

“Emergence of Epidemic Viruses in New hosts – Canine Parvoviruses and Canine Influenza Viruses”

By

Dr. Colin Parrish

**Baker Institute for Animal Health
Department of Microbiology and Immunology
College of Veterinary Medicine
Cornell University**

Date: 18 May 2015 (Monday)

Time: 11:00 am – 12:30 pm

**Venue: G5-217 (near Lift 7)
5/F, Green Zone, Academic 1
City University of Hong Kong
Tat Chee Avenue, Kowloon Tong**

For abstract, please refer to the attachment.

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~ All are Welcome ~

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About the speaker

Colin Parrish is the John M. Olin Professor of Virology, and Director of the Baker Institute for Animal Health and of the Feline Health Center, in the College of Veterinary Medicine. Dr. Parrish is a BSc (Hons.) graduate of Massey University in Palmerston North, New Zealand where he majored in Microbiology and Biochemistry. Dr. Parrish has a Ph.D. in Virology from Cornell Ithaca, and that was followed by postdoctoral studies on flaviviruses at Monash University in Melbourne, Australia. Dr. Parrish has been on the faculty of Cornell's College of Veterinary Medicine since 1988. Among his academic roles has been the organization and oversight of the Cornell DVM/Ph.D. program from 2002 to 2008. He has trained 18 Ph.D. students, and served as a advisor or supervisor for more than 45 students, postdoctoral fellows and other trainees. He has published over 150 papers, reviews and book chapters on various topics in virology and infectious diseases.

Dr. Parrish's research focuses on virology, virus structure and the evolution of new host ranges by viruses. The model systems that his laboratory investigates are the canine parvovirus, which is a cat virus that transferred into dogs in the 1970s to cause a global pandemic of disease, and canine influenza virus, which is a horse virus which around 2000 transferred into dogs to cause an epidemic of canine disease that is still continuing.

The work involves studying how viruses bind specific receptors to infect the cells of particular animals, how they enter and infect the cells, as well as how the viruses evolve in nature under different conditions. In the studies his laboratory uses a wide variety of approaches to their studies, ranging from molecular virology, structural biology, cell biology and evolutionary analysis. His laboratory has also been actively examining how animals protect themselves against infection through antibody responses, how and where the antibodies bind to the viral proteins, and how changes in the viruses and the antibodies influence the protection of the host.

Abstract

Our research concerns two different viruses that have jumped from one host to another. Those include the canine parvovirus and canine influenza virus. In each case the virus jumped from a different animal host to create a new epidemic virus in its new host – dogs. Two canine influenza viruses (CIV) have emerged in the past 15 years - the H3N8 CIV that emerged from equine influenza virus (EIV) around 2000 in the USA, and the H3N2 CIV that emerged from an avian influenza virus in China and Korea around 2005. Those viruses are both still circulating in dogs, and occasionally spill over to infect other animals, including cats. The CIV and EIV are very similar in many of their properties, but the CIV is restricted in infection of equine tracheal explant tissues. We have examined the genetic changes in the H3N8 CIV in the USA and compared those with the sequences of the EIV. There are changes in all gene segments, and we have used genetic analysis of the viruses to examine some of those for their host-specific effects. Those include changes in the HA within and around the sialic acid binding site – but it appears that the two viruses have specificities for the same sialic acids. We are examining the distribution of the different modified forms of sialic acids in dogs and horses, in particular the 4-O-acetylated forms that block activities of some viral NA enzymes, and show that those are highly expressed in equine respiratory tissues. Other changes in the NS1 and PA-X proteins are being examined for their host-specific effects.

In the case of the canine parvovirus (CPV), the virus emerged in 1978, and caused a pandemic of disease in dogs worldwide. We have shown that the virus evolved from a virus of another host, related to the feline panleukopenia virus. The canine host range of CPV was associated with a small number of changes in the viral capsid that controlled the interaction of the virus with the canine transferrin receptor (TfR). In this case the canine TfR differed from the receptor from the other hosts in that it contained a glycosylation in the apical domain, where the virus bound. The CPV proved to have a broad host range, and related viruses have now been isolated from many different hosts, and in some cases those viruses had mutations that were host-specific. We are now working to understand the host-specific variation in the viruses, and how that influences the interactions with the TfR.