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D R. King

S. H. Wheeler

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Myxomatosis in Western Australia

■ Myxomatosis can still kill vast numbers of rabbits, despite its introduction to the State 30 years ago.

Since myxomatosis was introduced to Western Australia in the early 1950s rabbit plagues have ceased. However, myxomatosis has not proved to be the whole answer to the rabbit problem. Rabbits continue to damage crops and pastures in many areas and resurgences of rabbit populations are reported from time to time.

Drs D. R. King and S. H. Wheeler *of the Agriculture Protection Board's research section have been studying the way myxomatosis epidemics occur and spread in rabbit populations in a range of Western Australian environments. This research will provide data for planning effective control strategies integrating the various control options available to farmers in the State.*

Myxomatosis was successfully introduced into south-eastern Australia in 1950 and spread rapidly with dramatic results. It was estimated that more than 99 per cent of rabbits infected with the disease died in 1950 and 1951. By late 1952, attenuated (weaker) strains of the myxoma virus began to appear and the mortality rate fell to about 90 per cent. In the next few years the mortality rate sometimes dropped to as low as 25 per cent.

However, the situation appears to have stabilised somewhat and under certain conditions, now 30 years after its introduction into Australia, myxomatosis still can kill large numbers of rabbits.

The factors which determine the mortality rate caused by myxomatosis are complex, but studies of rabbit populations at two sites in the South-West of Western Australia are helping us to understand them. This will allow a better understanding of how myxomatosis can be used to help control rabbits, and to make control less expensive.

The virus

The virus which causes myxomatosis was first noticed in South America in 1896, and is transmitted from one rabbit to the next mainly by biting insects such as mosquitoes and fleas.

The virus must be present in the rabbit in high concentration and for a reasonably long time for there to be a good chance of it being spread. Virus production is greatest at temperatures below the normal body temperature of a rabbit. The temperature beneath the skin of the head of a rabbit in the sort of temperatures common on winter nights in the South-West is more suitable for the virus. Because of this, a winter myxomatosis outbreak will be more severe than one in summer, provided there are enough biting insects to spread the disease.

The strain of virus present also influences the severity of an epidemic. Highly virulent strains will kill a large percentage of infected rabbits, whereas very attenuated strains will kill few rabbits. However, virulent strains can kill rabbits too quickly. The virus is then only available for a short time and therefore it is not likely that the infected animal will be bitten by an insect which can spread the disease to healthy rabbits.

A moderately virulent strain of virus spread in winter will produce the best results, killing a large percentage of the rabbits infected and being available for transmission by biting insects for a reasonable time.

■ The European rabbit flea.

Spreading the virus

Myxomatosis was introduced into Western Australia in 1951 and for the next two years it was spread throughout the South-West by inoculation teams which caught rabbits, infected them with the disease and released them. Successful epidemics only occurred in years of exceptional summer rains. The normally dry summers in the South-West hinder the spread of the disease, probably because of the low numbers of mosquitoes present in most areas.

The timing and severity of myxomatosis outbreaks were studied between 1969 and 1982 in rabbit populations at Cape Naturaliste near the south coast, and near Chidlow in the Darling Range to provide more information on the performance of the disease.

Cape Naturaliste

The study area at Cape Naturaliste is cleared pasture surrounded by coastal scrub. There is no reliable information on the timing and frequency of myxomatosis on the area before 1969, but it is believed that epidemics were probably spread by mosquitoes in early summer of some years. There are normally few mosquitoes in the area as it is elevated and windy and there is no free water for mosquito breeding sites.

European rabbit fleas, which are the main means of myxomatosis transmission in Europe, were introduced to the Cape Naturaliste area in May 1969. The aim was to see whether they would survive in the South-West and how fast they would spread.

These fleas are remarkably closely linked to their hosts; female fleas do not produce their own reproductive hormones but must obtain them by feeding on the blood of pregnant rabbits. This ensures that the fleas breed at the same time of year as rabbits and greatly increases the chances of young fleas finding a rabbit to parasitise. It also means that in the South-West, maximum flea numbers occur in winter when a myxomatosis outbreak will cause the greatest mortality amongst rabbits.

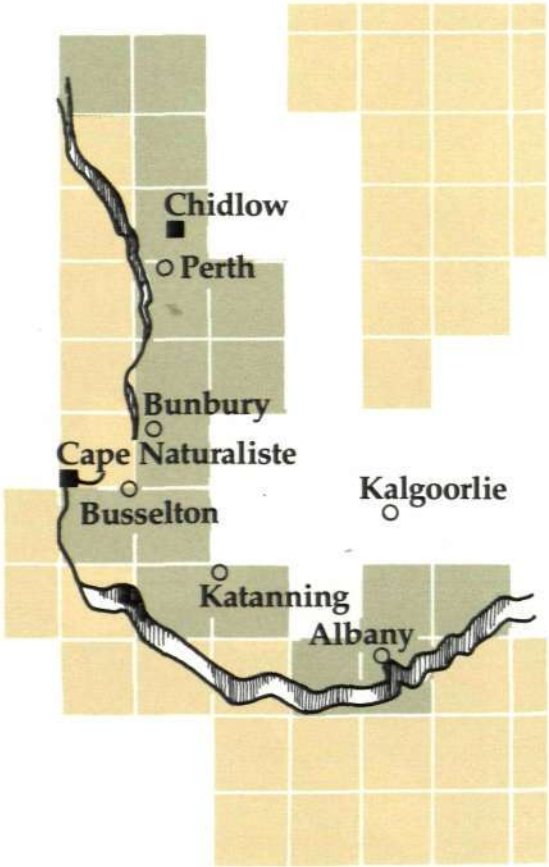
Fleas had spread over the whole area by late 1970. A myxomatosis outbreak had occurred in August 1969 in the area where the fleas were released, and there were additional outbreaks in 1970 and 1971. After these, there were gaps of two to three years between further outbreaks which occurred in the winters of 1973, 1975, 1978 and 1980. Large percentages of rabbits were infected each time and the mortality rate was also high.

Nevertheless, in years when there was no myxomatosis, rabbit numbers sometimes reached very high levels. In some years when myxomatosis occurred in winter, the breeding season continued longer into the next summer. More kittens born late in the season survived in those years than when there was no myxomatosis.

When myxomatosis was transmitted by the European rabbit flea the disease spread slowly out from the point where it was first seen and a high percentage of rabbits was infected. Although up to 85 per cent of the population was killed by these outbreaks, rabbit numbers on the area in the year after each outbreak were unacceptably high. The disease alone, even when spread by fleas, was not effective in controlling rabbits.

Rabbits which survived produced antibodies to the virus. If they contacted the disease again they did not develop the disease. Their kittens also acquired short-term protection against myxomatosis through antibodies they received from their mothers.

An outbreak of myxomatosis was predicted for the area in 1980. Plans were made to introduce a highly virulent strain of virus on to the area, before the start of the field strain outbreak, to see whether a virulent strain might be successful in spreading in competition with the expected outbreak and might provide a better kill. No myxomatosis had been seen on the area since 1978. Few of the rabbits present in 1980 would have survived the 1978 outbreak and therefore would not have antibodies to the virus.



■ The map shows the myxomatosis survey areas at Chidlow and Cape Naturaliste.

■ Traps near rabbit warrens (above) at Cape Naturaliste.

The virulent virus was deliberately introduced to one end of the study area when a field-strain outbreak began at the other end. Although the virulent strain did kill rabbits, it did not spread beyond the area where rabbits were infected. Eventually the outbreak caused by the field-strain covered the whole area.

Chidlow

There were no European rabbit fleas on the Chidlow study area until 1977. Myxomatosis outbreaks occurred in the summer of 1972 and 1975 and in the late summer and winter of 1976. The virus was apparently spread by mosquitoes in all outbreaks. The epidemics spread in a different way from those involving fleas; infected rabbits were distributed in a patchy fashion over the area, with a low likelihood of any single rabbit being infected. The mortality caused by myxomatosis was also lower than had been recorded at Cape Naturaliste.

Fleas were released in the area during the 1977 rabbit breeding season. By late that year they were found on almost all rabbits examined. An outbreak of myxomatosis occurred in June 1978 and reduced rabbit numbers by more than 95 per cent in the next three months. Numbers stayed at a very low level until the middle of 1981, when a moderate increase occurred.

Rabbit breeding continued throughout the summer of 1981-82, which was cooler and wetter than normal. Flea numbers also remained high. An outbreak of myxomatosis began in February and continued until May, and the rabbit population fell by about 75 per cent to a very low level. At least part of the drop in numbers resulted from predators such as wedge-tailed eagles killing diseased rabbits.

The role of the rabbit flea

The introduction of the rabbit flea to both areas seems to have altered the pattern and severity of myxomatosis outbreaks. While this can result in higher mortality in rabbits, flea-borne outbreaks do not necessarily provide adequate control, as is obvious from the study at Cape Naturaliste. They do, however, provide a control opportunity which can be valuable if it is used to advantage.

Additional "follow-up" control before the next breeding season can maximise the benefit obtained by killing more rabbits at a time when it is most important—at the end of summer when their numbers are low. Control is less expensive at that time than when rabbits are numerous. If the population can be pushed to a very low level other mortality factors such as predators can delay its recovery.

Myxomatosis, whether spread by rabbit fleas or not, is not the whole answer to rabbit control in the South-West, but it can provide opportunities to reduce the cost and increase the effectiveness of control programmes.