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The Establishment and Spread of Myxomatosis and Its Effect on Rabbit Populations [and Discussion]

J. Ross, A. M. Tittensor, W. D. Hamilton, R. M. May, R. M. Anderson, J. H. Lawton, P. Holme, R. A. Brown, H. V. Thompson and A. Gibbs

Phil. Trans. R. Soc. Lond. B 1986 **314**, 599-606
doi: 10.1098/rstb.1986.0075

Phil. Trans. R. Soc. Lond. B **314**, 599–606 (1986)
Printed in Great Britain

The establishment and spread of myxomatosis and its effect on rabbit populations

By J. Ross and A. M. Tittensor

Ministry of Agriculture, Fisheries and Food, Agricultural Science Service, Worplesdon Laboratory, Tangley Place, Worplesdon, Guildford, Surrey GU3 3LQ, U.K.

The establishment of myxomatosis, the spread of the disease and its effects on rabbit populations in Australia and in Britain are briefly reviewed. Though the disease is endemic, with regular outbreaks in most rabbit populations, its effect is now much less dramatic than previously. Recent epidemiological studies have shown that the rate of spread of infection, the proportion of rabbits infected and the proportion dying from the disease are very much smaller than recorded in earlier outbreaks. The reasons for these changes are discussed, and the epidemiology of the disease in Britain is compared with that in Australia.

Introduction

Myxomatosis is a disease of European wild and domestic rabbits, *Oryctolagus cuniculus*, caused by infection with myxoma virus. Such viruses occur naturally in certain *Sylvilagus* species in South America and the western U.S.A., in which only mild, though long-lasting, symptoms are produced. Because infection of European wild rabbits caused severe symptoms and was almost invariably fatal, several attempts were made to introduce the disease into areas in which rabbits were serious pests.

The history of attempts to introduce the disease illustrates the essential factors for the successful establishment of an infectious disease. There must be a sufficient density of potential hosts and a means of transmission that will ensure that the disease is transmitted from each infected host to at least one new susceptible host. The failure of some early attempts at introduction in which there was some transmission, which was not maintained, may have been due to reduction in population density (Shanks *et al.* 1955; Notini *et al.* 1952; Basse 1961).

Transmission of myxomatosis can occur by direct contact, by aerosol droplets and by physical means such as thorns, but it is mainly biting insects that are involved. Mosquitoes are the main vectors in Australia; in Britain, the rabbit flea, *Spilopsyllus cuniculi*, is predominant and in France both mosquitoes and rabbit fleas are important vectors. Lockley (1955) reported the lack of success of introduction on Skokholm Island, where no rabbit fleas occurred. In Australia, in the absence of rabbit fleas, several attempts failed to establish myxomatosis in areas that were too dry for mosquitoes (Bull & Mules 1944) or at times of year when mosquitoes were absent or scarce (Myers 1954).

The successful introduction of myxomatosis into Australia came at the end of a series of trials in spring 1950 (August–November) in the Murray valley. After apparently dying out at all sites, the disease flared up again at one site in March 1951. The reappearance of the disease coincided with the emergence of the mosquitoes *Culex annulirostris* and *Anopheles annulipes*; the area of initial spread was largely dependent on the distribution of these mosquitoes (Fenner & Ratcliffe 1965).

The spread of early outbreaks

In France, two infected rabbits were released on an estate near Paris in June 1952, when mosquitoes would have been present. From this single release, myxomatosis spread throughout Europe and reached Britain in 1953. The first outbreak was in Kent in October 1953, but within three weeks two other outbreaks were reported in East Sussex, each about 35 km from the first.

In Australia, myxomatosis spread rapidly from the original trial site, mainly along waterways of the Murray–Darling river system, at an average rate of three miles (five kilometres) per day (Brereton 1953).

In Britain the spread of the disease was recorded in detail. Other outbreaks occurred at considerable distances from the first and could have been due to human interference but it was thought that deliberate movement of infected rabbits did not influence the spread of the disease in the locality of the first outbreak. Attempts were made to eliminate the disease by fencing the areas of the first two outbreaks and killing all rabbits within the fenced areas. This was not successful. The monthly maximum linear spread of the disease varied from 0.9 miles (1.4 km) in October 1953 to 9.1 miles (14.6 km) in October 1954. (Armour & Thompson 1955).

The South American strains of virus, which caused the first outbreaks, killed virtually every rabbit that became infected and had catastrophic effects on the rabbit populations in Australia, France and Britain. The rapid replacement of the original, fully virulent viruses with moderately virulent strains was the first step in the coevolution of virus and host. The course of this coevolution is discussed fully by Fenner (1983) and by May & Anderson (1983) and will not be discussed here again.

Epidemiology

By the 1960s, rabbit numbers in Britain had recovered sufficiently for myxomatosis to be an obvious annual event in many populations. There was evidence of an annual cycle, with the disease present throughout the year, generally at a low level but with increased prevalence in the autumn (Vaughan & Vaughan 1968; Ross 1972). More recently, the pattern of disease has been shown to be more complex than previously thought. Studies of the dynamics of three farmland rabbit populations and of myxomatosis were made from 1971 to 1978 (Ross & Tittensor 1981, 1986). At one of the sites, rabbit density was very low and myxomatosis occurred only sporadically, but at the other two sites, with medium to high rabbit densities, the disease was present most of the time. These two sites, one of 435 ha in Hampshire and one of 188 ha in Sussex, were typical of the current situation. The pattern of the disease at the Hampshire site is illustrated in figure 1, in which are shown the numbers of infected rabbits seen, over the seven-year period, during each month of the year. The disease was present in every month, but with increased prevalence in spring (March–April) and again in autumn/winter (Sept.–Jan.).

Data from the two sites with frequent myxomatosis may be used to calculate the rates of spread and prevalence rates, and the changes in epidemiology may be examined. Data on myxomatosis occurring in February–July were labelled ‘spring’ outbreaks, data from August–January labelled ‘autumn/winter’ outbreaks.

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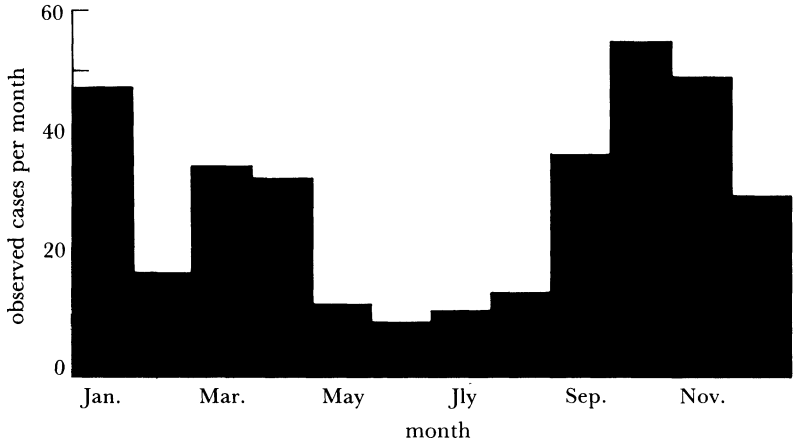

FIGURE 1. Monthly numbers of infected rabbits seen at the Hampshire site, 1971–1978.

Rates of spread

Rates of spread were obtained from maps on which the position of each infected rabbit seen was plotted. The maximum linear spread of the disease for each month of each outbreak was measured, and the mean monthly rates of spread for spring outbreaks and for autumn/winter outbreaks were calculated (table 1). The disease spread much more slowly than during the first outbreak in Britain (Armour & Thompson 1955), but with rates of spread similar to those reported by Chapple & Lewis (1964). The disease spread relatively quickly in 1953/4, because there was a high density of fully susceptible hosts, ensuring that fleas leaving an infected rabbit were likely to find susceptible rabbits. By 1964 rabbit numbers were much reduced, and, although the field strains of virus present were more effectively transmitted than the fully virulent virus, spread of the disease was retarded by the discontinuous distribution of rabbit social groups.

TABLE. 1. RATES OF SPREAD OF MYXOMATOSIS DURING SPRING AND AUTUMN/WINTER OUTBREAKS IN RABBIT POPULATIONS AT TWO FARMLAND SITES IN SOUTHERN ENGLAND, 1971–1978

	mean rates of spread (metres per month)	
	spring outbreak	autumn outbreak
Sussex site	169 ± 15	222 ± 114
Hampshire site	230 ± 157	500 ± 183

In the 1970s, although rabbit numbers were generally higher than in 1964, approximately 30% of the rabbits in the populations studied were immune (having previously recovered). The proportion of immune rabbits varied during the year; a mean of approximately 50% of adult rabbits had detectable antibodies at all times, but the proportions of immune young were low before the spring and autumn outbreaks, increasing during and immediately after the outbreaks (Ross & Tittensor 1986). Although there is evidence that such immune rabbits can be re-infected, it is unlikely that these rabbits act as sources of further infection (J. Ross, A. M. Tittensor & M. F. Sanders, in preparation). There would also almost certainly have been a degree of genetic resistance in the rabbit populations (Ross & Sanders 1984). Infection

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of rabbits from genetically resistant populations resulted in 32% of the rabbits developing only mild symptoms. The presence of immune and resistant rabbits would thus reduce the transmissibility of myxomatosis by 'removing' infective fleas.

The rate of spread of myxomatosis during autumn/winter outbreaks (500 m per month) at the Hampshire site was significantly ($t = 2.96$: $df = 12$: $p < 0.05$) higher than during spring outbreaks (230 m per month). Spring outbreaks were generally confined to areas over which the disease had spread during the preceding autumn/winter, and this may explain the relatively slow progress in spring. At the Sussex site, rates of spread in spring (169 m per month) and autumn (222 m per month) were both similar to the spring rate at the Hampshire site. The relatively slow spread in autumn at the Sussex site is probably because the site is small (188 ha), with most of the rabbit population concentrated on the southern part of the site. Myxomatosis rarely spread to the sparsely populated northern part, probably because of insufficient overlap of rabbit social groups. Figure 2 illustrates the progress of the disease at the Sussex site from

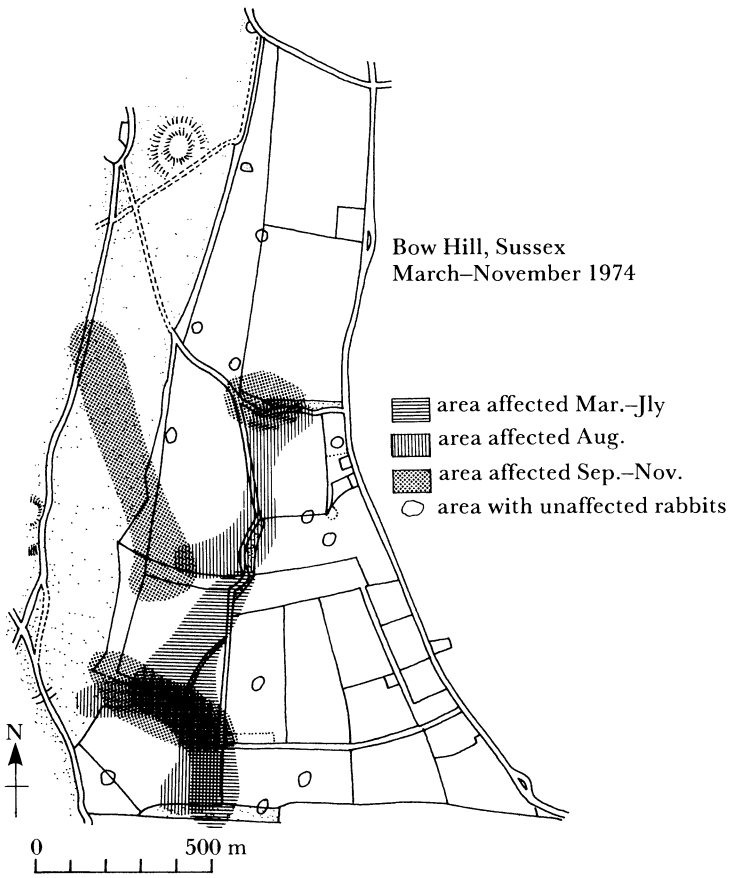

FIGURE 2. Areas affected by myxomatosis at the Sussex site, March–November 1974, and areas inhabited by rabbits but in which myxomatosis was not observed.

March to November 1974. Myxomatosis appeared in March after an absence of nearly two months, and spread over part of the area affected in the previous autumn. There was little further increase in the area affected until August, with considerable expansion in October, before the outbreak finished in November. Though myxomatosis was continuously present for 9 months, it did not spread to all the areas in which rabbits occurred.

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Prevalence rates

The prevalence rates for spring and autumn/winter outbreaks were estimated from the proportion of marked rabbits that were seen with symptoms of myxomatosis (table 2). At each site the autumn prevalence rate was significantly ($p < 0.01$) higher than the spring prevalence rate, reflecting the higher density of non-immune young rabbits present at that time. There

TABLE 2. MEAN PREVALENCE RATES OF SPRING AND AUTUMN/WINTER OUTBREAKS OF MYXOMATOSIS IN RABBIT POPULATIONS AT TWO FARMLAND SITES IN SOUTHERN ENGLAND, 1971–1978

	mean prevalence rate	
	spring outbreak	autumn outbreak
Hampshire site	16.7%	30.5%
Sussex site	15.0%	25.9%

could have been considerable undetected infection in young rabbits in the spring (Shepherd & Edmonds 1978; Parer 1977). It is known that young rabbits die from myxomatosis much more quickly than adult rabbits (Fenner & Marshall 1954) and could thus escape observation. The prevalence rates at the two study sites were very much lower than those noted during the original outbreak or in the 1964 outbreak because, by the 1970s, the proportion of immune rabbits and the degree of resistance had both increased in the rabbit population.

The reported prevalence rates for outbreaks in Australia (Williams *et al.* 1973; Dunsmore & Price 1972) are generally much greater than those shown in table 2. This difference may be due to the distinctive pattern of disease in Australia, where myxomatosis outbreaks are sharply defined with little or no evidence of disease during intervening periods, leading to greater fluctuations in the proportion of immune rabbits, from high proportions immediately after outbreaks falling to very low proportions at other times (Shepherd & Edmonds 1978). The presence of fewer immune rabbits means more susceptible hosts and therefore higher prevalence rates.

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Discussion

W. D. Hamilton, F.R.S. Comparison of the data for Australia and Britain suggests that there has been more progress towards resistance to myxomatosis in Britain. This may be because rabbits have been established longer in Britain and therefore may have a more diverse gene pool there. This in turn may help to explain their more rapid evolution of resistance.

J. Ross. Resistance in Britain is continuing to develop beyond the level found in Australia and may indeed reflect such a difference in the gene pool.

R. M. May, F.R.S. (*Department of Biology, Princeton University, New Jersey, U.S.A.*). Behavioural changes are also alleged in Britain, for example involving rabbits living above ground and hence in a less suitable habitat for a flea vector.

J. Ross. There is no real evidence for a change of habit. Some rabbits have always lived above ground part or all of the time, and there are no grounds for believing that the proportion has increased.

R. M. Anderson (*Department of Pure and Applied Biology, Imperial College, London, U.K.*). Dr Ross has presented a lot of information about case fatality rates, but does he have information about the virus involved from serological typing? And why does he expect a continuing decline in virulence? The virulence of a myxoma-type virus would be expected to be related to its transmissibility.

J. Ross. I agree on the second point. On the first: the strains present in Britain would be expected to kill 90–95% of susceptible rabbits. There appears to have been some increase in the prevalence of the more virulent strains recently, but the commonest is still a 3A strain, which kills 90–95% of infected animals.

J. H. Lawton (*Department of Biology, University of York, U.K.*). Is the genetic basis for resistance the same in Australia and Britain?

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J. Ross. We do not know. This would be an interesting study.

R. M. May, F.R.S. It would be interesting, for the same reason, to know if there has been any behavioural change.

P. Holme (*Imperial College, Silwood Park, Ascot, Berks. U.K.*). How has the changing face of British agriculture, with the removal of hedgerows and other sources of rabbit harbourage, affected the spread rate of myxomatosis?

J. Ross. The rate of spread does not seem to have altered since the second wave of outbreaks in Britain, at least up to the late 1970s. Hedge removal could impose barriers between rabbit populations inhabiting separate small sites, and this may explain the failure of the disease to spread to some of the groups described in our study.

R. A. Brown (*Imperial Chemical Industries PLC, Jealott's Hill Research Station, Bracknell, Berks., U.K.*). Even if rabbits did spend more time above ground than before myxomatosis, would it affect transmission by fleas, which move between animals, both above ground and between the mother and her offspring when she gives birth in a stop burrow?

J. Ross. Quite a lot of flea transmission also occurs outside the nest, and this may indeed be more important for spreading myxomatosis.

H. V. Thompson. This is one aspect where behaviour is clearly linked to disease transmission. A. Mead-Briggs showed that there was indeed quite a lot of flea interchange above ground. Last year there was an Environmental Impact Assessment in New Zealand of the case for introducing myxomatosis for rabbit control there, where there has been a heavy subsidy for control of a few large remnant populations. Does Dr Ross think this would be worth pursuing?

J. Ross. There are no rabbit fleas in New Zealand, so some vector would have to be introduced. The lack of vectors explains the failure of previous efforts to establish myxomatosis there. A similar situation exists in certain island populations where there are no vectors and where early attempts to establish the disease failed.

R. M. May, F.R.S. Even if a vector were introduced, there would not necessarily be transmission. In birds there are quite sensitive dependences between group living patterns and densities and the propagation of diseases.

A. Gibbs (*Research School of Biological Sciences, Australian National University, Box* 475 *P.O., Canberra, A.C.T., Australia*). With the recent release and establishment of rabbit fleas there are two kinds of vector in Australia. What does Dr Ross think the pattern of transmission is now likely to be?

J. Ross. The introduction of rabbit fleas in Australia led to increased prevalence of flea-borne winter and spring outbreaks of myxomatosis, with higher case fatality rates than in summer outbreaks which are mainly mosquito-borne. Winter outbreaks spread more slowly than summer outbreaks.

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R. M. May, F.R.S. It is clear that the disease has had a major influence on rabbit abundance in Britain. What limited rabbit populations before myxomatosis?

J. Ross. Britain had a very high rabbit population, but I am not sure whether it had reached a plateau before the disease was established. The increase after World War II was not, however, at the full potential rate, so some limiting factors must have been in play.

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