



ABCF MESSENGER

Official Newsletter of the American Boxer Charitable Foundation, Inc.
Virginia Zurflied, Editor

ABCF 2008 OFFICERS

President
William Trusedale, DVM

Founder
Vice President, Operations
John T. Connolly

Founder
Bruce Korson

Vice President
Robert Conrad, DVM, PhD

Secretary
Virginia Zurflied

Treasurer
Sharon Fosseen

Legal Counsel
Sharon Steckler

COMMITTEES

AKC/Canine Health
Committee
William Truesdale, DVM

Health & Research Committee
Joyce Campbell, DVM
Chairperson

Annual Auction Committee
Bliss Bancroft, Chairperson

Membership Committee
Bobbi Compton, Chairperson

Webmaster
Judy Voran
bjvoran@gmail.com

Newsletter Editor
Virginia Zurflied
4506 Sleepy Hollow Lane
Plant City, FL 33565
vzboxers@aol.com

Boxer Cardiomyopathy Bombshell!

The following is an excerpt from an announcement published today on the Washington State University web site. Our own Dr Kate Meurs has discovered the mutant gene responsible for Boxer Cardiomyopathy, and has developed a test for the gene!!!

To read the entire article, click on the link printed at the end of the excerpt, and stay tuned for more breaking news! Dr Meurs will be speaking at the upcoming ABC Specialty in Ft Mitchell, Kentucky, and we will be there taking notes!

Virginia Zurflied, editor

Vet cardiologist discovers gene for heart disease

Monday, Apr. 27, 2009

PULLMAN — WSU veterinary cardiologist **Kathryn M. Meurs** discovered a mutant gene in the Boxer breed that causes a type of heart disease that can be fatal in animals and humans.

Well known in the Boxer breed community, the disease is called Boxer cardiomyopathy. The more formal term is arrhythmogenic right ventricular cardiomyopathy or ARVC.

This is same type of heart disease that caused the sudden death of 1950s college and pro football great Joe Campanella at age 36, as he played handball with the new head coach of the Baltimore Colts, Don Shula.

In Boxers, the disease can be fatal and frequently occurs when the animals exercise or become excited. Occasionally, they perish from the disease while at rest, too.

“Dr. Meurs’ discovery of both the gene and its location is a tremendous achievement in the cardiology of humans and animals,” said **Bryan Slinker**, dean of WSU’s College of Veterinary Medicine, and a recognized cardiac disease researcher. “This achievement not only helps Boxer

breeders avoid this disease but it also provides an extraordinary advancement to the study of human heart diseases resulting from electrical conduction defects and the resulting heart muscle changes that occur.”

A copy of the Washington State article appears below:

Vet cardiologist discovers gene for heart disease

Monday, Apr. 27, 2009

Contact: Charlie Powell, WSU College of Veterinary Medicine, 509-335-7073, cpowell@vetmed.wsu.edu

PULLMAN — WSU veterinary cardiologist **Kathryn M. Meurs** discovered a mutant gene in the Boxer breed that causes a type of heart disease that can be fatal in animals and humans.

Well known in the Boxer breed community, the disease is called Boxer cardiomyopathy. The more formal term is arrhythmogenic right ventricular cardiomyopathy or ARVC.

This is same type of heart disease that caused the sudden death of 1950s college and pro football great Joe Campanella at age 36, as he played handball with the new head coach of the Baltimore Colts, Don Shula.

In Boxers, the disease can be fatal and frequently occurs when the animals exercise or become excited. Occasionally, they perish from the disease while at rest, too.

“Dr. Meurs’ discovery of both the gene and its location is a tremendous achievement in the cardiology of humans and animals,” said **Bryan Slinker**, dean of WSU’s College of Veterinary Medicine, and a recognized cardiac disease researcher. “This achievement not only helps Boxer breeders avoid this disease but it also provides an extraordinary advancement to the study of human heart diseases resulting from electrical conduction defects and the resulting heart muscle changes that occur.”

The disease is well known in Boxers because the breed has the highest incidence of this form of heart disease. ARVC is also known to be an inherited disease and breeders

sometimes avoided breeding to certain lines of Boxers yet were never completely sure if those lines had an increased risk of disease. Additionally, the disease tends to vary in severity between different dogs; key indications that the disease had a dominant genetic origin.

Meurs began looking at the disease as an extension of her work with inherited heart disease in cats and dogs. This work is somewhat similar to her work with breeds of cats that also suffer heart disease and for which she has also discovered mutant genes. Her lab developed a molecular probe for these mutations so that cat owners now have a mechanism for screening for the disease and breeding away from it.

Using an extremely powerful gene screening mechanism based on a massive computer chip at the Broad Institute at MIT with investigators Kerstin Lindblad-Toh and Evan Mauceli, Meurs looked at thousands of regions of boxer dogs' DNA simultaneously. The samples were collected with participation by members of the American Boxer Club and the American Boxer Charitable Foundation and were segregated into groups of dogs with the disease and those with no evidence for the disease.

Once computer analysis identified a specific region of interest, Meurs' lab evaluated thousands of DNA sequences in affected and unaffected dogs and identified a gene mutation in a gene that normally codes for the production of a key cellular adhesive protein. Subsequent studies done by WSU veterinary cardiologist, Sunshine Lahmers, demonstrated that the cellular adhesive proteins were located at the junction between cells in the heart.

Theoretically, the conduction defect is in some way responsible for a rapid, irregular heart beat that does not pump blood efficiently. When blood is not pumped efficiently, there may not be enough circulation maintained in the brain and other organs. This can lead to fainting episodes or even sudden cardiac death.

Over time, the right, lower chamber of the heart, called the right ventricle, begins to be infiltrated by a fibrous fatty tissue and often has decreased contractile ability. This change in the heart's tissues can spread to the wall between the heart

chambers and even the left ventricle.

The structural changes that result in functional impairment is the hallmark sign seen when a post mortem examination is performed on the animal's heart. Under the microscope, the normal muscle appears solid and dense. The affected heart muscle tissue is riddled with holes where the fibrous fatty tissue has infiltrated stretching it like unorganized lace.

Meurs' laboratory is now near obtaining a patent on her discovery and is perfecting a genetic testing probe for the gene mutation that will be used as a clinical screening device. Shortly, Boxer owners will have the ability to take a simple cheek swab of their dog and know whether or not it carries the mutant gene. Cost of the screening is expected to be about \$70 and available within the next 1-2 months.

"In many cases, after the disease is diagnosed it can be managed with medication for a long enough period of time in a dog's life that other diseases such as cancer will be the cause of death," said Meurs. "The medications are not very expensive and there are generic forms available, too. Average monthly costs are probably less than \$100."

Meurs said that, with her lab's service, Boxer owners and breeders will be able to identify dogs with the mutant gene and are likely to breed away from the disease.