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Modelling hypotheticals

A man should look for what is, and not for what he thinks should be.

Albert Einstein

With the collapse of the theory that RHDV had two modes of spread it became important to return to ideas put forward by David Chasey and others when RHD first began to spread into Britain (Chasey *et al*. 1997). They had found that, even before RHD became fully established, most rabbits already carried antibodies that protected them against acute RHD. These antibodies had indicated the presence of another naturally circulating virus that was non-pathogenic, and the scientists argued that such antibodies would most likely protect wild rabbits from acute disease. At the time, this idea had been further investigated by Peter White and colleagues who constructed a mathematical epidemiological model to explore the likely behaviour of virulent RHD as it spread into an environment where non-pathogenic immunising strains of lagoviruses already circulated (White *et al*. 2001).

In their theoretical model White and colleagues assumed that rabbits infected with the non-pathogenic virus would be fully protected against RHDV and that the non-pathogenic virus could not spread through the few rabbits that might contract RHD yet survive. Depending on the relative capacity of each virus to cause infection and spread, it was argued that, in some instances, the arrival of RHD should cause only a slight population depression that would be recouped within a couple of years. In other situations however, the disease might have small impact in some years but heavier impacts in others. In extreme cases however, RHD could drive populations steadily downwards over several years. It was further argued that if the pathogenic and non-pathogenic viruses were likely to compete in this way then one virus may eventually dominate over the other and drive it to extinction, at least on a local scale. The modellers also thought that RHD was likely to have the most noticeable impact in the south of England where a lower proportion of rabbits carried antibodies against the presumed non-pathogenic virus.

These predicted patterns of disease behaviour share some characteristics with the variable epidemiological outcomes previously described for different parts of Australia (Chapter 15). Indeed, there is some evidence that non-pathogenic RCV-A1 may have disappeared from marginal inland parts of its distribution after RHDV spread. An unpublished re-analysis by Liu and Strive of blood samples collected at the time has shown that the prevalence of specific RCV-A1 antibodies declined quickly in the first 2 years after the virulent virus arrived. Now, some 17 years later, there is no serological evidence that RCV-A1 was ever present in the area. Nevertheless, this does not confirm that the epidemiological model developed by White *et al*. (2001) is generally applicable. It was based on an entirely different set of assumptions and we now know that prior infection with non-pathogenic RCV-A1 in Australia does not fully protect all rabbits against acute RHD. Rabbits can also be re-exposed to the virulent virus