Rabbit abundance.

A dance between myxoma virulence, rabbit resistance and summer feed.

Highlights from a fifty-year review of how European rabbit fleas enhanced the efficacy of myxomatosis for controlling Australian Rabbits, by Brian Cooke.

A recent journal article by Brian Cooke reviewed fifty years of evidence to better understand how myxomatosis works in Australia, and to develop a conceptual model of the interplay between key factors. Key points are summarised below. The article is available in Wildlife Research, July 2022.

Following extensive research, the standard laboratory strain (SLS) of the myxoma virus (MYXV) was introduced to Australia in 1950. Spread by mosquitoes in early summer it reduced Australia's rabbit population, estimated at 1-3 billion, by over 90%. However, two things followed quite quickly. Within 2 years wild rabbits were evolving genetic resistance and new strains of the virus were appearing. Those strains ranged from so virulent that rabbits died before the virus could spread, to so benign that transmission by biting insects was ineffectual. Intermediate strains were a balance between virulence and transmissibility, but it looked like rabbits were winning the war.

It is interesting to note that MYXV strains have been changing faster since the introduction of RHDV (calicivirus) to Australia. Some modern strains of myxomatosis are far more virulent than the original SLS strain, but a highly virulent strain (Lu) did not lower the abundance of rabbits below 0.25 rabbits/ha.

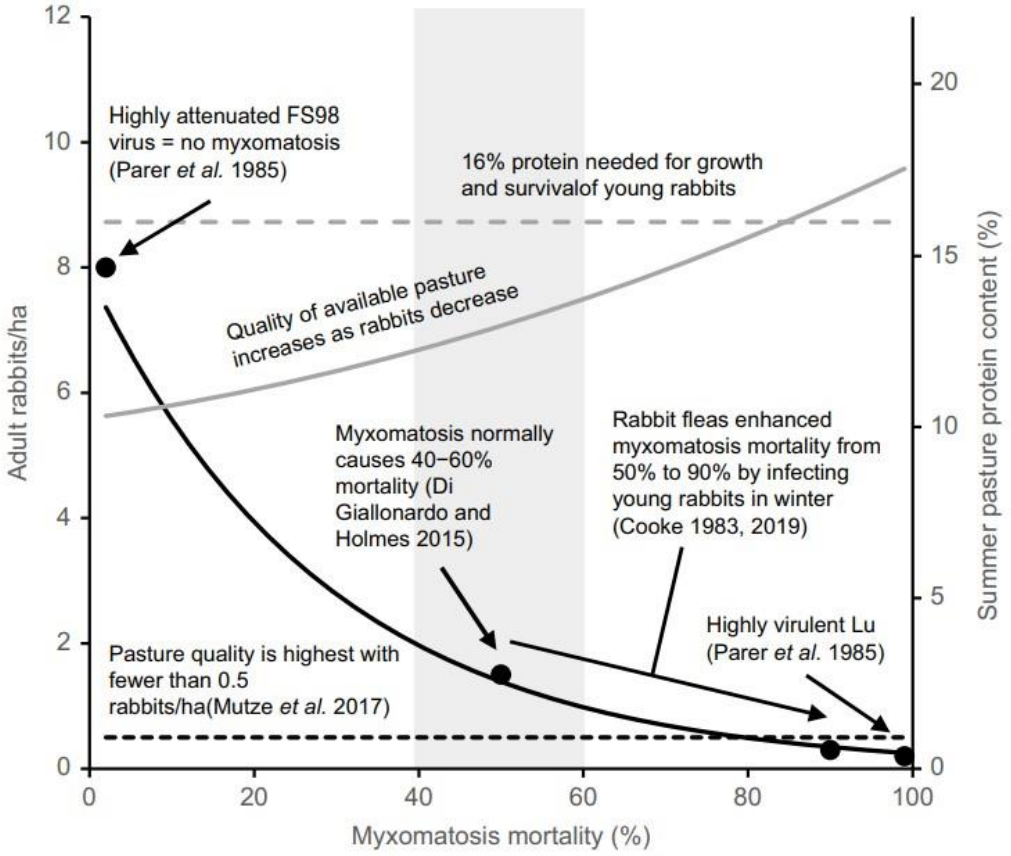
European rabbit fleas, which only breed on pregnant rabbits, were subsequently introduced to speed-up the transmission of myxomatosis, with laboratory trials beginning in 1996 and field trials in 1968. It soon became apparent that the fleas spread MYXV much earlier in the season, in winter and early spring rather than summer, and caused extremely high mortality rates; often from 80-96%. That was much higher than the 40-60% mortality rate associated with mosquito-borne summer outbreaks, although the European fleas did not survive where annual rainfall was less than 200–250 mm and warren micro-climates were too extreme.

The reduction in young rabbit numbers in early spring resulted in more pasture being available in late spring, extending the rabbits' breeding season and resulting in more late-born rabbits surviving into summer. This affect was apparent even for a highly virulent myxoma strain (Lu) used in a trial. The importance of good quality feed to rabbit breeding and survival is highlighted by their protein requirements; crude protein levels of 12% in feeds to maintain condition, 15% for pregnancy, 17% for lactation and 18% for growth. The early season success of myxomatosis contributed to more useful feed but the late season births and survival of young rabbits compensated to a degree for the greater mortality caused by flea-borne myxomatosis.

Rabbits in a small to medium sized warren will have a home range of about 10 – 16 ha, equivalent to around 0.5 rabbits/ha. When present in numbers above that density the grazing range overlaps that of other warrens exerting higher grazing pressure on pastures. Below that density there is more chance of palatable feeds being able to survive and of rabbit's nutritional needs being met.

Evidence from long-term field trials indicates that rabbit-flea borne MYXV, probably augmented by some predation, was able to keep rabbit populations relatively low (2 rabbits/ha) for long periods. Because of the high breeding rate of rabbits, only 2-3% of the young rabbits born each year need to survive to replace adults that die and to maintain the population.

The interplay between rabbit numbers (rabbits/ha), the relative virulence of myxomatosis (% mortality), and the availability of good early summer feed (% protein) is depicted in the following conceptual model.



As mortality (the success of virulence over resistance) 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.

The model helps explain why the effectiveness of MYXV changed as the balance between disease resistance and virulence evolved, and why highly lethal strains like Lu did not eliminate rabbits. It begs the development of a better understanding, and modelling, of the interaction of RHDV and MYXV. It would be useful to consider a similar model for RHDV, and one combining the two viruses (and other pathogens) and factoring in the increased vulnerability to RHDV exhibited by rabbits previously infected with MYXV.

This understanding also shows the importance of testing any new biocontrol agents before release to ensure that they would not reduce the ability of MYXV to infect young rabbits in winter and early spring. Otherwise, the net benefits of introducing the new control agent could be offset by a reduction in the efficacy of myxomatosis.

Key Points:

- Myxomatosis transmitted by European rabbit fleas kills young rabbits in winter and early spring, resulting in less grazing pressure and more good quality feed later in the season.
- High protein food allows effective late season breeding with enough young surviving to maintain the next generation.
- Rabbits can develop genetic resistance, yet viruses may develop greater virulence and maintain mortality rates.