“KILLER VIRUS HITS DOGS” headlined newspapers in 1978 reporting the outbreak of a pandemic of unknown origin. It spread rapidly across the globe and infected approximately 90 percent of the global dog population. Puppies were most affected, showing severe vomiting and diarrhea that killed many of them.

What caused such devastation? Thanks to Cornell researchers, we now know this disease as Canine Parvovirus (CPV), and dogs are now routinely vaccinated against it. In fact, Cornell scientists identified the causative viral agent and were able to develop a vaccine to prevent further spread of the epidemic within two years.

CPV emerged from a well-known cat virus called Feline Panleukopenia Virus (FPV) and only required a few mutations in the genome to enable the virus to infect dogs instead of cats, which begs the question: how does a mutation in the viral genome suddenly enable a virus to cross species barriers and infect a new host?

Virologist Dr. Colin Ross Parrish and his team are now getting to the heart of these questions at the James A. Baker Institute for Animal Health at Cornell University. His research team focuses on three viruses that originally exclusively infected other animals and then gained the ability to infect dogs: CPV and two different canine influenza viruses (CIV H3N8 and H3N2). Parrish and his team use these viruses as a model to study the evolution, emergence and mechanisms of viruses mutating to jump species.

We asked Dr. Parrish about his research, and where he sees it going in the future:

Looking back to the parvovirus outbreak 1978, what was particularly memorable for you?

I was in New Zealand at the time having just graduated from College at Massey University, and was working in a virus research and diagnostics laboratory. In a nearby colony of dogs several litters of puppies became infected by CPV during that period, and some of those developed myocarditis – a common disease manifestation when puppies are infected in the first few days of life. Puppies were often infected when they were very young during the first wave of the epidemic, as the bitches did not have maternal immunity to transfer to the puppies.

Why do you still study parvovirus? What do you hope to gain from studying canine parvovirus and canine influenza?

We still seek to gain new information about the viruses that will allow the development of more effective methods for preventing or treating these diseases in dogs, cats, and other animals.
CPV is still one of the few recent examples of a virus mutating and gaining the ability to infect a new host animal, and in this case to cause a pandemic of disease that continues until today. We have learned a lot about how CPV arose, and our ability to study the processes has increase with the development of new technologies. Much of the newer information also allows us to develop models for anticipating such emergence events in other viruses in the future, so that we can develop methods and procedures that would allow us to head those viruses off before they do emerge or become widespread.

As well as being causes of important respiratory diseases in dogs, the H3N8 and H3N2 canine influenza viruses are also examples of viruses transferring into new hosts and causing epidemics in that host. Since there are concerns about the emergence of influenza viruses in humans that could kill many thousands or even millions of people, having more information about how these two host transfers occurred which result in epidemics in dogs will allow us to better anticipate or prevent influenza (or other) viruses from transferring from other animals to humans to initiate outbreaks or epidemics – or pandemics.

You study host virus interactions in the laboratory and integrate these findings with evolutionary studies from virus evolution in the wild. Based on those studies, which factors determine the ability of a virus to infect a new host?

The basic events that have been defined include the different levels of barrier that may occur in the new host to block or slow down infection. Those may be associated with the receptor, natural physical barriers such as mucus or skin that block infection or transmission, innate immune responses, and also the necessity for the first hosts to be in contact with other susceptible animals to allow onward transmission, then for the population to be connected enough to allow an outbreak to occur.

Do you think viral host jumping events could potentially be prevented?

We aim to better predict which out of all the thousands of viruses that infect animals may be a threat to humans or other animals, and to identify new emerging viruses before they become widespread. We could then implement a variety of control strategies in their animal populations, prevent those viruses from being exposed to humans, stop outbreaks very early before the viruses become widespread, or allow the rapid development of new vaccines or vaccine strategies.

What was the biggest surprise in your results?

That the canine influenza viruses do not seem to become adapted to dogs to the level that they can spread more widely among household dog so that infection is only maintained in large animal shelters and kennels.

–by Simon Frueh, Immunology and Infectious Diseases Graduate Student

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