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EPIDEMIOLOGY OF MONGOOSE RABIES IN GRENADA

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Abstract: Rabies was found in 0.5 - 3.7% of mongooses (*Herpestes auropunctatus*) trapped in Grenada between 1968 and 1972. The difference in the proportions of rabid mongooses during this period was significant and suggested a fluctuation in the incidence of the disease. Serum neutralizing antibodies were found in 18.9% of animals examined, indicating a high transmission rate between mongooses. In addition the behaviour of rabid mongooses is described, and the virus titers in organs from some of these animals are recorded. Human, domestic animal, and livestock involvement in the basic mongoose rabies cycle is discussed.

INTRODUCTION

The introduction of the mongoose (*Herpestes auropunctatus*) into Grenada and the history of rabies on the island up to the end of 1971 have been reported by Everard, Murray, and Gilbert.[5] The current rabies epizootic apparently commenced in the mid-nineteen-forties, and thus would be synchronous with major outbreaks in some parts of the U.S.A., Canada, the U.S.S.R., Europe and other parts of the world.[6] It appears, nevertheless, that there may have been unrecognized cases earlier.[11] There is every indication that the present epizootic in Grenada is maintained in the mongoose, and that other wildlife is only rarely involved.

The small size of Grenada, its rural nature, and the ubiquity of the mongoose have provided an environment in which there is ready contact between man and domestic animals on the one hand and mongoose on the other. The tethering of cattle, sheep, and goats, the existence of a large stray dog population, and the freedom allowed to even the most domesticated dogs and cats, have afforded increased opportunities for contact. Fortunately, there is little evidence of a very high transmission rate to dogs, cats and livestock, and most of the subsequent human treatments are administered not as a result of domestic animal bites but after people have handled rabid animals.

This paper presents further data on rabies in mongooses on this island and some comments on the problem.

METHODS

As part of the Grenada Government rabies control program, mongooses were caught in wooden box-traps covered with wire mesh and baited with chicken heads. The date and locality of capture were recorded, and the animals were killed and bled, when moribund, by intracardiac puncture. Mongooses which attacked humans, domestic animals, or livestock without provocation were, if possible, killed during the incident and the head or carcass taken to the laboratory. Domestic animals and livestock which died under suspicious circumstances were also examined. In all cases, the brain was removed and examined in Grenada by the Fluorescent Antibody (FA) test.[8] In addition, organs and brain material from a few rabid mongooses killed while attacking were screened for virus at the Center for Disease Control (CDC), Lawrenceville, Georgia, by inoculating a 10% tis-

sue suspension into mice.[10] Sera from some mongooses were sent from Grenada to CDC and tested there for the presence of serum neutralizing (SN) antibodies.[1]

OBSERVATIONS AND RESULTS

Normally mongooses are diurnal, do not enter human habitations, move quickly across open spaces, and react rapidly to pedestrians and moving vehicles. Rabid mongooses were found to attack without provocation, to enter human dwellings readily, and often to move at night. Our observations indicated that more than half (5/8) of the mongooses run down by vehicles were rabid. In humans, the back of the legs and ankles, and the hands, were commonly bitten, and there are two records of individuals being bitten on the penis. Bites on livestock were usually on the lower legs and snout. Elsewhere, there have been reports of rabid mongooses attacking humans and then clinging on.[7] This was found to be a relatively common occurrence in Grenada and was one of the reasons why rabid mongooses were frequently killed, especially when they were clinging to the snout of a tethered animal. It appears as if the morbidity period in mongooses may be prolonged: we captured one attacking mongoose and kept it for 2½ days before it died of rabies, and during this time, it did not eat or drink, avoided bright light, attempted to maintain a sleeping posture, and showed no signs of 'furious rabies', although it bit onto a stick and held on while being transferred to a holding cage. We frequently found fluorescence in the brain of mongooses which apparently showed no abnormal trap behaviour, although this was sometimes difficult to interpret in a small trap in strange surroundings. The fact that these mongooses entered traps for food suggests that the disease had not progressed to the point of causing disorientation characteristic of attacking animals; nevertheless, it is presumed that they would eventually have died of rabies. Three of 127 trapped mongooses which we held captive showed signs of rabies, and in each case reluctance to eat was the first behavioural change observed. One animal was kept for 6-7 days during which time it appeared to be well in the morning, but was comatose and weak by the evening. This pattern of 'dumb' rabies was repeated but became more pronounced with a distinct avoidance of bright light. The second mongoose was held for 25 days before it stopped eating, resumed eating 2 days later for a short period, and then finally stopped again. It also was photophobic, shrieked when approached, and had a hunched paralytic posture when killed 31 days after capture. The third animal was kept for 16 days, but only during the last 3 days exhibited 'furious' rabies, bounding round the cage endlessly and becoming more frenzied when approached. We killed all three animals, and they were found to be FA-positive. Occasionally we received reports on the sly, non-retiring behaviour of mongooses which acted 'coyly' in the presence of humans and other animals, or crept up near them. This is considered to be a sign of 'dumb' rabies and presents a danger to children who may be prompted to touch or catch them.

We have no information on distances travelled by rabid mongooses, but an adult male was first caught at Mt. Hartman on 31 January, 1971, and was recaptured in the same area on 20 October, 1971, 21 January, 1972, and 3 February, 1972. It was killed on 17 March, 1973 near Secret Harbour on the Lance aux Epines peninsula after it attacked a dog (Fig. 1). The mongoose was confirmed rabid, and it was estimated that it travelled approximately 2000 meters by the most direct land route from the site of previous capture over a year earlier.

Cases of mongoose rabies during 1972 are recorded in Table 1 under four categories. These are: I, rabies-positive mongooses trapped under the government program; II, animals killed while attacking and found to be rabies-positive; III, animals which attacked but whose bodies were not recovered (clinically suspect); and IV, animals found run over on the road and rabies-positive. Data for 1968 through 1971 are published elsewhere,[8] but the mean values are given here. There

is no significant difference in the annual numbers of category II*, nor in the annual numbers of category III**, but in category I there is a highly significant difference ($p<0.001$) between the proportions of rabies-positive/examined mongooses which were 26/705, 11/1019, 9/1727, 61/1742 and 28/1404 for each of the 5 years. Between January and mid-June 1972, 24 of 651 (3.7%) mongooses examined in category I were positive, a slight increase over 1971. By mid-August 28 of 924 (3.03%) mongooses examined were positive, and since no trapped animals were found positive in September through December, the final figure for the year was 28 of 1404 (2.0%) rabies-positive.

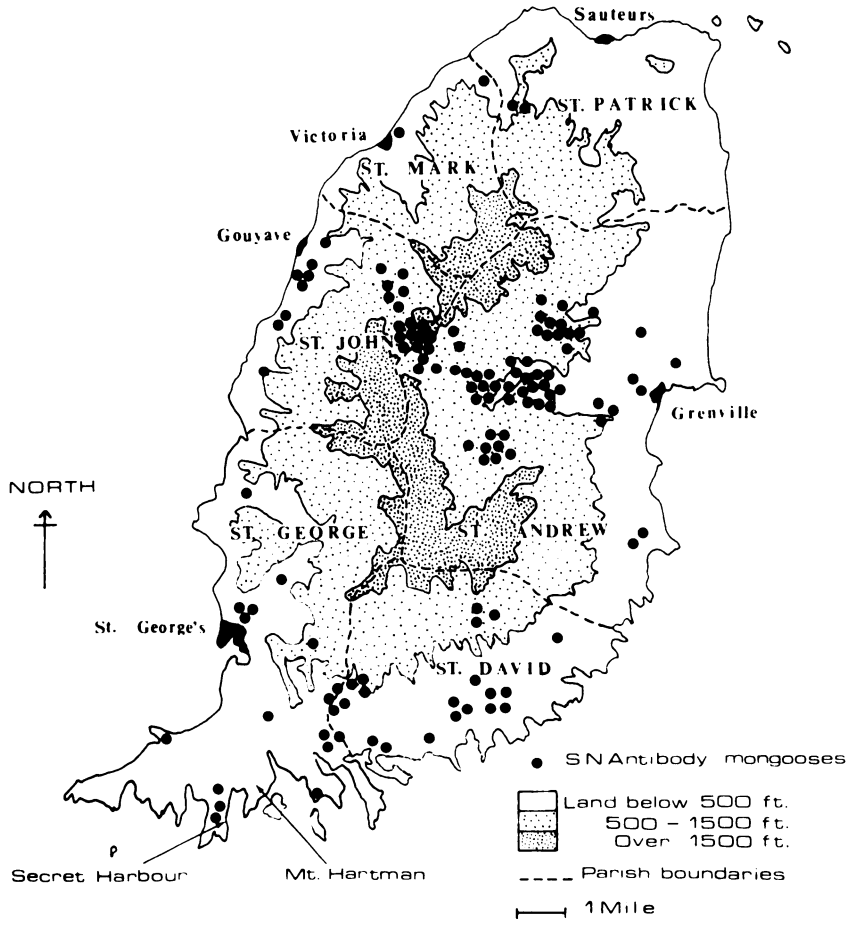

FIGURE 1. Map of Grenada, West Indies, showing the localities of capture for 124 of 127 mongooses with rabies SN antibodies.

* $X^2=7.216$, mean $20.4\pm$SE 6.1 (calculated range, 32.3 to 8.5)

** $X^2=2.181$, mean $13.2\pm$SE 2.7 (calculated range, 18.5 to 7.9)

All the known cases of rabies (clinically suspect and laboratory confirmed) from 1952 to 1972 are recorded by host in Table 2. The percentages are included to give an indication of the frequency of host involvement.

Between 1968 and 1972, 56.1% of human exposures necessitating antirabies treatment were due to mongooses, 23.7% to dogs, 6.5% to cats, and 13.7% to others, with a total of 139 cases. The number of exposures to mongooses annually was 12, 11, 22, 18 and 15 for each of the 5 years, respectively, a total

TABLE 1. Mongoose rabies in Grenada during 1972.

Category	Mean values between 1968 and 1971	1972	5-year total (1968 to 1972)
All trapped mongooses	1298.3	1404	6597
I, trapped rabid mongooses	26.8	28	135
% Category I / all trapped mongooses	2.06	2.0	(2.05%)*
II, attacking mongooses	21.0	18	102
III, clinically suspect mongooses	13.8	11	66
IV, mongooses dead on the road	1.0	1	5
Total rabid mongooses	62.5	58	308

* Five-year mean

TABLE 2. Clinical and laboratory confirmed cases of rabies in Grenada from 1952 to 1972 recorded by host.

Rabid animals	Total No. of cases 1952-1967	Percentage	Total No. of cases 1968-1972	Percentage
Mongoose (*Herpestes*)	143	39.8	308	79.2
Dog	88	24.5	19	4.8
Cat	3	0.8	9	2.3
Bovine	70	19.5	28	7.2
Sheep	9	2.5	7	1.8
Goat	7	1.9	8	2.0
Pig	4	1.1	6	1.5
Horse, mule, or donkey	10	2.8	1	0.3
Unknown	21	5.8	1	0.3
Man	3	0.8	1	0.3
Bat (*Molossus*)*	1	0.3	0	0
Opossum (*Didelphis*)*	0	0	1	0.3
Total Cases	359		389	

* Not confirmed

of 78 cases, while the numbers of human exposures to dogs for the 5 years 1968 to 1972 were 3, 8, 15, 6 and 1.

Table 3 shows the organ titers of seven attacking mongooses confirmed rabid by the FA test and sent to CDC for inoculation of tissue suspensions into mice. One trapped animal, which was positive by FA test and which had not shown abnormal trap behaviour, was also screened. Virus was isolated from the parotid and submaxillary glands, brain, and lungs. All the kidney tissue suspensions were negative, except in one case, where the $MICLD_{50}$ titer was less than 10^1. In some cases the titers may have dropped between collection and titration since these mongooses were held at —20 C for some months prior to shipment to CDC.

Of four samples totalling 672 Grenada mongoose sera tested for SN antibodies during 1971 and 1972, 127 (18.9%) were positive. The samples were collected at different times and in different localities, and trapping was not uniform throughout the island. The proportion of positives was fairly constant at 13/49, 17/99, 26/147, and 71/377. There is no significant difference between these figures. Recorded on the map of Grenada (Fig. 1) are the localities of capture for 124 of these positive mongooses. Titers of the 127 mongooses with antibody were as follows:—

30.7% in the range 1:2 to 1:10

18.1% in the range 1:11 to 1:20

22.0% in the range 1:21 to 1:100

22.0% in the range 1:101 to 1:1000

7.1% in the range 1:1001 to 1:6000

The highest titer recorded was 1:5900. Twelve of 13 sera from trapped rabies-positive animals which showed no behavioural abnormalities had no SN antibody; the positive one had a titer of 1:11.

DISCUSSION

The highly significant difference in the proportion of trapped rabid mongooses suggests a fluctuation of wildlife rabies observable even in the 5 years 1968 to 1972. The results for 1972 alone show that the incidence of the disease may fluctuate widely even within 1 year. The fact that there was no significant difference within either Categories II or III indicates that either contact between

TABLE 3. Organ titers* of rabid mongooses.

Mongoose No. and history	Brain	Parotid salivary gland	Submaxillary salivary gland	Lung	Kidney
1435 Bit 2 dogs and 1 Pig	1	trace (1/5)**	1	—	—
1465 Bit a 2½-year-old-child	+	+	—	trace (1/3)	—
1744 Bit a 13-year old boy on the hand and thigh	1	—	+	1	—
1780 Bit a 20-year-old woman	—	—	—	—	—
2120 Bit a cow	trace (2/5)	trace (2/5)	3.3	1.5	trace (3/5)
2601 Trapped	1	2	3.9	1.7	—
2686 Bit a 2-year-old child	1.5	4.7	2.9	trace (1/4)	—
2705 Bit a pig	1.3	4.3	3.9	trace (1/3)	—

* negative $\log_{10}$ LD_{50}-reciprocal

** one of five mice inoculated with a 10% suspension died

\+ positive with incomplete titration

— negative

rabid mongooses and humans or domestic animals is insufficient to show the fluctuations in the wildlife cycle, or inadequate reporting has not demonstrated the trend in these categories.

Although it appears from Table 2 that the percentage of rabid mongooses of the total number of animals diagnosed has increased from 39.8% (1952 to 1967) to 79.2% (1968 to 1972), it should be noted that there was little laboratory diagnosis of mongoose rabies prior to 1965 and that laboratory examination of all trapped mongooses was not undertaken until 1968.[8]

There was a significant decrease in the number of rabid dogs between 1952 and 1972, 5.5 rabid dogs per year between 1952 and 1967 (88/16) to 3.8 per year between 1968 and 1972 (19/5), and there is therefore every indication that the dog vaccination programs were at least partially effective.

Mongoose salivary gland titers tended to be two to four logarithms higher than those in the brain. Similar results were obtained from naturally infected foxes and skunks in the United States.[4] It should be noted that foxes experimentally infected with less than 10^3 mouse LD_{50} of virus had an incubation period of 38 days or over, with detectable virus in the salivary glands.[12,13] At present we have little information on the incubation period of the disease in mongooses including the three captive animals already mentioned.

Blood from polar foxes in the acute phase of fatal laboratory infection (9-11 days after inoculation) either contained no SN antibodies or they could be noted only at low dilution. Further, 2 of 5 polar foxes which failed to die following experimental inoculation had SN titers of 1/25 and 1/50 6 weeks post-inoculation.[9] Our observations on naturally infected mongooses are comparable as no SN antibodies were detected in 12 of 13 rabies-positive animals, and 18.9% had SN antibodies detectable in the blood as a consequence of natural exposure.

Immunity may presumably be maintained for some time in individual mongooses, and the number of immune animals would increase if the incidence of rabies were to rise. Our figures from sample populations present an index of rabies on an annual basis.[5] From this, we suggest that 0.5 to 3.7% of rabid mongooses in a population are capable of contacting a sufficient number of susceptible mongooses to maintain at least this level of rabies and induce and cause antibodies to be produced in 13.9 to 23.9% (18.9% $\pm$ SE 5.0 for 99.9% limits) of the population.

The development of antibodies in some Mexican freetail bats after laboratory inoculation is supported by field observations where a low prevalence of infection is accompanied by a high incidence of SN antibodies,[2] and it appears that this situation also pertains to Grenadian mongooses. The high prevalence of SN antibodies in colonial bats is attributed to continual exposure to the excretions and bites of rabid bats in a cave environment,[2] but a similar high prevalence in mongooses can only be explained by a high frequency of biting. As immune animals die of other causes and the population turnover is completed, more susceptible animals become available, and the cycle of disease and immunity is repeated.

It is useful in these matters to provide finite numbers, but it should be remembered that they are largely conjectural. Grenada (excluding the Grenadines) has an area of approximately 310 km^2. Available figures (Everard, unpublished data) indicate that 3.2 to 10.4 (mean 5.9) mongooses may be utilizing a hectare of land at any one time. The density calculated from four different population samples is not less than 2.5 or more than 12.6 mongooses per hectare. The island-wide population may therefore approximate 184000 mongooses. Estimating further, 18.9% or 34776 mongooses may have SN antibodies present, and 2.1% or approximately 3860 may be rabid at any period of time, thereby maintaining the epizootic in the wildlife population and the spillover contact with humans, domestic animals, and livestock.

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