



WINN FELINE FOUNDATION

For the Health and Well-being of All Cats

390 Amwell Road, Suite 402, Hillsborough, NJ 08844
908-359-1184 • Fax 908-359-7619 • www.winnfelinehealth.org

Molecular Evaluation of the Feline Alpha Tropomyosin Gene in Norwegian Forest, Sphynx, Siberian, Ragdoll and Maine Coon Cats with Familial Hypertrophic Cardiomyopathy

Grant 08-014

Molecular Evaluation of the Feline Myosin Binding Protein C Gene in Siberian Cats with Familial Hypertrophic Cardiomyopathy

Grant 08-015

Investigator: Kathryn Meurs, DVM, PhD, DACVIM

Washington State University

Feline hypertrophic cardiomyopathy (HCM) is the most common cause of heart disease in cats. Cats with HCM may live a normal lifespan, but unfortunately, many are at risk of serious effects of the disease, such as congestive heart failure, arterial thrombosis or even sudden death. There is no cure for the disease, and current treatments are only able to control clinical signs rather than treat the primary disease process.

HCM has long been known as familial in many breeds of cat. However, specific causative genes have only been identified in two breeds, the Maine Coon and the Ragdoll. While the mutations in both breeds are in the myosin binding protein C gene (MYBPC), they are separate mutations. In humans with familial HCM, over 450 causative mutations in 20 different genes have been identified. This degree of genetic heterogeneity is also likely to occur in the domestic cat.

Dr. Meurs has collected pedigrees and genetic samples from cats with familial HCM, including Norwegian Forest Cats, Sphynx, and Siberian cats, as well as from a small number of affected Maine Coon and Ragdoll cats that do not have their known breed mutations. The samples were screened for causative mutations in eight of the most commonly reported genes known to contain mutations causing familial HCM in humans (cardiac troponin I, troponin T, MYBPC, cardiac essential myosin light chain, cardiac regulatory myosin light chain, alpha tropomyosin, actin, and beta myosin heavy chain). Using PCR based sequencing, the coding and splice site regions of the genes were compared for nucleotide changes among HCM affected cats, the published DNA sequences, and control cats (those without HCM).

Unfortunately, no further causative mutations for HCM were identified in the genes screened using these samples. As well, the two known MYBPC mutations were not found in Norwegian Forest cats, Sphynx, or Siberian cats with HCM. The cats evaluated in this study may have familial HCM caused by a mutation in one of the other genes known to cause HCM in humans, or they could have a mutation in a gene that has not yet been associated with HCM. Evaluation of additional cardiac genes will be necessary to identify more genetic causes of feline HCM.

Reference:

Meurs KM, Norgard MM, Kuan M et al: Analysis of 8 sarcomeric candidate genes for feline hypertrophic cardiomyopathy mutations in cats with hypertrophic cardiomyopathy, *J Vet Intern Med* 23:840-3, 2009.

Prepared by: Susan Little, DVM, DABVP (Feline)

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