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Fifty-year review: European rabbit fleas, *Spilopsyllus cuniculi* (Dale, 1878) (Siphonaptera: Pulicidae), enhanced the efficacy of myxomatosis for controlling Australian rabbits

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ABSTRACT

European rabbit fleas were released among Australian wild rabbits in the late 1960s to supplement mosquitoes as vectors of myxoma virus. Data from study sites across southern Australia in the 1960s and 1970s are reviewed to discern common elements of flea-borne myxomatosis epizootics and a simple model is proposed to explain how virus virulence and food quality interact to determine rabbit abundance. Low, stable populations of rabbits implied that, despite virus attenuation and increased rabbit disease resistance, flea-borne myxomatosis was extremely important in controlling rabbit populations. Despite the enhancement of myxomatosis, livestock producers benefitted little from the additional pasture because marketing difficulties were not conducive to industry growth. Native wildlife likely benefitted, nonetheless.

Keywords: biocontrol, coevolution, conceptual model, conservation, epidemiology, livestock, pasture biomass, vector.

Introduction

Wild rabbits (*Oryctolagus cuniculus*) introduced into Australia in the mid- to late-19th century quickly became invasive pests. In only 70 years, they spread across the southern two-thirds of the continent, heavily damaging newly developing livestock industries and agriculture, severely damaging soils and native flora and eliminating native fauna (Stodart and Parer 1988; Williams *et al.* 1995).

In 1950, myxoma virus was deliberately introduced as a biological control agent (Fenner and Ratcliffe 1965). Although circulating benignly in its natural host, the South American lagomorph *Sylvilagus brasiliensis* (now *S. andinus*; Ruedas *et al.* 2017), the virus caused severe, usually fatal, myxomatosis in Australian wild rabbits. Transmitted by naturally occurring mosquitoes in early summer, it reduced Australia's estimated one to 3 billion rabbits by over 90% (Burnet 1952).

Burnet (1952) anticipated that the virus and its new rabbit host would co-evolve and within only 2 years wild rabbits were shown to be developing genetic resistance (Marshall and Fenner 1958). Attenuated strains of the myxoma virus that killed fewer rabbits also appeared. Grade I and II viruses killed rabbits too quickly for the virus to spread and Grade V viruses were so attenuated that there was insufficient virus in skin lesions for spread by biting vectors. Consequently, Grade III and IV viruses that caused intermediate mortality were optimally transmitted (Fenner and Ratcliffe 1965; Fenner and Fantini 1999).

Nonetheless, scientists were uncertain whether the rabbit and virus had reached a stable *modus vivendi* as Burnet expressed it. Rendel (1971) considered that rabbits were becoming so resistant to myxoma virus infection that it would be necessary to introduce additional highly virulent myxoma viruses or hasten the distribution of European rabbit fleas (*Spilopsyllus cuniculi*) to spread myxomatosis more efficiently. Williams and Parer (1972), studying myxomatosis at Lake Urana in New South Wales, were equally

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pessimistic; many young rabbits recovered from mosquito-borne myxomatosis but subsequently died, apparently from summer food shortage.

In retrospect, the problem in assessing the long-term effectiveness of myxomatosis arose because it was not understood how the disease regulated rabbit abundance. Some sort of virulence-dependent mechanism was assumed, but it was uncertain how it operated. Nonetheless, when European rabbit fleas were experimentally introduced to field sites in 1968 (Sobey and Conolly 1971; Sobey *et al.* 1977), ideas began to change. At most sites where the rabbit fleas became established, a shift in the timing of outbreaks of myxomatosis was reported. Instead of early summer mosquito-borne outbreaks among subadult rabbits, the fleas transmitted the virus among younger rabbit kittens in the winter and early spring (Fullagar 1977; Shepherd and Edmonds 1978*a*, Shepherd and Edmonds 1978b; King and Wheeler 1979; Cooke 1983). Furthermore, those outbreaks caused extremely high mortality (Fullagar 1977; King and Wheeler 1979; Cooke 1983) and rabbit populations that had been increasing fell back to the levels seen after myxomatosis was first introduced. The introduction of rabbit fleas reversed the post-myxomatosis recovery of rabbits in many areas.

Here, epidemiological studies of flea-borne myxomatosis conducted in the 1960s and 1970s are re-evaluated to derive a conceptual model to explain how myxomatosis regulated rabbit abundance at that time. Furthermore, additional ideas about rabbit population dynamics are incorporated to help interpret those earlier records. These come from studies on rabbit behaviour (Mykytowycz 1959), reproductive output (Gilbert *et al.* 1987), nutritional requirements (Cooke 2014) and ecological impacts (Mutze *et al.* 2016; Cooke 2021). Of particular importance is a re-assessment of how the virulence of myxoma viruses has been maintained as the driving force in keeping rabbit abundance low (Di Giallonardo and Holmes 2015; Kerr *et al.* 2017). Finally, livestock industry and conservation benefits resulting from the flea introduction are briefly discussed.

Background to the introduction of European rabbit fleas into Australia

Although rabbits brought into Australia in the 1800s carried some ectoparasites such as the rabbit louse, *Haemadipsus ventricosus*, and fur mites, *Leporacarus* (*Listrophorus*) *gibbus,* and *Cheyletiella parasitivorax*, European rabbit fleas were not included (Williams 1972). Nonetheless, when it was realised that fleas were spreading myxomatosis, newly introduced into Europe in 1952 (Allen 1956; Arthur and Louzis 1988), their use in Australia was immediately considered.

An attempt to import them in 1957 failed because the complexity of the flea's life cycle was not understood (Fenner and Fantini 1999), but on learning that the fleas bred only after feeding on pregnant female rabbits (Mead-Briggs 1964; Rothschild and Ford 1964, 1969), efforts were renewed. Dr Bill Sobey successfully imported fleas in 1966 and established a quarantined breeding colony at the CSIRO Division of Genetics in Ryde, Sydney, where the flea host specificity was assessed (Sobey and Menzies 1969; Sobey *et al.* 1977; Fenner and Fantini 1999).

The initial introduction and spread of rabbit fleas

When granted permission for field release in 1968, Sobey and Conolly (1971) found that the fleas established readily at Longford, Millambri and Wing Vee in New South Wales. Several other research groups across southern Australia then joined the program to evaluate the fleas' ability to spread myxomatosis. The locations of the main field study sites are shown in Fig. 1.

Typically, fleas became widely established within 2 years and showed regular peaks of abundance that lagged slightly behind rabbit breeding (Fig. 2). Myxomatosis spread best during late winter and spring when most rabbits were flea-infested (Shepherd and Edmonds 1978*b*; Cooke 1984).

Widespread releases of fleas in inland South Australia failed except in areas where average annual rainfall was more than 200–250 mm (Cooke 1984); a survey in 1986 confirmed that fleas mainly persisted in areas where annual average rainfall exceeded 250 mm (Fig. 3).

Laboratory experiments to explore the survival of adult fleas and larvae at different temperatures and humidities (Cooke and Skewes 1988) and measurements of rabbit burrow temperature and humidity at different localities (Cooke 1990) showed that burrow microclimates in arid areas were generally unsuitable for flea survival. Although the higher-rainfall zones suited for rabbit fleas covered only 23% of the rabbit distribution, they, nonetheless, amounted to 1.2 million km^2 and included the most productive agricultural areas of southern Australia.

Changes to myxomatosis epidemiology

Sobey and Conolly (1971) showed that rabbit fleas spread myxomatosis in advance of the expected early summer mosquito-borne outbreaks. This was confirmed by Fullagar (1977) who described two winter myxomatosis outbreaks carried by fleas in Canberra, and by Shepherd and Edmonds (1978*a*) in north-western Victoria where the fleas also spread the myxoma virus in winter and spring. In South Australia, Cooke (1983) not only showed that fleas transmitted the virus in late winter and spring, but also that flea-borne myxomatosis caused an estimated 80–90% mortality among young rabbits across four widely separated South Australian experimental sites (Penola, Keith, Mount Pleasant, and Belton). In Western Australia, at Cape Naturaliste, King *et al.* (1985) considered that flea-borne myxomatosis killed 80–96% of young rabbits, well beyond their highest estimate of 57% mortality observed with mosquito-borne

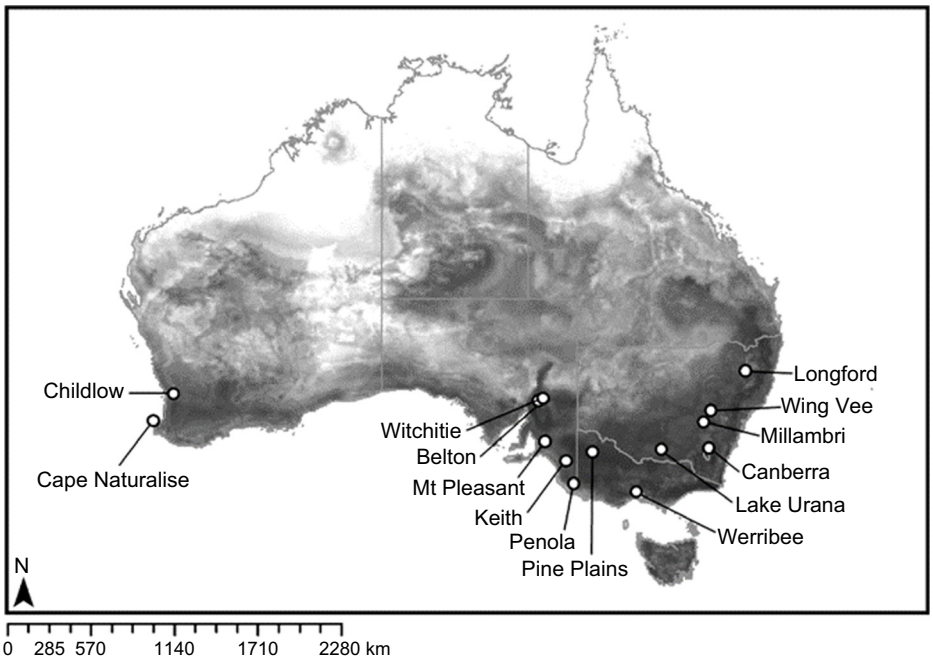

Fig. 1. Locations of experimental sites where European rabbit fleas were studied. The darkest shades on the background map indicate the highest probability of occurrence of rabbits (index of >0.6) as modified from Roy-Dufresne *et al.* (2019). Rabbits are absent from northern monsoonal Australia.

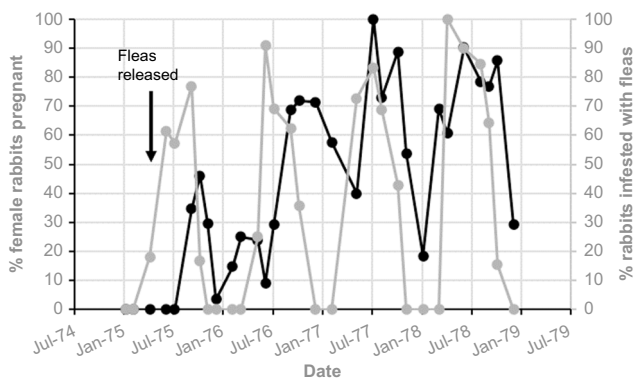

Fig. 2. Establishment of European rabbit fleas at Keith, South Australia. Percentage of female rabbits pregnant (grey symbols and lines); percentage of all rabbits (both female and male) with fleas (black symbols and lines). Source: B. Cooke, unpubl. data, Animal and Plant Control Commission, South Australia).

myxomatosis. Similar results were obtained at Chidlow (King *et al.* 1985).

Contrarily, Williams and Parer (1972) considered that there was no major change in the timing of myxomatosis when fleas were introduced at Lake Urana, New South Wales. Even later, Parer and Korn (1989) examined reports by government rabbit inspectors throughout New South Wales and found that the reported timing of myxomatosis outbreaks was little changed by the introduction of rabbit fleas. Nevertheless, Parer's own experiments at Lake Urana from 1968 onward established that the fleas could transmit the myxoma virus in early spring during the rabbit breeding season before mosquitoes became active (Parer *et al.* 1985).

The most likely explanation for these divergent opinions is that flea-borne myxomatosis killed many young rabbits, which disappeared without trace, their demise probably being attributed to predation, whereas uninfected young rabbits that survived until early summer contracted myxomatosis when they were subadults, survived longer and were readily seen (Cooke 1983, 2019).

Even if Williams and Parer's Urana study site was exceptional, the results obtained by other research groups showed that in most places the rabbit fleas changed the timing of myxomatosis outbreaks and greatly enhanced mortality among young rabbits.

Why flea-borne myxomatosis resulted in higher mortality

Cooke (1983) speculated that rabbit fleas might promote more virulent variants of the myxoma virus or even transmit a higher dose of virus among rabbits. However, Dwyer *et al.* (1990) disagreed. They compared data on the ability of mosquitos to spread myxoma viruses of different virulence from Fenner *et al.* (1956) with similar data on transmission by rabbit fleas (Mead-Briggs and Vaughan 1975) and concluded that fleas were less efficient vectors

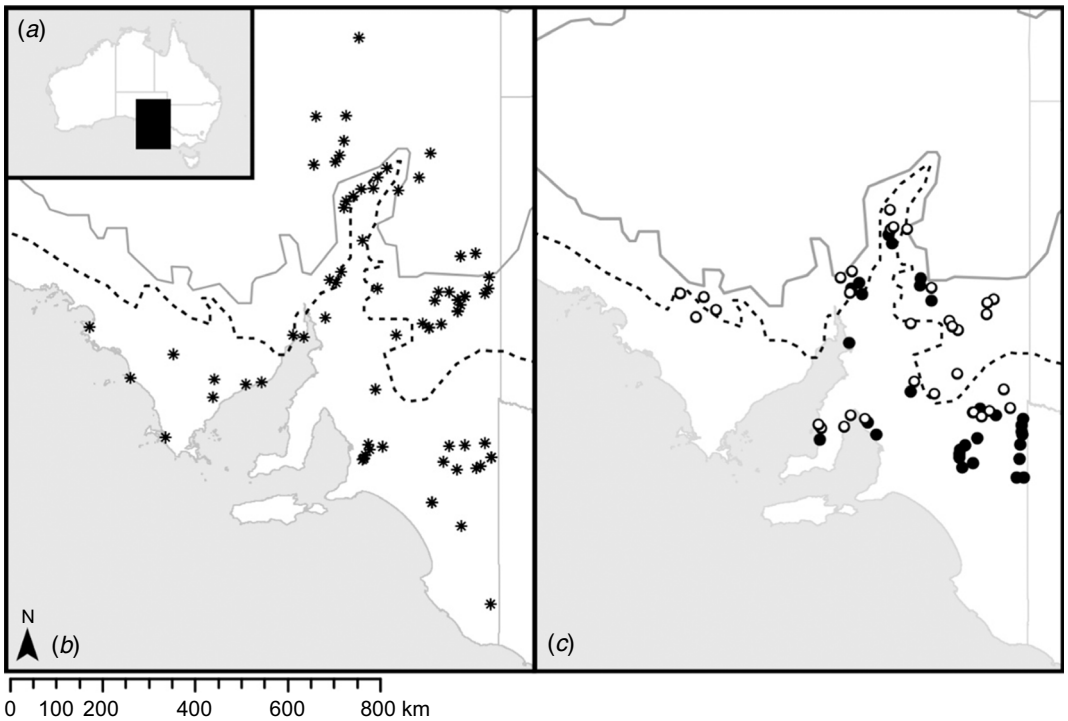

Fig. 3. Summary of the long-term results of releases of European rabbit fleas in South Australia. (*a*) General area within Australia where flea releases were evaluated. (*b*) Sites of releases made by South Australian Animal and Plant Control Commission. (*c*) Results from a limited survey in 1986, showing that rabbit fleas persisted mainly in areas where annual average rainfall exceeded 250 mm: 200 mm (grey line); 250 mm (broken line).

than mosquitos and did not favour the transmission of more virulent viruses. Dose effects in myxoma virus transmission also seemed unlikely. In laboratory experiments, extremely small subcutaneous doses of virus that mimicked mosquito bites were enough to cause severe infection (Fenner and Fantini 1999).

The high mortality of rabbits when rabbit fleas are vectors is therefore most likely because rabbits become infected during the rabbit breeding season when very young (Fenner and Marshall 1954) and low spring temperatures further enhance mortality (Mykytowycz 1956; Marshall 1959). European rabbit fleas are most abundant on rabbits between July and November (Fig. 2) and rabbit burrow temperatures are lowest in mid-August, often about 10–12°C in areas where rabbit fleas occur (Cooke 1990). Frosts are common in early spring too, so ambient temperatures for largely nocturnal young rabbits are below 12°C, and often close to freezing.

Changes in rabbit abundance

Few of the Australian studies of rabbit fleas continued long enough to properly assess long-term changes in rabbit abundance. Nonetheless, rabbit populations generally declined. At Mount Pleasant in South Australia, for example, where fleas became permanently established, samples of 25 rabbits needed to assess flea infestations could initially be obtained in only 2–3 h by shooting over 50 ha. Later, it took two evenings of spotlighting across 3000 ha to collect a sample. The study was eventually terminated for lack of rabbits (Cooke 1983).

At Witchitie and Belton, also in South Australia, better information was available because regular 6-weekly spotlight transect counts of rabbits were maintained for almost 30 years. Furthermore, although the sites are only 32 km apart, and are influenced by the same major weather events, differences in elevation and total rainfall meant that they sat astride the boundary of the permanent distribution of rabbit fleas (Cooke 1983).

At Witchitie, fleas were introduced in 1969 and persisted despite the rabbit population crashing due to overgrazing and water shortage. Nonetheless, the fleas became scarce 8 years later, following drought in 1977, and disappeared completely during drought in 1982 (Cooke 1982, 1983). During the years when the fleas were abundant, the rabbit population remained very low; however, rabbits regained former abundance after the fleas were eliminated (Fig. 4*a*). By contrast, at Belton, where rabbit fleas became permanently established after 1974, the rabbit population fell from an average density of about 2.0 rabbits per hectare to 0.2 rabbits per hectare by 1982, and remained at that low level for the following 12 years (Fig. 4*b*).

In the high-rainfall years of 1968–69, before fleas were introduced, the rabbit populations at both Witchitie and

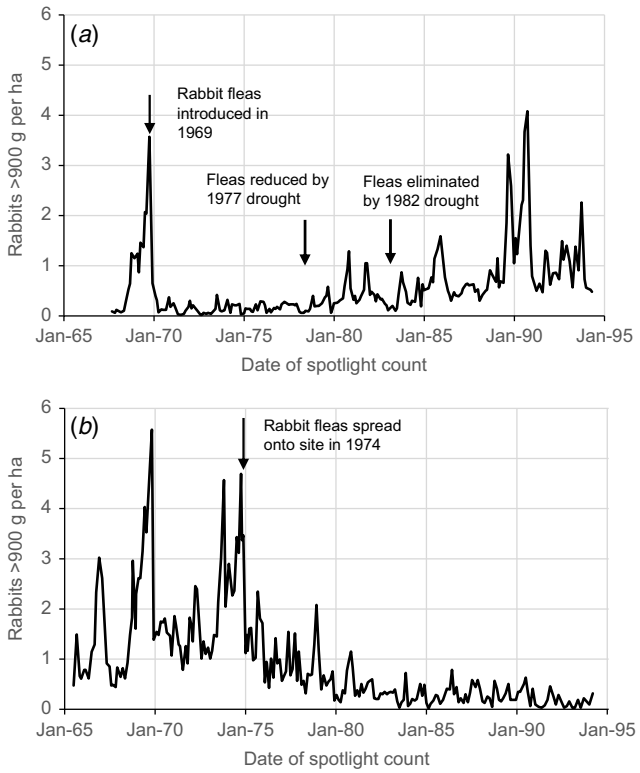

Fig. 4. Spotlight counts of rabbits at (*a*) Witchitie and (*b*) Belton converted to approximate rabbit population density (rabbits per hectare), recognising that only rabbits weighing more than 900 g are usually seen in the spotlight beam.

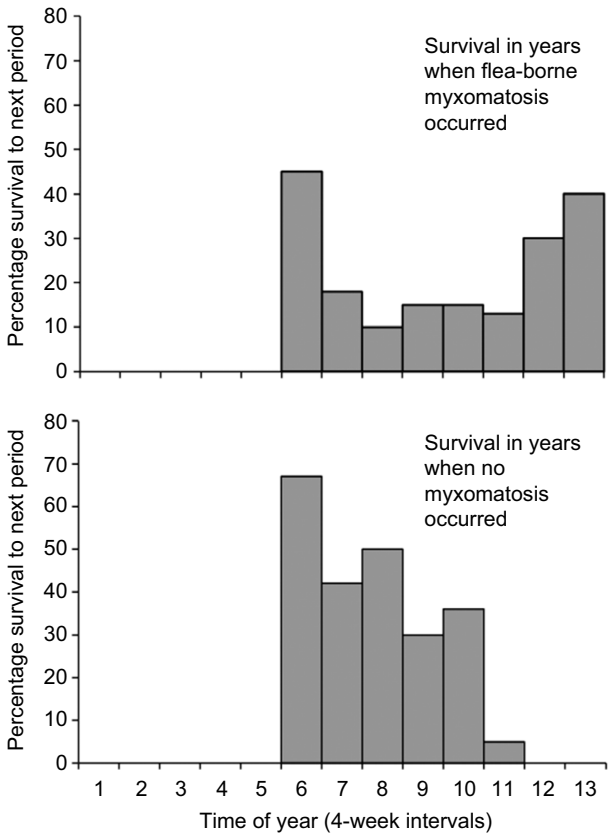

Fig. 5. (*a*) In years when flea-borne myxomatosis spread at Cape Naturaliste (1971, 1973 and 1975), the rabbit breeding season was extended and rabbit kittens born late in the season survived well; (*b*) the survival of late-born kittens was poor in years when there was no flea-borne myxomatosis (1972, 1974 and 1976). Figure has been adapted from data in King and Wheeler (1979).

Belton fluctuated in parallel. However, Fig. 4 shows that in the wet years of 1973–74, the rabbit population at Belton increased but there was no similar build-up at Witchitie where rabbit fleas had been established (Cooke 1983). Subsequently, flea-borne myxomatosis at Belton suppressed the expected rabbit build-up in the high-rainfall period during 1989–91, even though rabbit abundance increased strongly at Witchitie where rabbit fleas had died out.

At Cape Naturaliste in Western Australia, rabbit fleas were introduced when the study commenced in 1969 and there was a steady downward trend in spotlight counts of rabbits during the 11-year study. The rabbit population was reduced to about 25% of initial levels despite flea-borne myxomatosis, spreading only every second year (King and Wheeler 1979; King *et al.* 1985).

Rabbit populations persisted despite extreme disease mortality

King and Wheeler (1979) observed that at Cape Naturaliste, winter–spring outbreaks of flea-borne myxomatosis occurred in alternate years. The researchers analysed live-trapping data and compared the survival of young rabbits born at different times during the rabbit breeding seasons in years with and without flea-borne myxomatosis outbreaks (Fig. 5). In years when there was no myxomatosis, young rabbits born early in the breeding season survived well but those born late in the breeding season survived poorly. By contrast, in years when myxomatosis spread, fewer rabbits born early in the season survived but extra litters were produced late in the season and those young survived well.

The observations showed that flea-borne myxomatosis in winter and early spring caused heavy mortality and, in these years, young rabbits born late in the breeding season were a large proportion of those recruited into the breeding population in the following year.

Experiments carried out by Parer *et al.* (1985) provided further insights into the impact of flea-borne myxomatosis. The researchers divided their Lake Urana study area into four sections and, in two sections, a highly attenuated, vaccinating strain of myxoma virus called FS98 (Sobey *et al.* 1983) was spread using European rabbit fleas in early spring. In the other sections, fleas carrying the highly virulent Lausanne (Lu) virus were simultaneously released.

More virus was released on fleas in each section during later spring months.

Comparing the results obtained by Parer *et al.* (1985) with those of King and Wheeler (1979), it follows that (1) Lu myxoma virus caused extremely heavy mortality when spread among young rabbits by fleas, (2) field strains of myxoma virus at Cape Naturaliste were also highly lethal if spread by fleas, although not as extreme as was Lu, (3) the effect of spreading immunising virus FS98 on fleas was equivalent to an absence of myxomatosis, (4) production and survival of late-born young off-set some of the mortality caused by virulent viruses, and (5) flea-borne myxomatosis apparently reduced intraspecific competition for food, extending productivity of adult female rabbits and improving survival of young rabbits born late in the season.

Shepherd and Edmonds (1978*a*, p. 399) also inferred that late breeding compensated for high mortality from flea-borne myxomatosis, stating that 'outbreaks of myxomatosis during the winter or spring have coincided with the establishment of the European rabbit flea in the Mallee region. The severity of these outbreaks has varied from causing complete suppression of the normal spring increase in rabbit numbers to being completely ineffective in a year in which late spring rains allowed rabbit breeding to extend into the early summer.'

Fullagar (1977) also noted that after flea-borne winter outbreaks of myxomatosis near Canberra, most young rabbits recruited into the breeding population were born between winter disease outbreaks, outside the usual winter–spring rabbit breeding season.

From every study site, the same consistent picture has emerged. Although flea-borne myxomatosis killed large numbers of young rabbits, some born at the end of the normal breeding season or in summer were recruited into the adult rabbit population (Fig. 6).

Increases in pasture abundance associated with flea-borne myxomatosis

The shift in the timing of myxomatosis outbreaks meant that there were fewer young rabbits present during the main winter–spring pasture growing season. Less pasture vegetation was eaten and, in early summer, more fodder was available (Cooke 2021). The data, shown in Fig. 7, are averages from three pasture quadrats photographed every 4–8 weeks over 21 years. Peaks of pasture abundance in spring (October) in each year have been plotted against rainfall during the first 9 months of the corresponding year to correct for differences in rainfall. Estimated spring pasture biomass (kg ha^{-1}) more than doubled on the measured quadrats (Cooke 2021).

As rabbit density had averaged about two rabbits per hectare before rabbit fleas became established, and female rabbits at Belton produced an estimated average of 29 young per year (Gilbert *et al.* 1987), young rabbits would have consumed most of the palatable vegetation growing around each warren (Eldridge and Myers 2001).

From studies at Cape Naturaliste in Western Australia, where flea-borne myxomatosis broke out in alternate years, King and Wheeler (1979, p. 868) also concluded that 'the summer food supply in terms of quantity of standing dry pasture appears from photographs to be better in years when myxomatosis occurred than in other years.'

Food quantity or quality?

As adult wild rabbits eat about 100–150 g dry matter daily (Cooke 2014) and the estimated population densities of adult rabbits are between one and five rabbits per hectare (Fig. 4), they could not immediately eat a high proportion of the vegetation available in spring. Even if pasture vegetation eaten by their kittens was included, it would not amount to the 1000–3000 kg dry matter per hectare normally available (Fig. 7). However, because rabbits are highly selective feeders, seeking plant growing tips, flowers, and other high-quality components, it is more likely that as summer weather intensifies, and most annual vegetation matures, sets seed, and dries off, there is considerable intraspecific competition for the few highly nutritious components that remain. Older, experienced rabbits with large feeding territories would obviously have a major advantage over very young rabbits that confine themselves to the immediate, overgrazed surrounds of their warrens.

Food quality and survival of late-born young rabbits

In 1951, when myxomatosis was just becoming established and rabbits were still abundant in many areas, Myers (1962, p. 6) stated that the pastures 'were poor and obviously overgrazed, with an almost complete absence of better-quality rye [grass] [*Lolium* spp.] and clovers [*Trifolium* spp.] and with numerous weeds present.' However, the enormous growth in the sheep meat and wool industries after myxomatosis reduced rabbit abundance, indicated that there was a dramatic increase in available pasture vegetation (Waithman 1979). Nonetheless, as rabbit populations recovered, their grazing again reduced the amount of pasture vegetation and, as shown in Fig. 7, it was only after rabbit fleas were introduced that more pasture vegetation again became available.

The extra vegetation available in spring implies that high-quality components also increased and potentially explains how female rabbits could raise extra litters and why late-born young survived well. Nonetheless, such an idea would founder if it could be demonstrated that despite increased abundance, food quality in late spring and summer was never adequate for the production and survival of rabbit kittens.

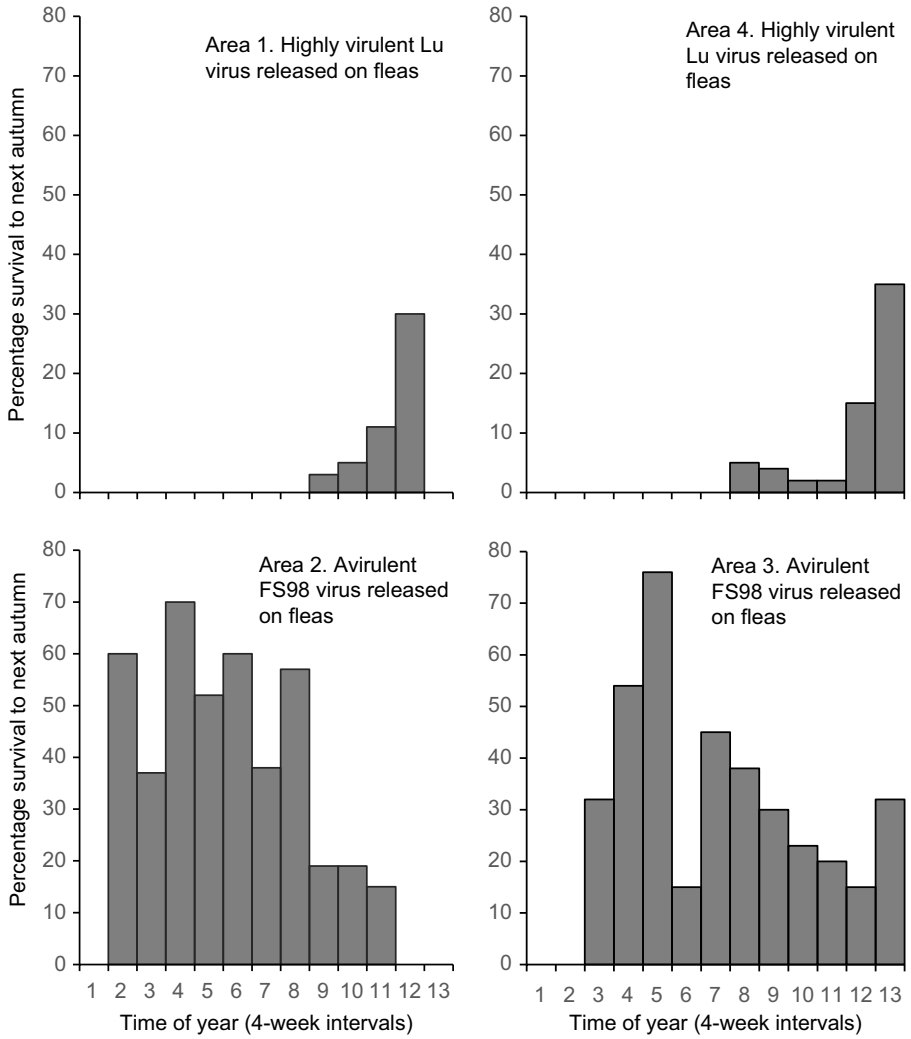

Fig. 6. Data have been redrawn from Parer *et al.* (1985), illustrating differences in recruitment of young rabbits in areas where highly virulent and avirulent viruses were released on rabbit fleas in 1978. Rabbits began breeding early in all areas, but only rabbits born late in the season avoided lethal Lu infection.

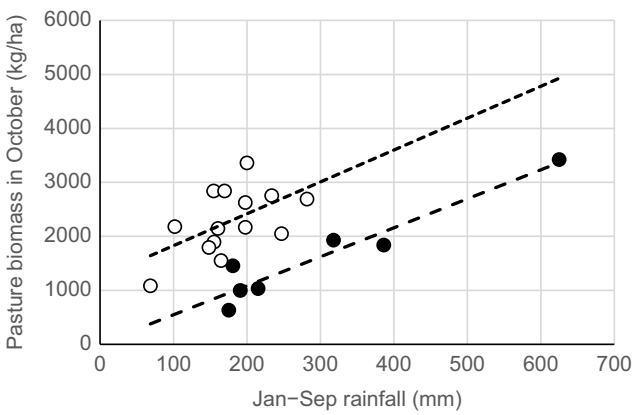

Fig. 7. Pasture biomass at Belton in October in relation to accumulated rainfall between January and September. (●), Before flea release; (○), after flea release (reproduced from Cooke 2021).

Some limited data are available on the quality of food eaten by rabbits immediately following the initial release of European rabbit fleas (Cooke 1974). To simplify matters, crude protein is used here as a convenient indicator of food quality because it is positively correlated with both the digestibility (energy content) and water content of vegetation (Cooke 2014). Analysis of freshly ingested vegetation from the stomachs of rabbits shot in the surrounds of the Witchitie study site showed that in late 1970, as fleas were still becoming established, the average crude protein content of food eaten was 12.5%. However, by late 1971 and during the following summer, the ingesta samples from individual female rabbits averaged 16.4% crude protein (range 12.6–25% crude protein; Cooke 1974). As rabbits require foods containing 12% crude protein for maintenance, 15% crude protein for pregnancy, 17% for

lactation and 16% for growth (National Academy of Sciences 1977), this suggests that food quality was adequate only for the maintenance of adult rabbits in late 1970, whereas in late 1971, it was adequate for rabbits to produce litters and to enable young rabbits to grow.

As no data on food quality were collected at Witchitie before rabbit fleas were present, or after they died out, it cannot be concluded that the improvement in food quality was a consequence of flea-borne myxomatosis reducing rabbit grazing pressure. Nonetheless, the possibility that summer food quality might always be too poor to enhance survival or for rabbits to produce late litters can be ruled out.

Rabbit social and territorial behaviour

Rabbits form tight social groups, often two or three females and one or two males living together in a warren (Mykytowycz 1959). In the breeding season, a territory immediately around the warren is defended, although individual adult rabbits roam over larger areas of 10–16 ha to feed (Cooke 1974; Stott 2003). At high warren densities, such as, for example, one warren per hectare, home ranges of rabbits from different warrens overlap extensively. This social behaviour has ecological significance because rapid improvements in pasture vegetation become obvious only when rabbit populations are held below 0.5 rabbits per hectare, i.e. when there is fewer than one warren in 10–16 ha (Cooke *et al.* 2010; Mutze *et al.* 2016). At that density, rabbits no longer constantly graze everywhere, and in seldom-visited areas, shrubs can regenerate successfully, and highly palatable plants can recolonise and out-compete inedible weeds (Mutze *et al.* 2016).

Such observations suggest that 0.5 adult rabbits per hectare is a key density below which the quality of pasture vegetation is usually high enough for young rabbits born late in the breeding season to survive well, and partially offset mortality caused by flea-borne myxomatosis.

Productivity of rabbits

Gilbert *et al.* (1987) calculated that female rabbits at Urana produced about 38 young per year, on average, whereas Cape Naturaliste and Belton rabbits produced 30 and 29 young per year respectively. Adult rabbit survival rates at these sites were similar at about 50–55% annually. Consequently, stable populations would be maintained if only 2–3% of rabbits born survived into the following year. Given such productivity, rabbit populations could withstand heavy disease mortality, and even small reductions in mortality could lead to quite large increases in rabbit abundance. For example, if 10% of rabbits survived, the adult rabbit population would be expected to increase two- to three-fold in the following season.

Overall, however, it can be concluded that flea-borne myxomatosis, probably augmented by predation (Cooke 2019), must have had an overriding influence to have kept rabbit populations low and stable for long periods (Fig. 4). Trout *et al.* (1992) confirmed this in Britain by using insecticides to eliminate rabbit fleas from experimental plots and demonstrating large increases in wild rabbit populations.

The underlying importance of virus virulence–rabbit resistance co-evolution

The standard laboratory strain (SLS) myxoma virus originally released in Australia quickly developed into an array of less virulent field strain viruses while simultaneously rabbits developed genetic resistance to myxomatosis (Fenner and Ratcliffe 1965). Although this lessened disease impact, it did not progress to a point where the myxoma virus no longer killed rabbits. Instead, increases in rabbit disease resistance were generally matched by increases in virus virulence that maintained a moderately high mortality rate in infected rabbits, usually judged to be between 40% and 60% (e.g. fig. 2 in Di Giallonardo and Holmes 2015). It is now generally accepted that this arms race has continued for over 70 years because some modern-day virus strains are far more virulent than the originally introduced SLS virus (Fenner and Fantini 1999; Kerr *et al.* 2017).

According to Di Giallonardo and Holmes (2015, p. 4) 'phylogenetic studies have revealed that there have been both decreases and increases in virulence among those MYXV [myxoma virus] lineages circulating in Australia, such that high virulence may be favoured in some localities. Accordingly, the evolution of virulence in MYXV is likely to be more nuanced than previously anticipated, and that it is in part mediated by local differences in rabbit ecology (including population size and density) and resistance.'

Before rabbit fleas were widely introduced, the dynamic balance between virus virulence and rabbit resistance had, no doubt, reached a point where transmissibility was maximised, although at a level of mortality well below that seen as myxomatosis first spread. Nonetheless, the introduction of rabbit fleas changed that, by enabling the disease to spread among young rabbits in winter and spring.

A conceptual model

A simple conceptual model relating rabbit abundance to disease mortality is illustrated in Fig. 8. It has been drawn using available data, including the observation by Parer *et al.* (1985) that rabbit populations did not fall below about 0.25 adult rabbits per hectare even when they spread the highly virulent Lu virus on fleas. It also anticipates that myxoma viruses that kill about 50% of adult rabbits would hold adult rabbit population densities at one or two rabbits per hectare and that rabbits immunised with attenuated FS98, equivalent to no myxomatosis, increased to densities exceeding eight rabbits per hectare (Parer *et al.* 1985).

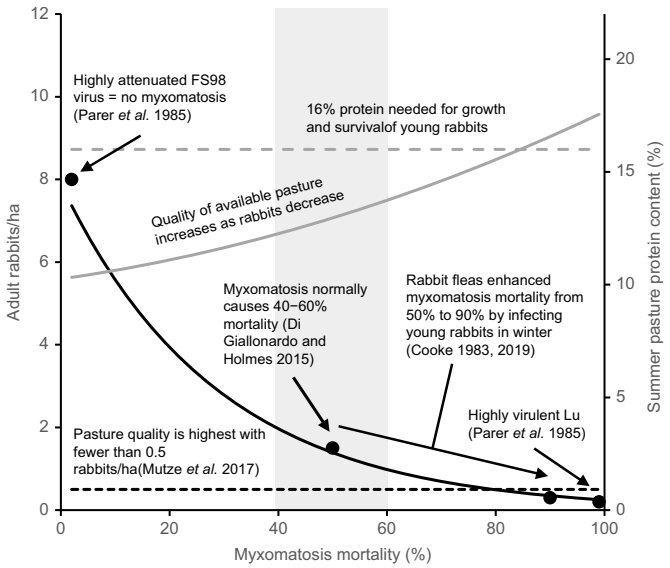

Fig. 8. Model indicating how rabbit abundance (rabbits per hectare) changes when fleas enhance myxomatosis mortality. As mortality increases, rabbits become less abundant (solid black line) but more high-protein food is available (grey line), especially after rabbit abundance falls below 0.5 rabbits per hectare (black broken line). When summer pasture provides food with more than 16% protein (grey broken line), additional litters are produced and late-born young rabbits survive better, offsetting high mortality caused by myxomatosis.

Myers (1962, p. 7) recorded that when myxomatosis was first introduced into Australian rabbits, the autumn–winter spread of myxomatosis was sometimes observed. However, he considered those outbreaks to be 'prolonged, low-level epizootics, probably reflections of ectoparasite activity and contact infection during the breeding seasons when social activities lead to increased contact between individuals.' Winter spread caused high mortality of rabbits (Mykytowycz 1956), but its slow spread probably caused far less mortality overall than did the intense outbreaks among rabbit kittens after rabbit fleas were introduced, and adult rabbit population density was held between about 0.25 and 0.50 rabbits per hectare at Witchitie and Belton respectively (Fig. 4).

Discussion

This review has several useful outcomes. First, it has shown that there were substantial benefits from introducing the fleas, although these were not widely appreciated at the time. This was because most published studies were aimed at recording the epidemiology of flea-borne myxomatosis rather than the long-term agricultural or ecological impacts. Nonetheless, events are now better understood because of recent re-evaluation and a better understanding of rabbit impacts on pasture productivity (Mutze *et al.* 2016; Cooke 2021).

It can be concluded that rabbit fleas enabled considerable savings of spring pasture biomass across temperate southern Australia (King and Wheeler 1979; Cooke 2021). However, unlike the situation following the introduction of myxomatosis (Waithman 1979), the extra pasture production was not followed by increased wool and meat production. This was because the release of fleas coincided with a peak of about 180 million in Australia's sheep population in 1970 when competition with synthetic fibres saw a Wool Deficiency Payments Scheme instigated to keep wool prices high, while stabilising the industry. This was followed by a global beef industry crash in 1973–74 (Greenwood *et al.* 2018) and the widespread loss of lucerne crops in 1977 with the arrival of the spotted alfalfa aphid (*Therioaphis trifolii*) in Australia (Hughes *et al.* 1987). Consequently, any benefits of flea-borne myxomatosis were impossible to discern within the succession of major disruptions to agricultural productivity.

Nonetheless, the extra pasture vegetation available in early summer no doubt helped grazing industries in general and is likely to have had important conservation outcomes. After the release of European rabbit fleas, swamp wallabies (*Wallabia bicolor*) expanded their distribution through western Victoria, south-eastern South Australia, and the Riverina area in western New South Wales (Cooke 2020). The area they have occupied beyond their historic range corresponds with areas where rabbit fleas are now permanently established (Cooke 2020).

A second outcome from this review is the development of a conceptual model of how rabbit abundance is regulated by the virulence of the myxoma virus, mediated through summer food quality. It potentially explains several of the anomalous observations that have long confused researchers. For example, it provides an explanation for Williams and Parer's (1972) observation that, prior to flea introduction, young rabbits recovered from myxomatosis in late spring, yet died from apparent food shortage in early summer. That could be explained if rabbits were abundant enough to reduce the available summer pasture quality to less than the 16% crude protein needed for growth of young rabbits, even though it was adequate to maintain adult rabbits.

The curve in Fig. 8 was drawn to illustrate this concept. It might be improved, should more information come to hand; however, in the meantime, it helps understand why the effectiveness of myxomatosis has changed as rabbits developed disease resistance and explains why releasing highly lethal myxoma viruses such as Lu did not lower rabbit abundance much more than did the naturally circulating field strain viruses carried by fleas (King and Wheeler 1979; Parer *et al.* 1985; Berman *et al.* 2006). The model can also be used to envisage how rabbit populations might increase if there was a substantial reduction in virus virulence and why flea-borne myxomatosis is less effective

in some habitats (e.g. semi-urban areas where rabbits with access to watered golf courses, ovals and gardens can produce extra litters in summer).

There is an obvious need to extend this simple model to more complex situations. Specifically, it would be useful to know how the introduction of rabbit haemorrhagic disease virus (RHDV) into Australia in 1995 might have changed the impact of flea-borne myxomatosis. It is unclear whether the release of RHDV enhanced or reduced the benefits of myxomatosis overall, despite some synergisms that increased rabbit mortality (Barnett *et al.* 2018). Kerr *et al.* (2019) showed that the myxoma virus has undergone faster evolutionary change since the spread of RHDV among Australian wild rabbits and it is important to assess the implications for future biological control. If any additional biocontrol agents were to be considered for introduction into Australia, it would be essential to know how they might complement or reduce the usefulness of myxomatosis by changing the timing of outbreaks and changing rabbit abundance as a result.

The ongoing coevolution of myxoma virus virulence and rabbit resistance is of critical importance. The model assumes that natural selection to maximise virus transmission is linked to steady mortality of about 50% in susceptible young adult rabbits if infected in summer. That translates as a much higher mortality among rabbit kittens under cold conditions in late winter and spring. This mortality rate has been maintained as a dynamic balance in an arms race where the myxoma virus has countered every increase in rabbit resistance with an increase in virulence (Fenner and Fantini 1999; Di Giallonardo and Holmes 2015; Kerr *et al.* 2017).

Using 12 years of capture–mark–recapture data from Turretfield in South Australia, where both RHD and myxomatosis affected the rabbit population, Fordham *et al.* (2012) assessed the influence of environmental, competition and disease conditions on rabbit survival and recruitment. They found that disease had the largest overall impact on rabbit survival, explaining 80% of the variance in survival rates. They also found that environmental as well as epidemiological conditions constrained rabbit survival, especially among younger animals. Variations in rabbit kitten recruitment patterns were determined by a combination of climate, competition, and disease settings that accounted for 68% of the variance. Temperature, especially, had a strong negative influence on kitten recruitment. They also concluded that recruitment responded positively to RHD, and negatively to myxomatosis, the former probably being mediated through a disease-driven effect on intraspecific competition for food.

The conclusions about the impact of diseases reached by Fordham *et al.* (2012) are broadly consistent with the simple, independently derived model proposed from this review. The same key elements such as temperature and the importance of food supply in mediating recruitment following disease outbreaks have been identified. However, the simpler model presented here has the advantage that it was developed from data collected before RHDV was introduced into Australia and it takes a step towards describing the mechanisms for interactions among environmental, disease and rabbit population variables rather than ranking their likely importance. It should be seen as a step towards building more complex models involving two or more diseases.

These findings are important for future rabbit management in Australia. In considering further use of biological control agents such as new diseases or vectors, it would be essential to ensure that flea-borne myxomatosis is not unduly disrupted by reducing its spread among young rabbits during the cooler winter months. This would ensure that the advantages of using myxomatosis are maintained by (1) maximising mortality (partly by enhanced predation), (2) increased humaneness, because, unlike older rabbits, young rabbits show few signs of disease before dying and there is seldom prolonged disease and secondary infection, (3) rabbits that survive myxomatosis but have a compromised immune system and are more likely to die from subsequent RHD and (4) the fact that there can be considerable benefits for livestock production and wildlife by eliminating young rabbits before they heavily damage pastures.

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