Rabbits are doing what comes naturally – again

By Louise Lawrence, CSIRO Entomology

So much has been written about rabbits – probably as many words as there are rabbits – with more to come. Domesticated rabbits came with the First Fleet but it wasn't until 1859 when Thomas Austin released 26 on his property in Victoria that their take-over began. Little did Austin know what damage his desire for recreational hunting would cause to Australia.

Over the next 70 years, rabbits became Australia's most successful feral animal. Their economic damage to agriculture through crop loss and competition with livestock was matched only by their ecological damage.

They devastated huge areas of countryside destroying native vegetation and causing erosion. Their presence was also a boon to other introduced feral animals such as foxes and cats for two reasons. Small native animals no longer had cover in landscapes denuded by rabbits and they were themselves a food source for feral predators.

Search for biological control

The methods tried to control rabbits were many and varied – warren ripping, shooting, poisons, even car exhaust gases piped down into warrens. Then the search for biological control agents began. The story of myxomatosis is well known, including the fact that the scientists involved injected each other with the virus to prove it was safe for humans.

Initially the release of myxomatosis in 1950 was a huge success as it killed 90 per cent of the feral rabbits who caught the disease and reduced the population from an estimated 600 million rabbits to under 100 million. But within a few years, rabbits had begun to develop resistance to it. Then the virus 'fought back' and became more virulent until an equilibrium was reached.

Myxomatosis still kills 40–50 per cent of the rabbits it infects.

The latest biological control attempt has been with rabbit haemorrhagic disease virus (RHDV), also known in Australia as rabbit calicivirus. RHDV, an infectious hepatitis specific to rabbits, was first described in China in 1984 where it was causing mortality of more than 95 per cent in infected rabbits (an infected rabbit dies within 72 hours).

In June 1991, the virus was brought into quarantine at the CSIRO Australian Animal Health Laboratory in Geelong for comprehensive testing. During field trials in 1995, it escaped from Wardang Island off the South Australian coast, with the help of insects.

The virus then raced across the Flinders Ranges through the arid zone, killing large numbers of rabbits as it went. But in cool, humid parts of the country, mortality was much lower.

Was there a link?

Could there be a link between this and the fact that some wild Australian rabbits tested before the release of calicivirus had antibodies to RHDV?

Those rabbits with high levels of these antibodies were discovered to have some protection to RHDV. It seemed probable that there were other viruses out there that were similar to but not identical to RHDV but were non-pathogenic – that is they did not kill the rabbits they infected.

The possibility was that these viruses were providing rabbits with immunity to RHDV. While attempts to isolate such a virus in wild rabbits in Australia had in the past been unsuccessful, another non-lethal rabbit calicivirus (RCV) had already been identified overseas.

When the serious search for these 'other' viruses began in Australia, scientists from the Invasive Animal Cooperative Research Centre, led by Dr Tanja Strive from CSIRO Entomology, faced a number of unknowns. Where in Australia should they look? How would they detect the virus? Where in the rabbit would it be found? At what age were rabbits infected and in what season?

It was a real 'needle in a haystack' search.

Virus search

The scientists began by trapping young rabbits at several sites in south-east Aus-

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tralia which had annual rainfall of above 600 mm. They tested the rabbits to pick up nucleic acids from any Lagoviruses, the group of caliciviruses that infect rabbits and hares and cause RHDV.

They finally found the elusive virus in rabbits from Michelago in southeast New South Wales and named it rabbit calicivirus Australia 1 (RCV-A1). The highest concentration of the virus was found in the small intestine. It appears that the virus is concentrated in faecal matter then shed into the environment in pellets which suggests a faecal-oral transmission route.

The scientists believe it is possible that a RCV-A1 ancestor arrived with the first rabbits and that it has evolved into its current form while, in Europe, different RCV viruses evolved. The disease-causing forms of RHDV are believed to have evolved later from European non-pathogenic (non-disease causing) forms. Studies are now underway to investigate where in Australia it is present and if there is perhaps more than one non-pathogenic calicivirus.

It is possible that an increased prevalence of non-pathogenic caliciviruses in high rainfall areas is providing rabbits with some immunity to RDHV and hence reducing its effectiveness. There was certainly no sign of an RHDV outbreak in the populations where RCV-A1 was found.

Proof was needed

But control infection studies needed to be done to provide proof that RCV-A1 is indeed non-pathogenic and, importantly, to find if it can indeed provide protective cross-immunity to RDHV.

In trials that looked at whether RCV-A1 was pathogenic, scientists infected rabbits with the virus then looked at its progress thereafter. No animals showed any signs of the disease over the whole period.

They also challenged a small group of rabbits infected with RCV-A1 with RHDV. All the rabbits in the control group (no RCV-A1) died but half of the treatment group survived. Although only small numbers of animals were used in this study this supports the idea that the new virus can interfere with RHDV – though it appears the protection is not complete.

These results showed that previous infection with the new virus can prevent lethal RHDV infection. This means that where the new virus is present more rabbits may survive an RHDV outbreak. The study also showed that rabbits previously infected with RCV-A1 that survived RHDV infection had boosted antibodies to RHDV.

Further trials are needed on greater numbers of rabbits to confirm these findings but the evidence so far supports the fact that the new virus may be responsible for reduced effectiveness of RHDV in some areas of Australia.

Scientists searched for evidence of the benign calicivirus by trapping young rabbits at several sites in south-east Australia with annual rainfall of more than 600 mm.

Scientists are now focussing on field studies of rabbit populations where both viruses are present to ascertain when during the year they are active, how the two viruses interact and if there is a window of opportunity when RHDV could still be effective.

They are also looking at the distribution of RCV-A1 in Australia to determine whether it is patchy or continuous and what effect factors such as geographical features, patchy rabbit distribution and different habitats have on the virus.

The potential effects of climate change and whether further spread of the virus can be prevented are also being investigated. Added to these issues is the possibility that rabbits are building up a genetic resistance to RHDV.

Lessons to be learned

One thing is sure. Rabbit numbers are building up again and there are management lessons to be learned from this. With funding support to the Invasive Animals Cooperative Research Centre from the Australian Government, Meat and Livestock Australia, Australian Wool Innovation and the Foundation for Rabbit Free Australia, CSIRO scientists are working with NSW Industry and Investment's Vertebrate Pest Research Unit and the Elizabeth Macarthur Agricultural Institute to investigate other strains of RHDV.

The aim is to identify strains of the virulent virus that may be able to better overcome cross protection by RCV-A1 or potential developing genetic resistance to the current RHDV strain.

While this research on enhancing biocontrols is being carried out, other management strategies need to be used in the areas affected by RCV-A1. These include integrated pest management and the Myxoma virus. If a window of opportunity for the use of RHDV is found, then good use should be made of it and control attempts should be made to eradicate affected populations.

A combination of control methods should provide better rabbit control thus reducing their impact and improving the management of ecosystems.

It is important that land managers work together on control efforts and check to see if they are working. They should regularly check their rabbit numbers, and do follow up treatments if they are needed.

Biocontrol is the most effective control tool but it never kills 100 per cent of rabbits. Even if the kill rate is 95 per cent or better, land managers have to follow up and use the tried and true old fashioned control methods on the survivors. Or, who knows, they might breed like rabbits.

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