

Myxomatosis in the Mallee region of Victoria, Australia

By ROSAMOND C. H. SHEPHERD, J. W. EDMONDS,
I. F. NOLAN AND A. GOCS

*Keith Turnbull Research Institute, Vermin and Noxious Weeds Destruction
Board, Department of Crown Lands and Survey, Frankston, Victoria, 3199*

(Received 9 January 1978)

SUMMARY

Sharp reductions in the wild rabbit (*Oryctolagus cuniculus* (L.)) population in the Mallee are associated with annual myxomatosis epizootics. The extent to which the population reductions are the direct result of the epizootics varies with time of epizootic occurrence. All grazing animals in the Mallee are under nutritional stress each summer and autumn. When the epizootic occurs during the early summer heavy losses occur in a previously healthy population. Similar losses which occur in the late summer and autumn are the result of a nutritional stress – epizootic complex. The end result in each case is a population reduction of about 80 %.

This reduction occurs in a population which is the most resistant to myxomatosis known in Victoria and in association with epizootics caused by field strains of myxoma virus of moderate virulence only.

The earlier summer epizootics are of considerable economic importance because they sharply reduce the pressure on the limited food available for other grazing animals.

INTRODUCTION

Wild rabbit populations in the Mallee region of north-western Victoria have been subjected to summer epizootics of myxomatosis in most years since 1951. Although these epizootics were not observed in detail before 1970, reports from field staff of the Vermin and Noxious Weeds Destruction Board suggested very strongly that the variation in time of occurrence, between November and April, resulted from variations in the emergence time of mosquito vectors and that the severity of the epizootics was dependent on the numbers of mosquito vectors and the numbers of young rabbits surviving from the preceding breeding season.

By 1966 resistance to myxomatosis had increased to a level at which about 30 % of rabbits survived infection with the Standard Laboratory Strain (S.L.S.) of myxoma (Douglas, 1968). This strain had killed about 99·5 % of rabbits infected when first released in 1951. The strains of myxoma in the field were all moderately virulent and no highly virulent or severely attenuated strains were being recovered (Douglas, 1968).

The year 1967 was one of very severe drought with the lowest rainfall recorded since records were first kept in the Mallee. The rabbit population generally was

very low. A study of resistance levels, field strain virulence and the occurrence of antibodies to myxoma virus was begun at Pine Plains in the Mallee region in 1968. This study was expanded in 1970 to include regular assessment of population numbers and changes in the age structure of the population. The European rabbit flea *Spilopsyllus cuniculi* (Dale) was released in part of the area in 1970 (Shepherd & Edmonds, 1976). This paper reports on the epidemiology of myxomatosis before the spread of the rabbit flea through the experimental area.

MATERIALS AND METHODS

Virulence of field strains of myxoma

Field strains were collected by shooting infected rabbits, cutting an eyelid from each rabbit and storing it in a portable refrigerator. The strains were graded in the laboratory on the response of five laboratory rabbits to challenge with a small dose of passaged material. The grading system used was that described by Fenner & Ratcliffe (1965).

Estimation of rabbit numbers

Rabbits were counted along marked transects over a total distance of 30 km. Counting was done by spotlight from a vehicle moving at about 20 km per hour. At least two counts were made on each visit.

Collection of rabbits

Live rabbits were captured by spotlighting, or, on two occasions, by baiting with the narcotic alpha-chloralose, and returned to the Keith Turnbull Research Institute. When rabbits were not required alive they were shot by rifle, usually by spotlighting at night. Blood samples and eyes were taken from all killed rabbits. The blood samples were refrigerated at 4 °C as sera or on filter paper strips (Sobey, Conolly & Adams, 1966). Eyes were preserved in 10% formalin.

Antibody to myxoma

Sera were tested for the presence of antibody to a soluble antigen of myxoma by the methods of Mansi (1957) or Sobey *et al.* (1966).

Ageing of rabbits

Eye lenses were dried to constant weight and ages estimated by the method of Myers & Gilbert (1968). This method is accurate to about 150 days and is also useful for grouping older rabbits.

RESULTS AND DISCUSSION

Observations of the regular summer epizootics were begun in 1969 and continued during 1970, 1971 and 1972. In each epizootic the field strains were in the middle range of virulence and no highly virulent strains were collected although there is some evidence that field strains in the Mallee region are more virulent than elsewhere in Victoria (Edmonds, Nolan, Shepherd & Gocs, 1975).

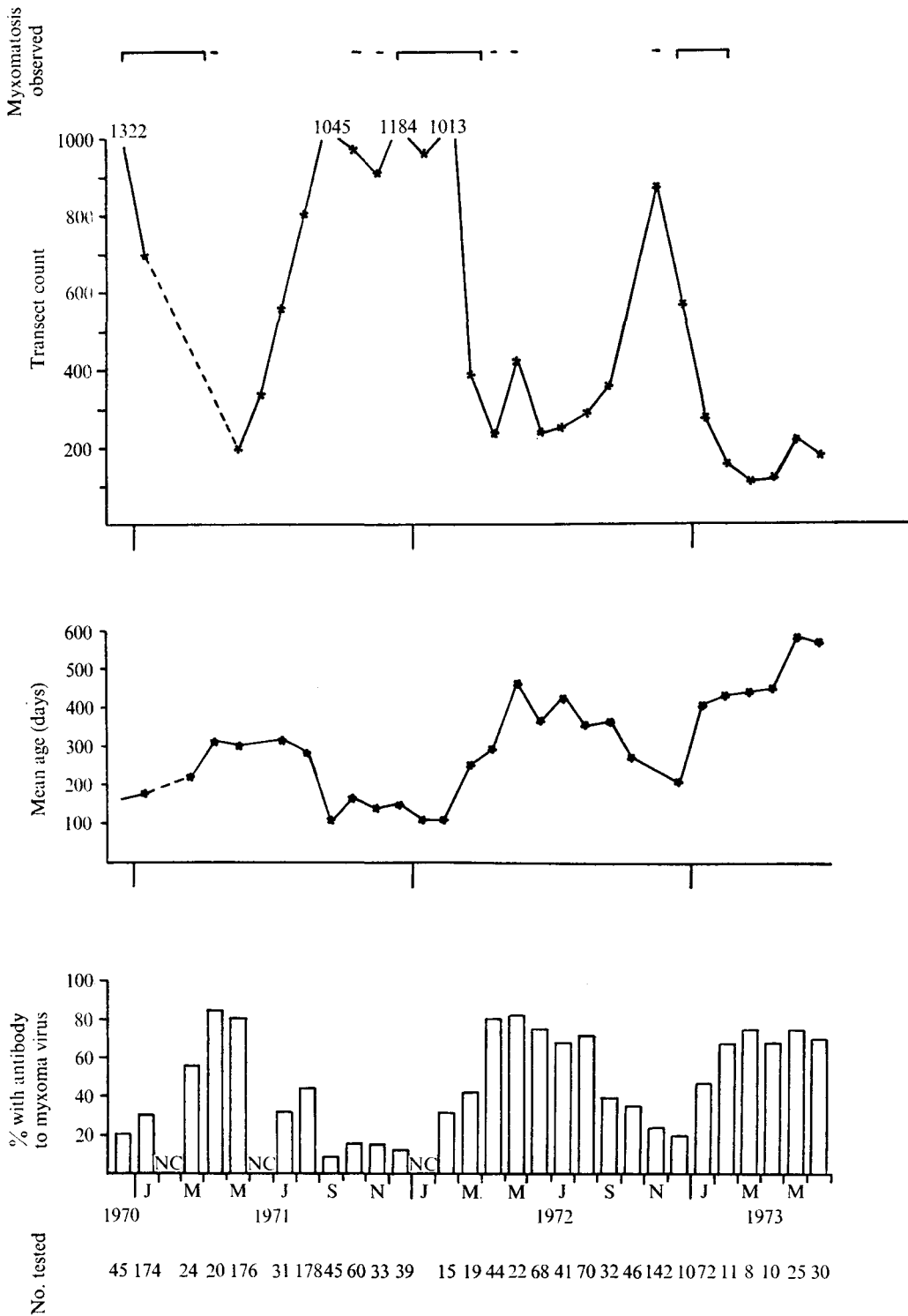


Fig. 1. The percentage of rabbits carrying antibodies to myxoma virus, the mean age of rabbits and the number of rabbits counted over a 30 km transect, and the periods over which myxomatosis was observed, at Pine Plains in the Mallee region of Victoria. Observations were made at monthly intervals from December 1970 to June 1973. NC, no collection made that month.

When regular counts began in December 1970 the rabbit population had recovered from the severe drought of 1967. Peak numbers observed were about 200 per spotlight-km with a mean of 45 per spotlight-km (Fig. 1). The occurrence of a severe myxomatosis epizootic, which was first observed during the third week in December and spread quickly through the area, was accompanied by a sharp reduction in rabbit numbers, to about half of the pre-epizootic count in three weeks and to about one-third in five weeks. The population continued to drop but the continuing decline after about mid-February was probably due to further population reduction under the stress of autumn food shortage, although an occasional case of myxomatosis was seen until April.

The population recovered rapidly during the following breeding season although it did not reach the December 1970 numbers.

The effects of this myxomatosis epizootic and the following breeding season on the percentage of rabbits with antibody to myxoma virus are shown in Fig. 1. The percentage rose from 20% in the pre-epizootic population to 80% in the post-epizootic population and fell again to 10% with prolific winter and spring breeding.

Similar patterns of epizootic, breeding and antibody occurrence recurred in 1972 and 1973 but the timing of epizootics and the timing and productivity of the breeding season varied. Sporadic cases of myxomatosis were seen during the spring of 1971 but the epizootic did not begin until midsummer when the population was already under severe nutritional stress. The count dropped by about 60% between the February and March collections but the percentage of rabbits carrying antibody rose to about 40% only. By April 80% of rabbits collected were carrying antibody and the population had declined to about 20% of the November peak. This pattern was due in part to continuing myxomatosis but heavy mortality among the starving younger rabbits and the better survival of old rabbits, which had been infected in previous epizootics and carried antibody, were mainly responsible for the late increase in the percentage of rabbits carrying detectable antibody.

The population reduction was the response initially to severe nutritional stress alone, then to myxomatosis in a population already under stress, and finally to very severe nutritional stress.

The breeding season of 1972 began in July but was generally ineffective until September when the population numbers began to rise sharply. An epizootic began in November and continued for about eight weeks. Although young rabbits continued to enter the population the peak in numbers was passed in November and the population declined by 75% in two months. The percentage of collected rabbits with antibody again lagged behind the population decline but there is no evidence for any cause other than myxomatosis for the population decline. The lag in the increase in antibody occurrence was probably the result of the initial myxomatosis mortality being most severe in the kittens and sub-adults which are less easily collected but the possibility that other diseases contributed cannot be completely discounted.

The three mosquito-borne epizootics which occurred between December 1970 and December 1972 varied in detail but the end result in each case was similar, an

eventual population reduction of about 80 % from the preceding spring peak with about 80 % of the surviving rabbits carrying detectable antibody to myxoma virus.

Although it is known that part of the experimental area is used as a refuge during severe droughts and that there is some migration of old rabbits into the area each autumn (Edmonds, Shepherd & Lewis, unpublished data) the increase in the mean post-epizootic age in 1972 (Fig. 1) and the maintenance of the increase in 1973 emphasize the severe mortality in young rabbits during the population declines of the summers of 1972 and 1973 and the greater longevity of adult rabbits under less stress in a smaller population.

The reduced stress on the older rabbits during and after an early summer epizootic was accompanied by reduced nutritional stress on other grazing animals including native species and domestic animals. Although the end result each autumn was similar the earlier epizootics were of considerable value to the landholder. It is regrettable that no precise investigations have been made into this aspect of the effects of comparatively minor variations in the epidemiology of myxomatosis.

This work was supported by the Wool Research Trust Fund. We are grateful to Mrs Q. Dam for excellent technical assistance. We are particularly indebted to Dr W. R. Sobey for comment on the draft.

REFERENCES

- DOUGLAS, G. W. (1968). Observations on the virulence of field strains of myxoma virus and on genetic resistance in wild rabbits in Victoria. *Australian Vermin Control Conference* 1968, p. 86.
- EDMONDS, J. W., NOLAN, I. F., SHEPHERD, ROSAMOND C. H. & GOCS, A. (1975). Myxomatosis: the virulence of field strains of myxoma virus in a population of wild rabbits (*Oryctolagus cuniculus* (L.)) with high resistance to myxomatosis. *Journal of Hygiene* **74**, 417.
- FENNER, F. & RATCLIFFE, F. N. (1965). *Myxomatosis*. Cambridge University Press.
- MANSI, W. (1957). The study of some viruses by the plate gel diffusion precipitin test. *Journal of Comparative Pathology* **67**, 297.
- MYERS, K. & GILBERT, N. (1968). Determination of age of wild rabbits in Australia. *Journal of Wildlife Management* **32**, 849.
- SHEPHERD, ROSAMOND C. H. & EDMONDS, J. W. (1976). The establishment and spread of *Spilopsyllus cuniculi* (Dale) and its location on the host, *Oryctolagus cuniculus* (L.), in the Mallee region of Victoria. *Australian Wildlife Research* **3**, 29.
- SOBEY, W. R., CONOLLY, D. & ADAMS, K. M. (1966). Myxomatosis: a simple method of sampling blood and testing for circulating soluble antigens or antibodies to them. *Australian Journal of Science* **28**, 354.