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A MYXOMA VIRUS EPIZOOTIC IN A BRUSH RABBIT POPULATION

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Abstract: During the spring and summer months of 1964 more than 95% of a population of brush rabbits *(Sylvilagus bachmani)* became infected with Californian myxoma virus. The characteristics of the epizootic and its effect on this species reinforce the assumption that the brush rabbit is an endemic reservoir of the Californian myxoma virus.

Several lines of evidence suggest that the brush rabbit is an endemic reservoir of the virus which is responsible for outbreaks of myxomatosis in California rabbitries.[1,2,4,6,7] This suggestion gained support when myxoma virus was isolated from a brush rabbit and when antibodies to the viral antigens were found in the sera of two other individuals that were trapped in the vicinity of an outbreak.[4] However, the natural history of the disease, as it exists in populations of brush rabbits, could only be inferred from the results obtained with captive animals that had been infected with the virus.[2]

In May, 1964, one of us (J. H. M.) trapped a brush rabbit which had a myxoma lesion near the base of one of its ears. When four other rabbits from the same population were found to be infected with the virus, it became evident that an epizootic was occurring in this population. This report is concerned with some aspects of this epizootic.

THE DISTRIBUTION OF BRUSH RABBITS WITHIN THE STUDY AREA

The brush rabbits involved in this epizootic were members of several subpopulations which inhabited the Coyote Hills area of Alameda County, California. The area includes a low range of hills located on the northern shore of the San Francisco Bay, together with an adjacent flood plain. The land contiguous with the flood plain has been intensively cultivated for many years and consequently is devoid of brush rabbit habitats. As a result, the brush rabbits of the Coyote Hills were isolated from the nearest populations of the same species by a distance of at least 8 km.

The rabbits were largely confined to the dense vegetation which grew along the sides or tops of a system of levees located on the flood plain or to isolated patches of chaparral which remained on the otherwise grass-covered hills. The areas between the levees were planted to hay and thus acted as barriers to the movement of rabbits from one subpopulation to another except by way of the levees. The interchange of individuals of the chaparral populations with those of the levee populations occurred only when high densities developed locally or when the flood plain was inundated during the winter rains.

SAMPLING PROCEDURES

The rabbits were captured in unbaited wire mesh traps. Each animal was transferred to a cloth bag, welghed, sexed, and inspected for the presence of lesions and ectoparasites. After one ear was tatooed with an identifying number and a sample of blood taken, the rabbit was released in the same area in which it had been trapped. During the period covered by this report 446 rabbits were trapped, many of them on two or more occasions.

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VERIFICATION OF VIRAL INFECTION

During the epizootic the lesions of several rabbits were sampled for the presence of myxoma virus by inducing a mosquito to probe the lesion and then allowing the same mosquito to feed on the shaved skin of a domestic rabbit. In each case the signs that developed in the domestic rabbit were the same as those observed in domestic rabbits infected with strains of the virus that had been isolated in San Mateo or San Diego Counties.[4]

DETECTION OF ANTIBODIES TO THE SOLUBLE ANTIGENS ASSOCIATED WITH MYXOMATOSIS

The sera of the brush rabbits were tested for the presence of antibodies by gel diffusion precipitation tests as described by Mansi.[3] Antigen preparations consisted of saline extracts of frozen and thawed tumors that were removed from domestic rabbits infected with Californian myxoma virus. Undiluted sera of brush rabbits were pipetted into the circumferential wells and the antigen into a central well. The plates were maintained at room temperature. If no precipitation bands were evident by 48 hr the test was regarded as negative.

The sera from brush rabbits with mature, infective lesions usually reacted with the antigen to produce two precipitation lines. At about the time the lesion became converted into a scab, one of the precipitins disappeared, but the second precipitin was present in the sera for from 4 to 6 months after the lesions were formed.

TABLE 1. Monthly Frequencies of Brush Rabbits with Observable Myxoma Lesions or Antibodies (1964-1965)

	Adult rabbits		Immature rabbits		Total rabbits	
Month	With lesions	With antibodies	With lesions	With antibodies	With lesions	With antibodies
June	3/22(.14)	12/22(.55)	20/44(.46)	21/44(.48)	23/66(.35)	33/66(.50)
July	11/41(.27)	35/41(.85)	59/87(.68)	73/87(.84)	70/128(.55)	108/128(.84)
Aug.	2/13(.15)	13/13(1.0)	17/45(.38)	42/45(.93)	19/58(.33)	55/58(.95)
Sept.	0/1	1/1	0/1	1/1	0/2	2/2
Oct.			*		0/12(.00)	10/12(.83)
Nov.			*		0/18(.00)	11/18(.61)
Dec.			*		0/32(.00)	17/32(.53)
Jan.			*		0/54(.00)	8/54(.15)
Feb.			*		0/32(.00)	4/32(.13)
March	0/36(.00)	3/36(.08)	0/ 4(.00)	0/ 4(.00)	0/40(.00)	3/40(.07)
April	0/29(.00)	4/29(.14)	0/14(.00)	0/14(.00)	0/43(.00)	4/43(.09)
May	0/ 8(.00)	0/ 8(.00)	0/11(.00)	0/11(.00)	0/19(.00)	0/19(.00)

*Because of the cessation of the breeding season in July and the annual moult which occurred during September, age group determination was not always possible.

TIME COURSE OF THE EPIZOOTIC

The progress of the epizootic is indicated in the data summarized in Table 1. It is evident that the greatest number of infections took place during the last 2 weeks of June and the first part of July. By the end of August almost all of the members of each of six different subpopulations had been infected. A variety of circumstances prevented extensive sampling during the last 4 months of the year, but none of the rabbits trapped during this interval had discernible lesions. The progressive decrease in the fraction of rabbits with antibodies in their sera is

regarded as resulting from antibody decay rather than the absence of infection. This inference was borne out by the antibody status of rabbits that were recaptured several months after they were known to have had lesions. Six rabbits which had lesions in late June or early July were trapped again on the 23rd or 24th of December. In December there were strong precipitins in two, weak precipitins in three, and none in the sixth rabbit. One of the rabbits that gave a strong antibody response in December was retrapped in April, at which time it lacked perceptible precipitins. Three other rabbits that were known to have been infected during June or July, 1964 also lacked precipitins at the time they were trapped during the March to June interval of 1965.

These apparent antibody decay times were similar to those observed in rabbits that were removed from the population and maintained in captivity from the time they had newly-formed lesions. For example, rabbit #42 was trapped on June 11, 1964, at which time it had a newly-formed lesion. Serum samples taken from this rabbit July through September contained at least one antibody; those taken after October 7 lacked antibodies that could be detected by the double diffusion test. A lesion was present on rabbit #257 at the time it was trapped on August 23, 1964. Its sera gave positive tests with myxoma virus-soluble antigens from September through February 21, 1965, and negative tests thereafter.

Two explanations may account for the presence of antibodies in rabbits that were trapped in 1965, particularly those taken during March and April. Either these individuals represented cases with a persistent antibody response, or they may have been infected late in 1964 and were members of a subpopulation that was not included in the 1964 sampling. Because none of the animals in question had a tatoo at the time of their 1965 capture, there is no way to discriminate between the two possible explanations.

LESION CHARACTERISTICS

The lesions observed in the Coyote Hills rabbits were morphologically similar to those that developed in experimentally infected brush rabbits. The earliest stages seen consisted of slightly thickened areas of skin. Eventually, these areas became sharply delineated from the surrounding skin and were rarely more than a centimeter in diameter (Figure 1). Scab formation took place in about 2 weeks and, after the scab sloughed, a superficial scar was left which remained evident until overgrown with new pelage.

FIGURE 1. Lesion on young brush rabbit (**Sylvilagus bachmani**) naturally infected with Californian myxoma virus.

Of the 94 rabbits that had observable lesions, 92 had lesions located at the base of one or both ears and two had lesions on the lower lip. Because secondary lesions are rarely observed in experimentally infected brush rabbits, the presence of bilateral lesions in 40 rabbits was of particular interest. The frequency of bilateral lesions was not obviously correlated with the age, sex, or collecting site of the infected animals.

INFECTION AMONG WEIGHT CLASSES

During the height of the epizootic, lesions were found on rabbits of all weight categories with the exception of 11 kittens that weighed 150 grams or less. It is possible that maternally transmitted immunity

provided protection against infection or the appearance of symptoms in these kittens, but, if this were so, the passive immunity was of a relatively short duration because almost all of the kittens had lesions by the time they weighed 400 grams or were 12 weeks old.

It is conceivable that either very young rabbits did not survive infections or that they were not exposed to infection until after they had left the nests. Our observations and those of Orr[6] indicate that brush rabbit kittens abandon their nests when they weigh about 75 grams and that for the next few weeks they increase in weight at a rate of about 50 grams per week. The interval between infection and the appearance of lesions is about 1 week for both mature and young brush rabbits.[2] Thus a 150-gram kitten with a lesions could have been infected shortly after it left its nest, but not before.

EFFECT OF INFECTION ON VIABILITY AND FECUNDITY

Infected kittens that were trapped more than once showed weight gains that were comparable to weight gains observed in kittens from myxoma-free populations. Because the infection rate was almost 100%, it was not possible to make a direct comparison of the survival frequency of infected and uninfected rabbits. However, we did not see a marked difference in the density of the adult populations of June 1964 and June 1965.

Among the does that were trapped during the epizootic, seven were in the late stages of pregnancy. One of these had a scabby lesion, and five had precipitins in their sera. Ten of the does that were lactating at the time they were taken included two with healing lesions and six with precipitins in their sera. Thus neither prior or concurrent infection with myxoma virus seemed to have a marked effect on the reproductive potential of female brush rabbits.

DISCUSSION

A striking feature of the Coyote Hills epizootic was the high percentage of infected rabbits in the areas that were sampled. It is apparent that the population was converted from being almost uniformly susceptible to almost completely immune in the interval of a few months. It is not surprising that the virus failed to reappear in the 1965 rabbit kittens if it is assumed that an unbroken chain of infections in brush rabbits is the principal way that the virus is perpetuated in an isolated population. Between August 1964 and February 1965 few, if any, brush rabbits were born, and during the same interval the presence of a large number of immune individuals would serve as a barrier to the transfer of virus from the occasional infective animal to the few susceptible rabbits that might have been present at the same time.

The absence of any obvious change of the virus during the epizootic and the lack of any observable effect of infection on the viability or fecundity of the rabbits is consistent with the notion that the Californian myxoma virus-brush rabbit relationship has achieved the stability of a long-standing association.

Because the epizootic was in progress at the time we began our investigation, we can only speculate as to the time of its origin. Even more obscure is the source of the virus that initiated the epizootic. However, the fact that a second epizootic occurred during 1966 indicates that the source need not have been external to the population. Details of the 1966 epizootic and the possible mechanisms of interepizootic survival of the virus will be presented in a subsequent report.

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