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Discovering kindred

We cannot destroy kindred: our chains stretch a little sometimes, but they never break.

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It is common to meet with some surprises when tracing genealogies or simple family histories. On my wife's side of the family, for example, it was known that great-grandfather Hein had arrived in Australia from Hamburg in Germany in September 1854, and that seemed a good starting point for documenting the Australian branch of the family tree. But suddenly, from records held by port authorities in Melbourne, it became obvious that he had arrived with his parents, Johann Wilhelm Hein and Johanne Dorothea Strauch, as well as several brothers and sisters. This meant that the genealogy took several more years to complete as additional large family branches had to be contacted and persuaded to take part (Hein 1978). Finally, a country football ground was hired to accommodate everyone and allow enough space for cars, barbecues, folding chairs and picnic rugs. A beautifully-presented book on the family history and genealogy was launched and memories willingly shared along with yeasty plum-cake topped with streusel, Kaiser buns, bienenstich and other traditional deutsche-küchen.

There is more than a whiff of a similar possibility as we begin to explore the genealogy of RHDV, with some trepidation that things could get a little out of hand as we delve back deeper in time. The saga began when Lorenzo Capucci showed that a non-pathogenic calicivirus related to RHDV was circulating in domestic rabbits. It opened the door for speculation that similar non-pathogenic virus family groups were circulating in wild rabbits, and indeed there was tantalising evidence readily at hand.

Before virulent RHDV spread through Britain, Drs Roger Trout and David Chasey told me, most wild rabbits already carried antibodies that cross-reacted in tests for RHDV antibodies. As well, it was found that those rabbits that carried antibodies were fully protected against experimental RHDV infection. In the absence of any obvious disease outbreaks it seemed logical to argue that the antibodies had been generated by infection with a closely related virus of low pathogenicity. This virus, circulating inconspicuously, had effectively immunised the rabbits against lethal infection (Chasey *et al.* 1997).

Stéphane Marchandeau and his colleagues found that similar, non-pathogenic viruses were almost certainly present in wild rabbits in France too. In their case, however, things were even more puzzling. Although the majority of rabbits had died when RHD first swept through the wild rabbits at the Chevreloup Arboretum near Paris, ELISA tests showed that serum from the cadavers contained antibodies like those formed against RHDV. But how could this be so? The disease usually kills within 2–3 days and it takes at least 4 days for even tiny traces of antibody to show up in rabbits that survive infection. Stéphane sought Antonio Lavazza's help to sort out the problem. Together they reasoned that the antibodies in the rabbits could not have been produced by the lethal infection; instead, they must have been the result of an