton (1954), one possessed atypical inclusions and another had no detectable inclusions in either the bat or in two mouse passages. Five bats collected in a Florida study by Venters, et al. (1954) possessed atypical inclusions which could not be differentiated from Negri bodies by microscopic examination alone, but rabies virus could not be isolated from these bats. A high percentage of bat brain tissues from which rabies virus was isolated were negative for Negri bodies in an extensive southwestern United States study by Burns and his associates in 1956. Negri bodies are not always demonstrably present in experimentally in-

fected bats. as reported by Stamm, et al. (1956).

As negrigenesis of fixed virus is generally lowered or absent. Burns, et al. (1956) suggested the possibility that unbroken passage through several generations of bats may have produced a modified virus with many but not all the physical properties of a fixed virus. However, as the majority of the bats tested by Burns and associates were colonial species, the findings of Scatterday (1958a) in Florida may be significant. While eight of 15 rabid free-living bats had demonstrable Negri bodies, none of eight rabid colonial bats possessed detectable inclusions. Brain material from these eight colonial bats produced rabies in inoculated mice.

TRANSMISSION OF THE VIRUS

As late as 1954 it was thought that the problem of rabies transmission by bats was limited to South and Central America, since the sanguivorous bats are not found elsewhere (Carneiro, 1954). The human rabies deaths with history of bat bite which have occurred in the United States, although few in number, indicate that the problem is present in the United States. The possibility that bats may constitute a reservoir for wildlife rabies has been noted by many workers.

The presence of the virus in the brain tissue of a bat does not necessarily mean that the bat can transmit the disease, but the saliva and salivary

gland tissue in a large number of cases has been found to be infective. The presence of rabies virus in the salivary glands of experimentally inoculated little brown myotis bats 168 hours post-inoculation and after was demonstrated by Breuckner, et al. (1954). The results of this experiment might, of course, have been different had a bat rabies virus strain been used.

The three major questions concerning bat transmission of rabies in the United States involve: (1) the source of the virus for insectivorous bats, (2) transmission from bat to bat, and (3) transmission from bat to other animals.

As discussed previously in this paper, the original source of rabies in bats in Florida has not been determined. The absence of connections between the native bats and bats from more southerly areas, the non-migratory habits of the native species, and the limited possibility of accidental importation of infected bats eliminates the majority of logical sources.

Malaga-Alba (1954) reported finding vampire bats only 100 miles from the United States border and also reported (1953) finding vampire bat rabies in two Mexican states bordering Texas, one of which also borders New Mexico and a third state which borders Arizona. Insectivorous bat rabies has been reported from these three states in the United States. Sanborn (1954) noted the long migratory flights of Mexican free-tailed bats,

citing a specimen banded at Carlsbad Caverns, New Mexico, and recovered 800 miles away in Mexico. The possibility of Mexican free-tailed bats acquiring rabies from vampire bats was discounted by Malaga-Alba (1957), however. As Mexican free-tailed bats were never seen roosting in caves occupied by vampire bats, Malaga-Alba concluded that rabies was probably enzootic in Mexican free-tailed bats and not the result of casual contact with vampire bats.

The rabid bats of species Chilonycteris personata reported from the State of Veracruz, Mexico (Grimes, et al., 1955), were taken from a cave inhabited by vampire bats.

There is, of course, the possibility of the virus' having been present, undetected, in bats in the United States for a long time, as discussed earlier in this paper.

The transmission of the rabies virus from bat to bat is indicated by the high level of infection in insectivorous bats in the United States (Irons, 1958). That exposure takes place within a colony is indicated by the progressive development of neutralizing antibodies in a colony reported by Tierkel (1957). As greatly reduced populations of cave bats have been noted by speleologists during winter surveys in the early 1950's, Mohr (1954) suggested that the reduction might have been due, to some extent at least, to rabies.

The possibility of inter-

species transmission of bat to bat may be indicated in the case of the discovery of neutralizing antibodies in Texas cave myotis bats (Burns, et al., 1956) on the basis of their sharing their habitat with Mexican free-tailed bats (Eads, et al., 1956). A large percentage of Mexican free-tailed bats were found to be rabid or to possess neutralizing antibodies in the same study by Burns and his asscciates.

The long distances travelled by insectivorous bats gives them ample opportunity to contract or spread the virus to other bats. A big brown bat was recorded by Mumford (1958) as having travelled at least 142 miles in normal movement. Two of 77 little brown myotis bats released 228 miles from the area of capture were found back at that area within two years (Smith and Hale, 1953). The hoary bat is reported by Dalquest (1948) as migrating from the State of Washington to winter in southern or central California. All three species mentioned here have reportedly been found to be rabid in four or more states, the hoary bat being found rabid in eight states to date.

The possibility of transmission between bats, particularly of different species, because of carnivorous activities should not be overlooked. Engler (1943) reported that a big brown bat killed and ate part of a long-eared myotis bat when both were placed in the same sack following capture. One of two Mexican free-tailed bats kept in a cage with sever-

al pallid bats was partially eaten, as reported by the same author. That carnivorous activity between bats not held in captivity does take place is demonstrated by the report of Bishop (1947) of a hoary bat in New York being observed with the mutilated body of an eastern pipistrelle which it refused to abandon. A report by Orr (1950) concerned a hoary bat shot while pursuing a small pipistrelle-sized bat in California, indicating that such activity is not rare.

The possibility of rabies spread in breeding season fights of vampire bats was suggested by Johnson (1948). Malaga-Alba (1957) noted that Mexican free-tailed bats often fight among themselves and other species, especially at roosting time and during the mating season. In normal jostling at roosting time, the propensity of bats to react to disturbance with open mouth and biting would indicate the ease with which an infective bite might occur. As early as 1796, according to Ackert (1914), Cuvier called attention to the abundance of nerves in the flying membranes of bats. Ackert noted that both Schobl and Sabussow had found the nerves to be present in five layers in these membranes. This abundance of nerves in such a thin membrane at a site likely to be nipped in normal jostling would seem to provide an easy route for the entrance of the virus into the nervous system.

As rabid insectivorous bats have been reported to

make unprovoked attacks on humans, it is logical to assume that such attacks may also be made on bats of the same and different species, providing the possibility of transmission thereby.

Little brown myotis bats were determined to be susceptible to conjunctival and intranasal exposure to both dog and fox street virus rabies strains by experimental means (Reagan, et al., 1956). This indicates other possible routes of infection for the rabies virus to take in contact between bats.

Mohler (1911) reported that rabies virus may be present in milk of lactating rabid animals. Mugrage (1930) found no evidence to support this, but suggested the possibility of the mother's licking the mouths of her young or licking her breasts and covering them with infective saliva. Two suckling bats from separate litters with rabid mother insectivorous bats in Florida proved positive for rabies, with no evident source for the infection other than feeding (Schneider, et al., 1957).

Transmission of the rabies virus by ectoparasites would first require the presence of the virus in the blood stream. This was demonstrated in a study of experimentally infected little brown myotis bats (Reagan, et al., 1955). The virus appeared 24 hours following inoculation and remained active up to 48 hours in the blood. The virus could not be demonstrated in the blood from 48 hours on to 192 and 216 hours, when symptoms

appeared in the bats. In a later experiment, ticks of species Ornithodoros turicata were allowed to feed on infected little brown myotis bats (Reagan, et al., 1956a). After a seven-day incubation period, the ticks were macerated and rectally instilled into hamsters most of which died from rabies. Bell, et al. (1957) failed to demonstrate viremia in rabbits, and virus was not detected in ticks of two species which fed on the infected rabbits from the first day of inoculation until death. No evidence of multiplication of the virus could be obtained in ticks either directly inoculated or fed on infectious materials. Attempts at mechanical transmission by allowing ticks partially engorged upon virus suspensions to complete engorgement upon experimental animals also failed.

Transmission of the rabies virus from insectivorous bats to other animals may be accomplished by bite by infected bats or by ingestion of infected bats by predators. Although there are many records of unprovoked attacks by rabid insectivorous bats, there is no way to determine whether these bats were infective or not. Incidental bite was the method whereby two of the reported human bat rabies deaths in the United States acquired the infection. Bat-pet associations were reported to be numerous in Texas (Irons, et al., 1957) and thus be of possible public health importance. While 13 of 26 suckling hamsters bitten by little brown myotis bats experimentally infected with a dog street virus strain develop-

ed rabies (Reagan, et al., 1957), experimental bat rabies virus transmission by bite has been less successful. Monkeys confined with naturally and experimentally infected Mexican free-tailed bats failed to develop rabies or neutralizing antibodies, although the monkeys were bitten and ingested infected bats (Burns, et al., 1958). Guinea pigs and mice bitten by experimentally infected pallid bats also failed to develop the disease, but the bites of the pallid bats were not deep enough to cause hemorrhage in the guinea pigs. The monkeys, guinea pigs mice, and other animals all died from rabies when subsequently challenged with bat rabies virus.

Bell (1959) reported the transmission of rabies to suckling mice by the bite of a naturally infected California myotis bat which had previously attacked a man in western Montana. Mice of 21 days of age inoculated intramuscularly with salivary gland material showed no sign of illness, and later intracerebral inoculation of 21 day old mice with salivary gland material produced no sign of illness. Suckling mice in both tests showed a high proportion of illness and death. Thus, although transmission by bite has been shown for suckling mice, transmission to adult animals other than humans by the bite of a bat infected with a bat strain of the virus has yet to be accomplished.

Hull (1955) stated that transmission of the rabies virus through ingestion of food contaminated with it is exceed

ingly rare. Hull noted that Wyrsykowski had showed the virus to be destroyed quickly by gastric juices. Mohler (1911), however, stated that abrasions of the lips, mouth, and pharynx are too frequent to eliminate the possibility of transmission by feeding on infected materials.

Thus, either through incidental bite or through ingestion with upper alimentary canal injury present, predators may acquire the rabies virus. High populations of raccoons, skunks, and foxes have been noted in the vicinity of bat caves in Texas (Irons, et al., 1957), and raccoons were found to feed heavily on bats when other food is scarce (Eads, et al., 1955).

Reports of opossum and skunk in North Carolina (Sperry, 1933) and mink in Kentucky (Goodpaster and Hoffmeister, 1950) feeding on bats support the statement that predation on bats by other animals is fairly widespread, even if seldom observed. Constantine (1958) reported that occasional exposure to predation of Seminole bats in Georgia occurred, especially in the cold months when the bats would be torpid or lethargic. As the most readily captured bats, however, those ill from rabies would be likely to be eaten with greater frequency than unaffected bats.

SUMMARY

Bat-transmitted rabies was first discovered in southern Brazil in 1908, but the vampire bat was not proven to

be the vector until about 1918. The disease was soon reported in other South American countries, appearing in Trinidad in 1925. The first human death from the disease occurred in Trinidad in 1929, but it was not until 1931 that the human deaths were traced to vampire bat bite. Although the vampire bat (Desmodus rotundus murinus) was implicated as the vector, fruit-eating bats (Artibeus planirostris trinitatis) were found to harbor the virus. Human rabies deaths attributed to vampire bat bite have since occurred in British Guiana and Mexico. Experimental work with the vampire bat in Trinidad showed that several forms of the disease may be exhibited, including an asymptomatic form and a form with complete recovery.

The first case of bat-transmitted rabies in the United States occurred in 1953. This was the first indication that insectivorous bats might be involved in transmission of the rabies virus. Although the first report came from Florida, reports from other states followed in rapid succession. A human rabies death which occurred in Texas in 1951 was reported to have been the first human rabies death in the United States attributed to the bite of an insectivorous bat. Because of the frequent reports of human attack by rabid bats, studies of normal-appearing bats, especially in areas where large colonies were present, were undertaken. Along with the isolation of rabies virus from both sick and apparently normal bats, neutralizing antibo-

dies for the rabies virus were detected in serum pools from large numbers of bats. A bat rabies worker in Texas died from rabies in 1956, but there was no history of animal bite. The first clearly established case of human rabies death from the bite of an insectivorous bat occurred in California in 1958.

A total of 17 species of insectivorous bats have been reported to be found naturally infected with rabies in 19 states. The disease in insectivorous bats has been found as far north as British Columbia and has been reported from Germany and Yugoslavia.

The virus in insectivorous bats may behave in an atypical manner. It has been found in the central nervous system but not in the salivary glands and vice versa. The virus has even been found in the hibernating fat when not detectable in either the central nervous system or the salivary glands. The degree of negrigenesis varies widely, and Negri bodies are frequently undetected in bats proven positive for rabies by animal inoculation. Atypical inclusions have been found in both rabid and non-rabid bats.

The original source of the virus in insectivorous bats in the United States has not been determined. Because of the chronological order of reports of bat rabies from southern Brazil north to the United States, transmission from vampire bats to migratory insectivorous bats seems to be the most logical source.

With the possible exception of the human cases, transmission of bat rabies virus strains by insectivorous bats of the United States has not been observed to occur. The high level of incidence in bats and the presence of neutralizing antibodies within bat populations indicate transmission from bat to bat, but this has not yet been proven. There is a possibility that the virus in bats may be of high infectivity but of low virulence, at least for the bats themselves.

Predation on bats by other animals has been observed, which indicates that bats are possible reservoirs for the virus in other wildlife if transmission does occur between bat and predator.

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