European rabbit fleas: contributions to rabbit control in Australia

Background

When myxomatosis was first introduced into Australia, mosquitoes were found to be the main vectors.

However, when myxomatosis was later introduced into Europe (including Britain), European rabbit fleas *Spilopsyllus cuniculi* were the major vectors.

Australian scientists tried to import the fleas, but so little was known about their breeding cycle those attempts failed.

Mead-Briggs (1960) subsequently discovered that fleas must feed on pregnant female rabbits and lay eggs in the nest alongside nestling rabbits for successful reproduction.

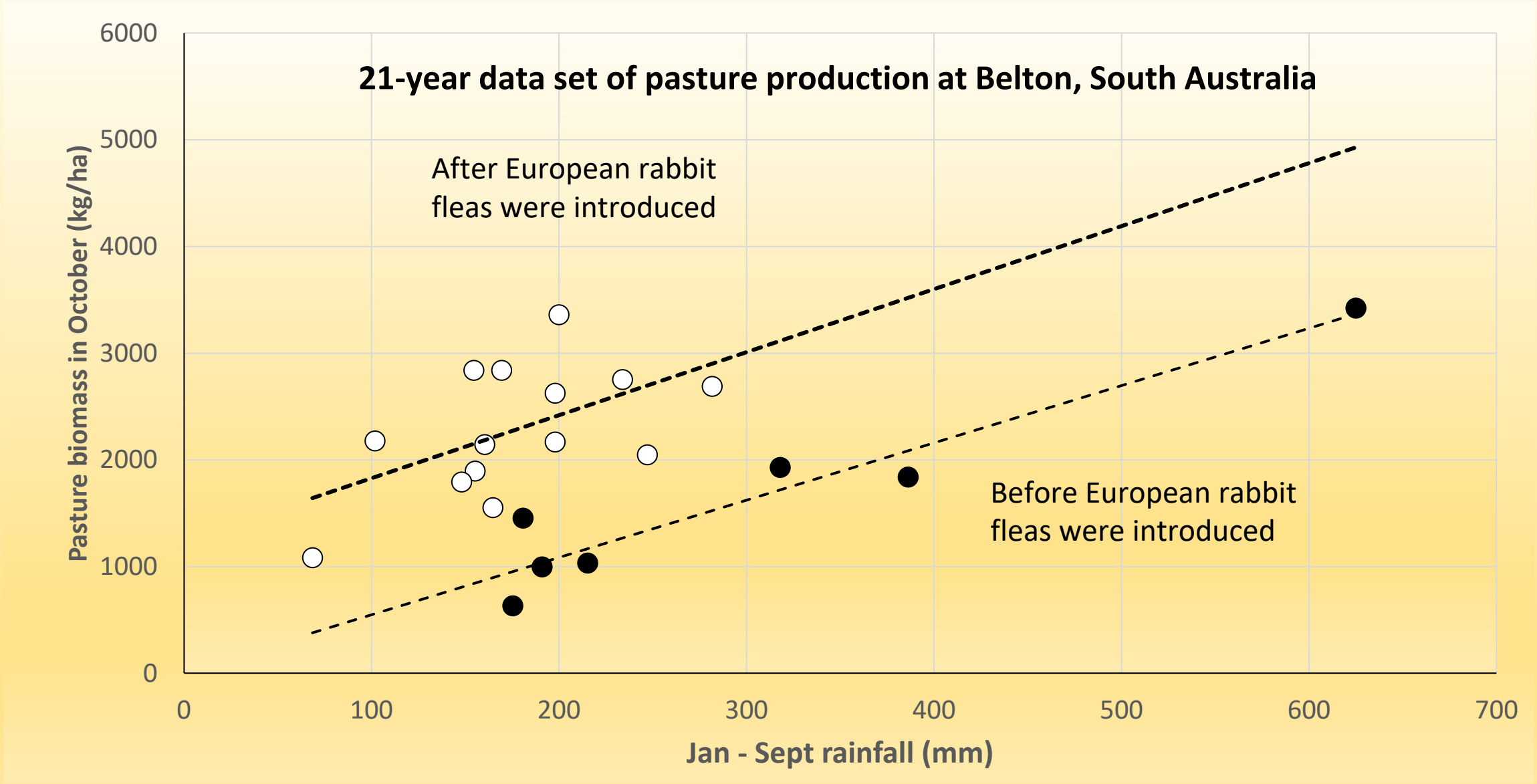
That allowed Bill Sobey from CSIRO Genetics to successfully import some and maintain a quarantine colony (Sobey, Menzies and Conolly 1974)

Improved rabbit control

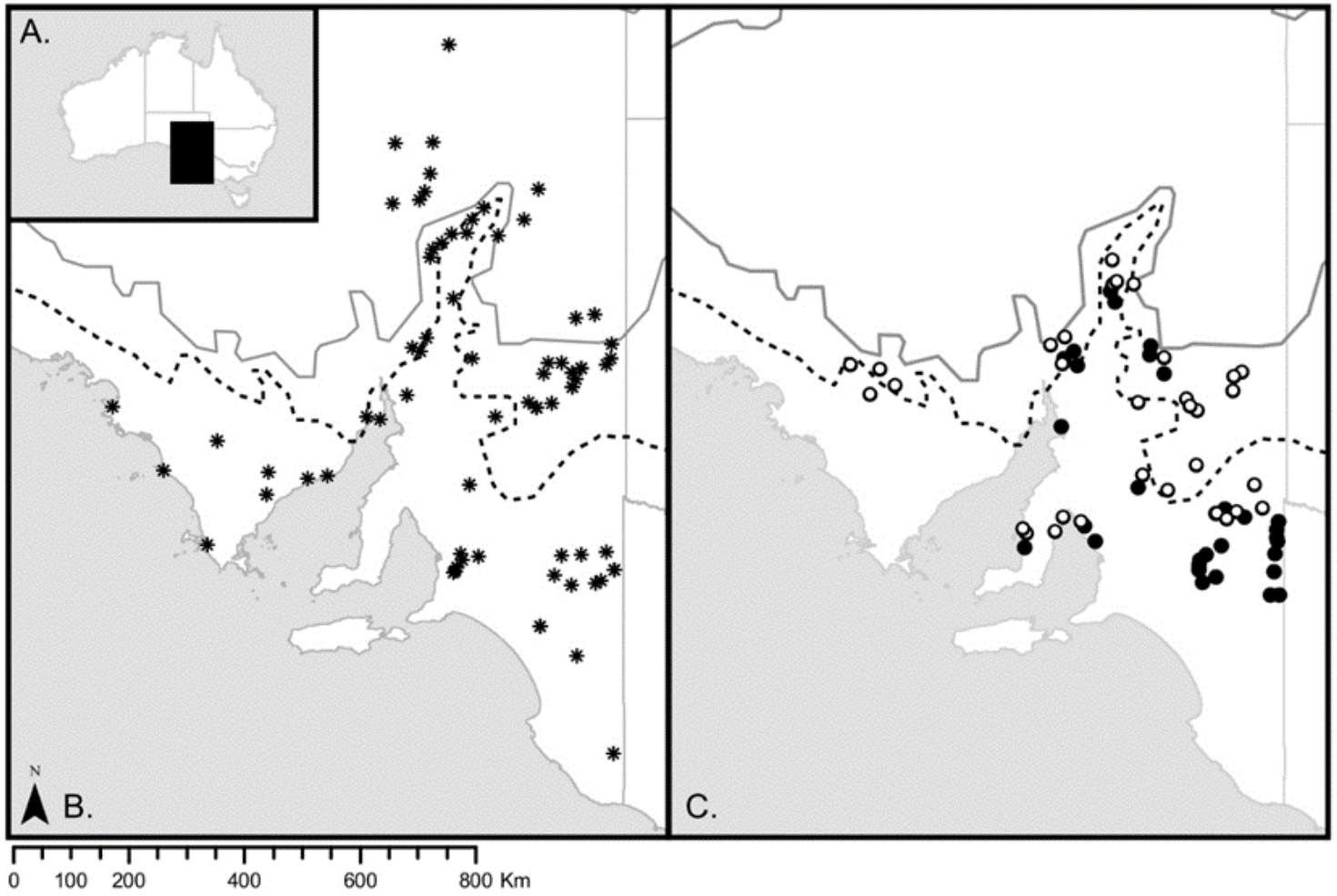
On release, after quarantine assessment, the fleas quickly became established in higher rainfall areas and changed the epidemiology of myxomatosis

Instead of mosquitoes spreading the virus in late spring, the fleas spread the virus among much younger rabbits in the colder winter months. This increased mortality from about 50% to over 90% because young rabbits are more susceptible to myxomatosis than older rabbits and low temperatures further enhanced mortality (Fenner & Marshall 1954; Marshall 1959).

Because young rabbits were killed during the winter pasture growing season there was more pasture biomass available in spring. It was also of better quality (Cooke 2022).


21-year data set of pasture production at Belton, South Australia
After European rabbit fleas were introduced
Before European rabbit fleas were introduced
Pasture biomass in October (kg/ha)
Jan - Sept rainfall (mm)
0
1000
2000
3000
4000
5000
6000
0
100
200
300
400
500
600
700

Unfortunately, European rabbit fleas were unable to persist in inland Australia where rainfall is less than 250 mm annually



A. General area covered by map

B. Sites where European rabbit flea releases were made

C. Sites subsequently surveyed to detect European rabbit fleas

Broken line = 250 mm rainfall

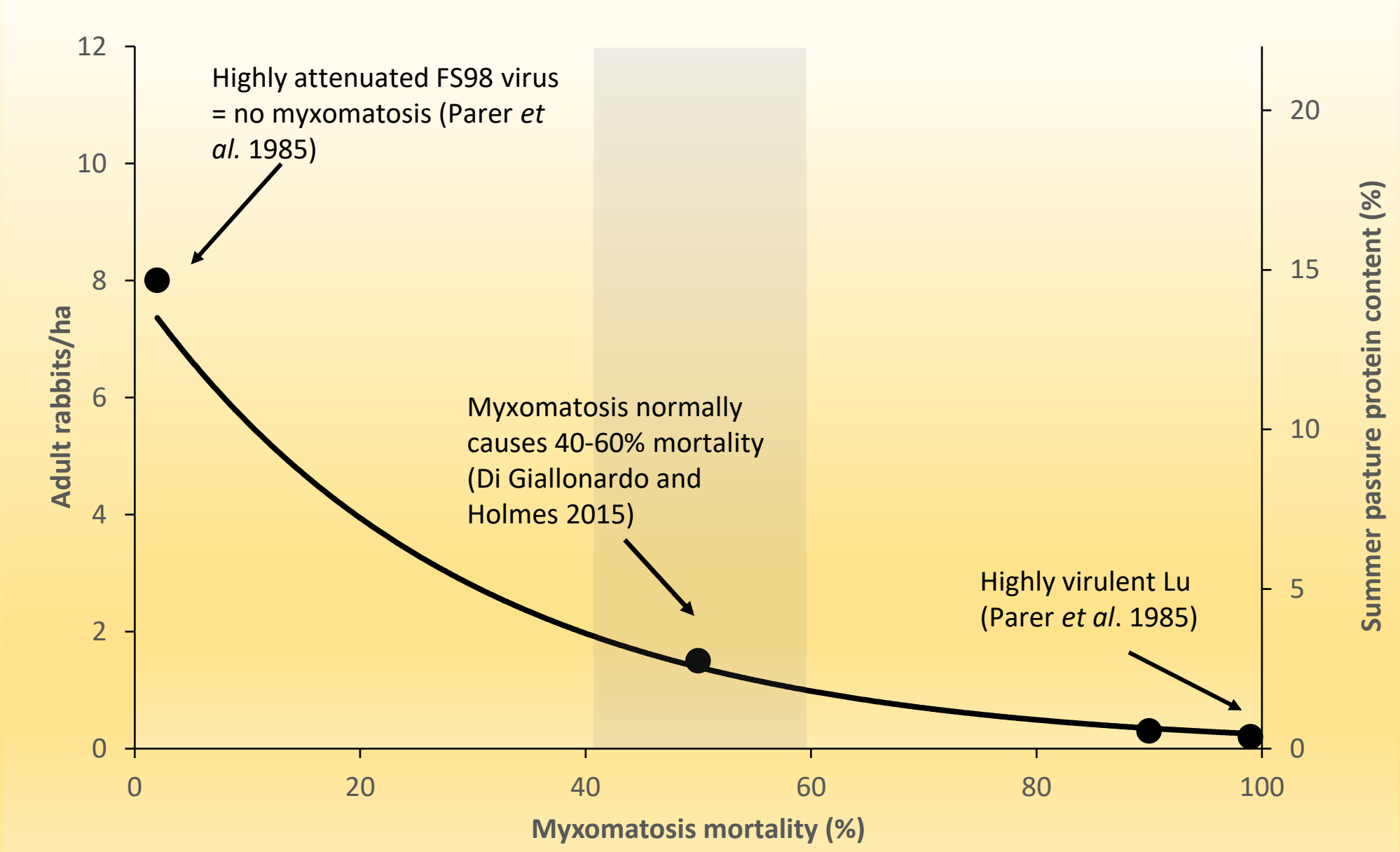
Figure prepared by Emilie Roy-Dufresne

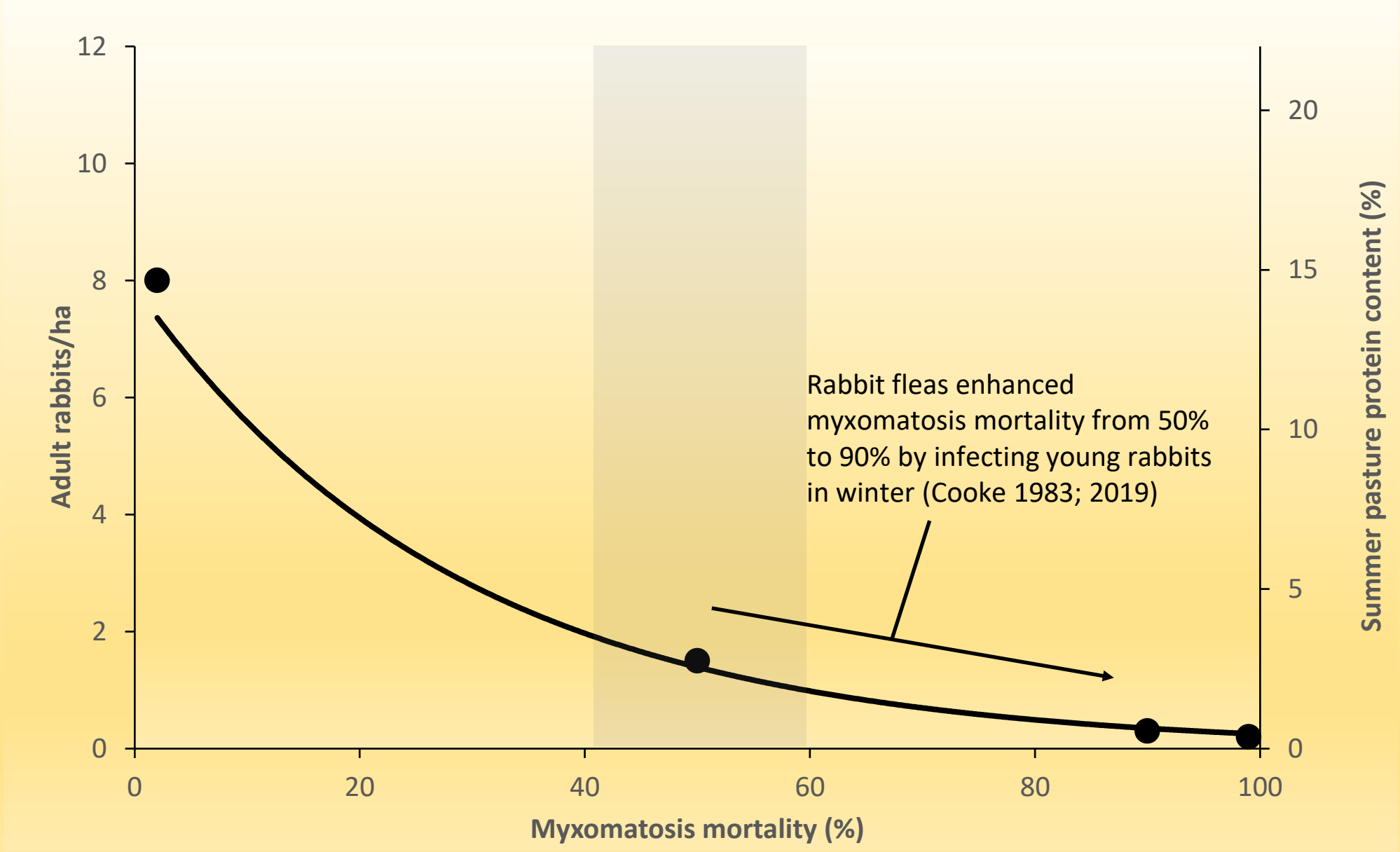
A comprehensive model

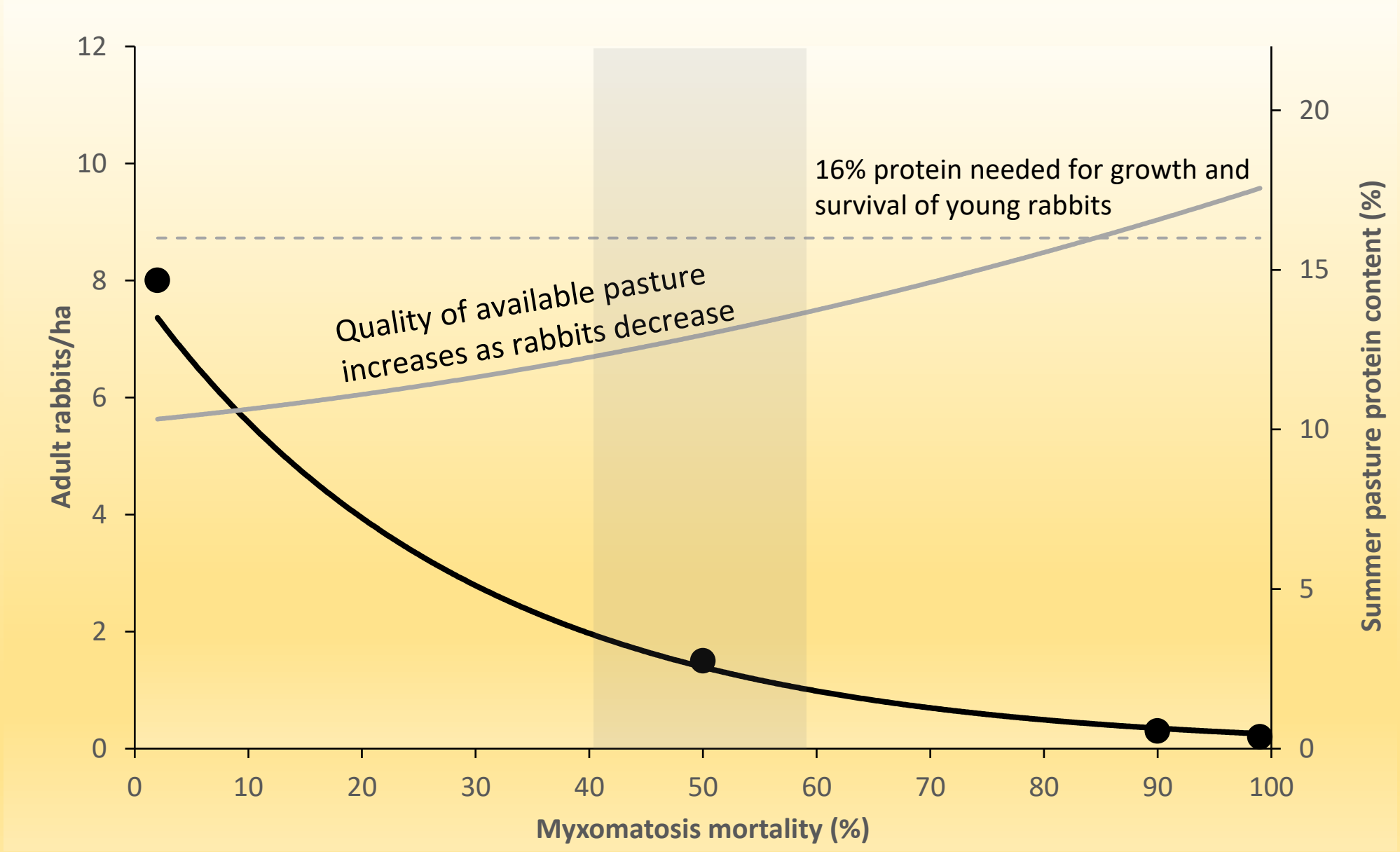
To accommodate all relevant published information, the best epidemiological model is one that relates myxomatosis mortality (%) as a measure of virus virulence to adult rabbit density (adult rabbits/ha).

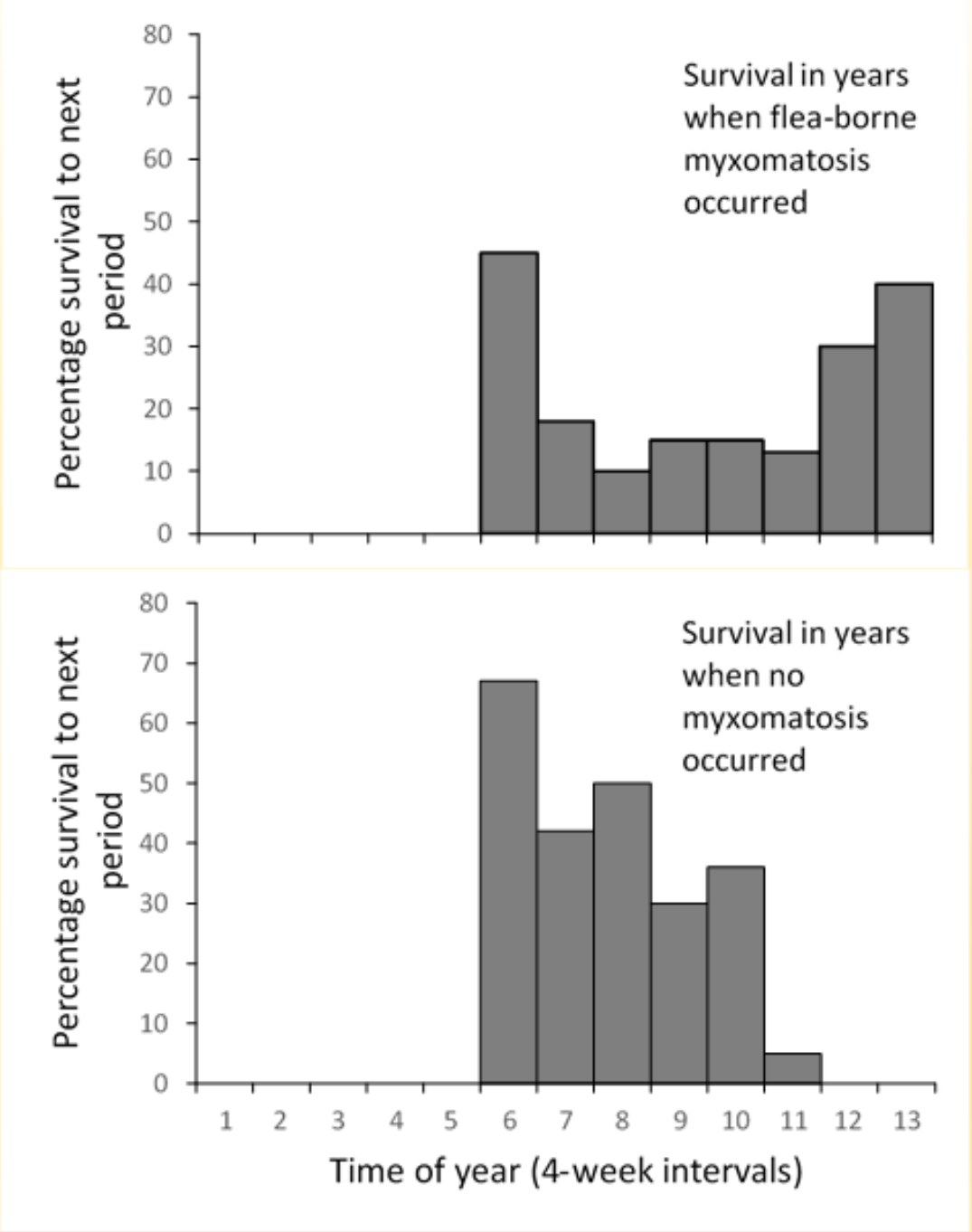
This is an inverse curvilinear relationship, not a linear one. As myxomatosis kills an increasing proportion of young rabbits, food supply increases allowing rabbits to breed into early summer. By that time, the abundance of fleas has dwindled, and temperatures are higher allowing more rabbits to survive the disease.

This explains why increasing virus virulence (e.g., releasing highly virulent virus like Lausanne virus) only produced marginal gains beyond those produced by European rabbit fleas spreading field strain viruses in winter.

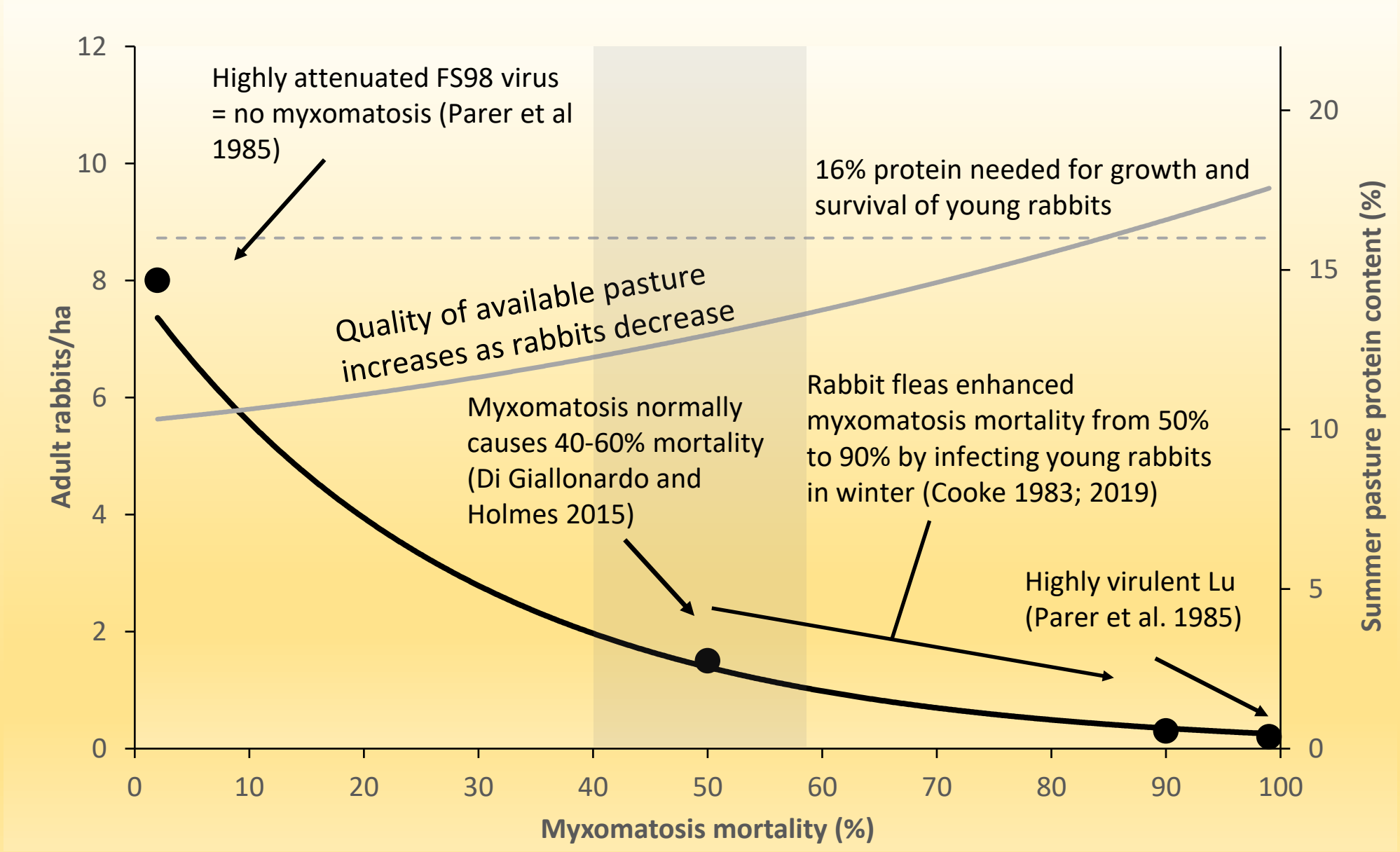

Highly attenuated FS98 virus = no myxomatosis (Parer *et al.* 1985)
Myxomatosis normally causes 40-60% mortality (Di Giallonardo and Holmes 2015)
Highly virulent Lu (Parer *et al*. 1985)
Adult rabbits/ha
Summer pasture protein content (%)
Myxomatosis mortality (%)
0
2
4
6
8
10
12
0
5
10
15
20
0
20
40
60
80
100


12
10
8
6
4
2
0
Adult rabbits/ha
20
15
10
5
0
Summer pasture protein content (%)
0
20
40
60
80
100
Myxomatosis mortality (%)
Rabbit fleas enhanced myxomatosis mortality from 50% to 90% by infecting young rabbits in winter (Cooke 1983; 2019)


12
10
8
6
4
2
0
Adult rabbits/ha
20
15
10
5
0
Summer pasture protein content (%)
16% protein needed for growth and survival of young rabbits
Quality of available pasture increases as rabbits decrease
0
10
20
30
40
50
60
70
80
90
100
Myxomatosis mortality (%)



From King and Wheeler (1979)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70
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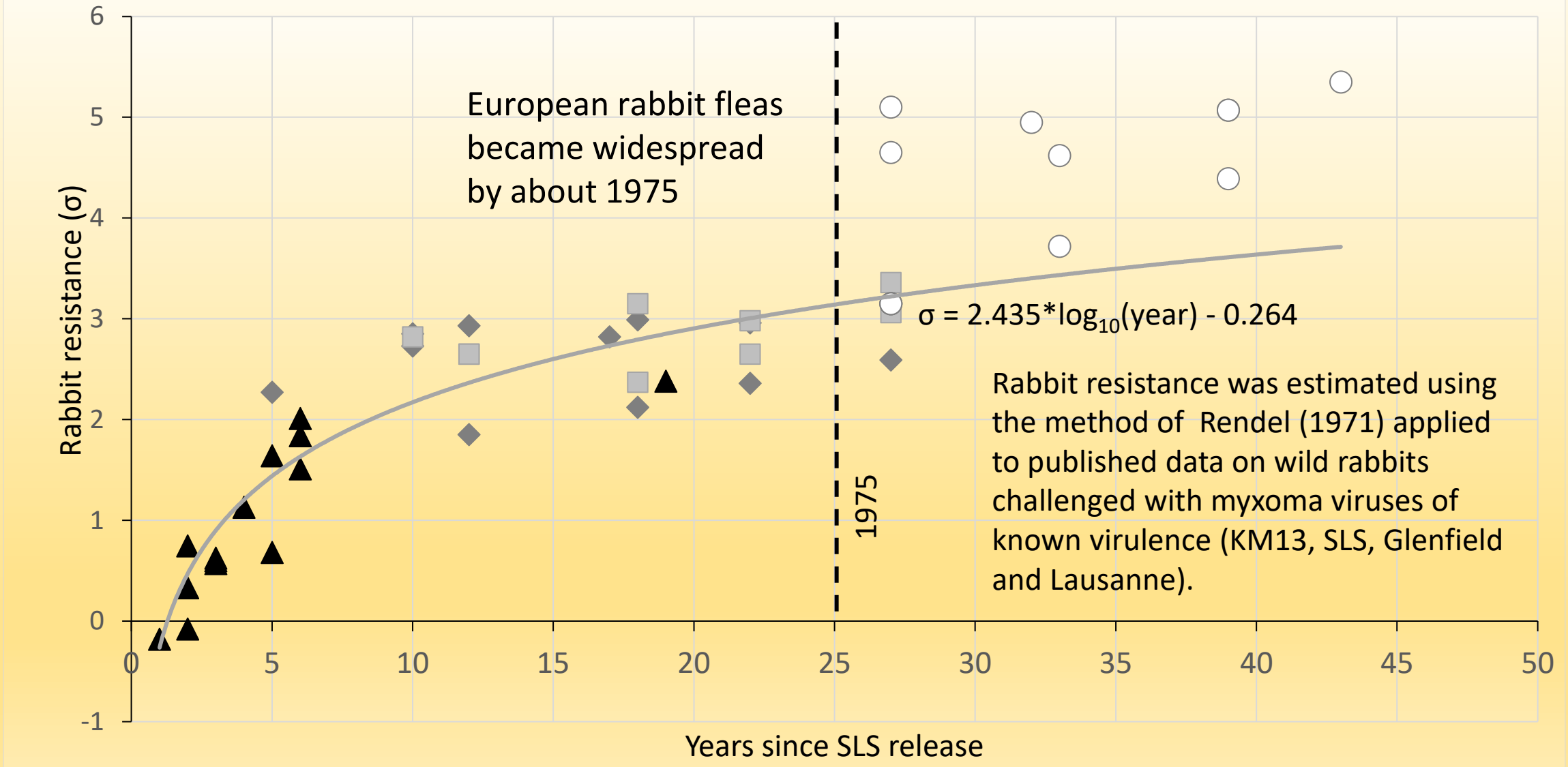
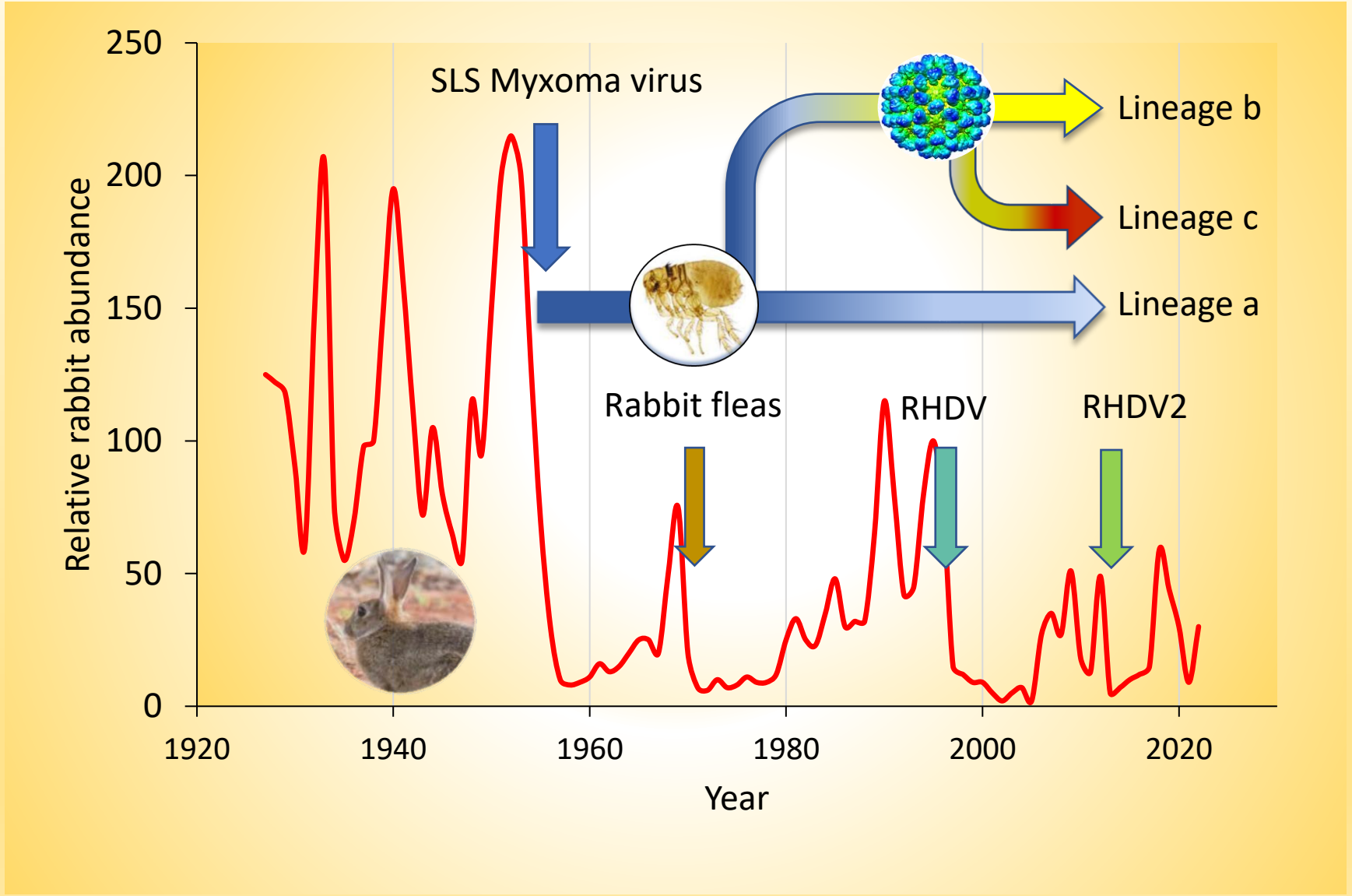
90
100

Implications

Knowing more about the role of European rabbit fleas in spreading myxomatosis helps us think about biological control agents for future rabbit control

- It is generally considered that rabbits and myxoma virus are in an ‘arms race’ and myxoma virus virulence adjusts to every gain in rabbit resistance. But is there field evidence for this in the case of the release of European rabbit fleas?
- Does the introduction of European rabbit fleas provide an explanation for the emergence of a second genetically recognizable lineage of myxoma virus as Kerr *et al*. (2017) suggested?
- Do we have the best vectors available? Should we import *Caenopsylla laptevi relicta* which is winter breeding and adapted to arid areas? It might be a better vector in inland Australia than the ‘Spanish’ rabbit flea *Xenopsylla cunicularis*.


European rabbit fleas became widespread by about 1975
σ = 2.435*log10(year) - 0.264
Rabbit resistance was estimated using the method of Rendel (1971) applied to published data on wild rabbits challenged with myxoma viruses of known virulence (KM13, SLS, Glenfield and Lausanne).
1975
Rabbit resistance (σ)
Years since SLS release
6
5
4
3
2
1
0
-1
0
5
10
15
20
25
30
35
40
45
50


250
200
150
100
50
0
Relative rabbit abundance
1920
1940
1960
1980
2000
2020
Year
SLS Myxoma virus
Lineage b
Lineage c
Lineage a
Rabbit fleas
RHDV
RHDV2

Introduction of new vectors?

Although Spanish rabbit fleas were introduced into Australia over 30 years ago, any effect they had on the epidemiology of myxomatosis was obscured by the spread of rabbit haemorrhagic disease soon after.

However, if European rabbit fleas are effective because the are abundant in winter and spread myxomatosis in colder weather, then Spanish rabbit fleas may not be the best available vectors because they are summer breeders and spread myxomatosis in the hotter months when it is less effective.

Nonetheless, there are other winter breeding vectors such as the rabbit flea *Caenopsylla laptevi relicta* that might be considered for use in inland Australia.

Summary

Making decisions about the biological control of rabbits is now known to be very complex.

Interactions between virus, host and other factors such as seasonality and food supply make it increasingly difficult to reach decisions on what to do next. We need to proceed with care.

Nonetheless, there are ways forward that might be exploited to maintain a high level of vector maintained biological control and help us stay on top of Australia’s rabbit problem in the long-term.