



# THE WINN FELINE FOUNDATION

For the Health and Well-Being of All Cats

1805 Atlantic Ave., PO Box 1005, Manasquan, NJ 08736-0805  
Voice 732-528-9797, ext 31 Fax 732-528-7391 www.WinnFelineHealth.org

## **Genetic Characterization of Feline Calicivirus Associated with Fatal Hemorrhagic Disease**

**Investigator: Melissa Kennedy, DVM, PhD**

University of Tennessee

*Grant #03-009*

Feline calicivirus (FCV), one of the most common causes of upper respiratory tract disease in cats, is highly contagious, often associated with other disease syndromes, and routinely leaves survivors shedding virus for months to years. A virulent hemorrhagic-like disease has recently been associated with FCV that results in significant mortality in cats. This study characterized two FCV isolates, at the genetic level, associated with this hemorrhagic-like disease.

The genome of six FCV isolates were sequenced, five of which had been isolated previously at the College of Veterinary Medicine, University of Tennessee. Two of these had been associated with hemorrhagic disease, and three had been associated with classical signs of FCV. One isolate was obtained from the United States Department of Agriculture (USDA). In addition, genome sequence data of six other FCV isolates were obtained from GenBank for genetic analysis. Published articles provided partial sequence data from three FCV isolates associated with hemorrhagic disease. Five serum samples from cats infected with one of the isolates associated with hemorrhagic disease, a serum sample from the other of the virulent isolates, and a negative control serum were used in serum virus neutralization assays.

Virus neutralization assays were done to determine the relationship of the various FCV isolates – both hemorrhagic-type and classical-type -- to one another. The results suggested that the two virulent isolates share a degree of homology at the neutralizing epitope level, but are distinct from the other isolates.

Comparison of the nucleotide sequences of the entire genome revealed a high homology, with two exceptions. Two vaccine viruses appeared to be identical to disease-associated field isolates. Phylogenetic trees based on the complete genome amino acid sequences for all FCV isolates revealed that they belong to the same genotype regardless of geographical origin or clinical signs. Similar comparisons of the capsid protein gene also pointed to a single genotype.

Researchers concluded that similarities between the two hemorrhagic isolates were no greater than when either virus was compared to any other strain of FCV. This suggests that each FCV isolate has a distinct and unique origin. It is the clustering of the hemorrhagic isolates within a

small but significant genetic region of the capsid gene, however, that leads the researchers to opine that the disease phenotype may have a genetic basis.

**For further reading:**

Carter MJ, Milton ID, Turner PC, et al: 1992, Identification and sequence determination of the capsid protein gene of feline calicivirus. *Arch Virol* 122:P223-235.

Dawson S, McArdle F, Bennett M, et al.: 1993, Typing of feline calicivirus isolates from different clinical groups by virus neutralization tests. *Vet Rec* 133:13-17.

Pedersen, NC, Elliott JB, Glasgow A, et al.: 2000, An isolated epizootic of hemorrhagic-like fever in cats caused by a novel and highly virulent strain of feline calicivirus. *Vet Microbiol* 73:281-300.

Schorr-Evans EM, Poland A, Johnson WE, Pedersen NC: 2003, An epizootic of highly virulent feline calicivirus disease in a hospital setting in New England. *J Feline Med Surgery* 5:217-226.

Prepared by: Betty White, ©2008