UNIVERSITY OF COPENHAGEN

Faculty of Health and Medical Sciences

Hjertesygdom hos Kat
- Update 2024

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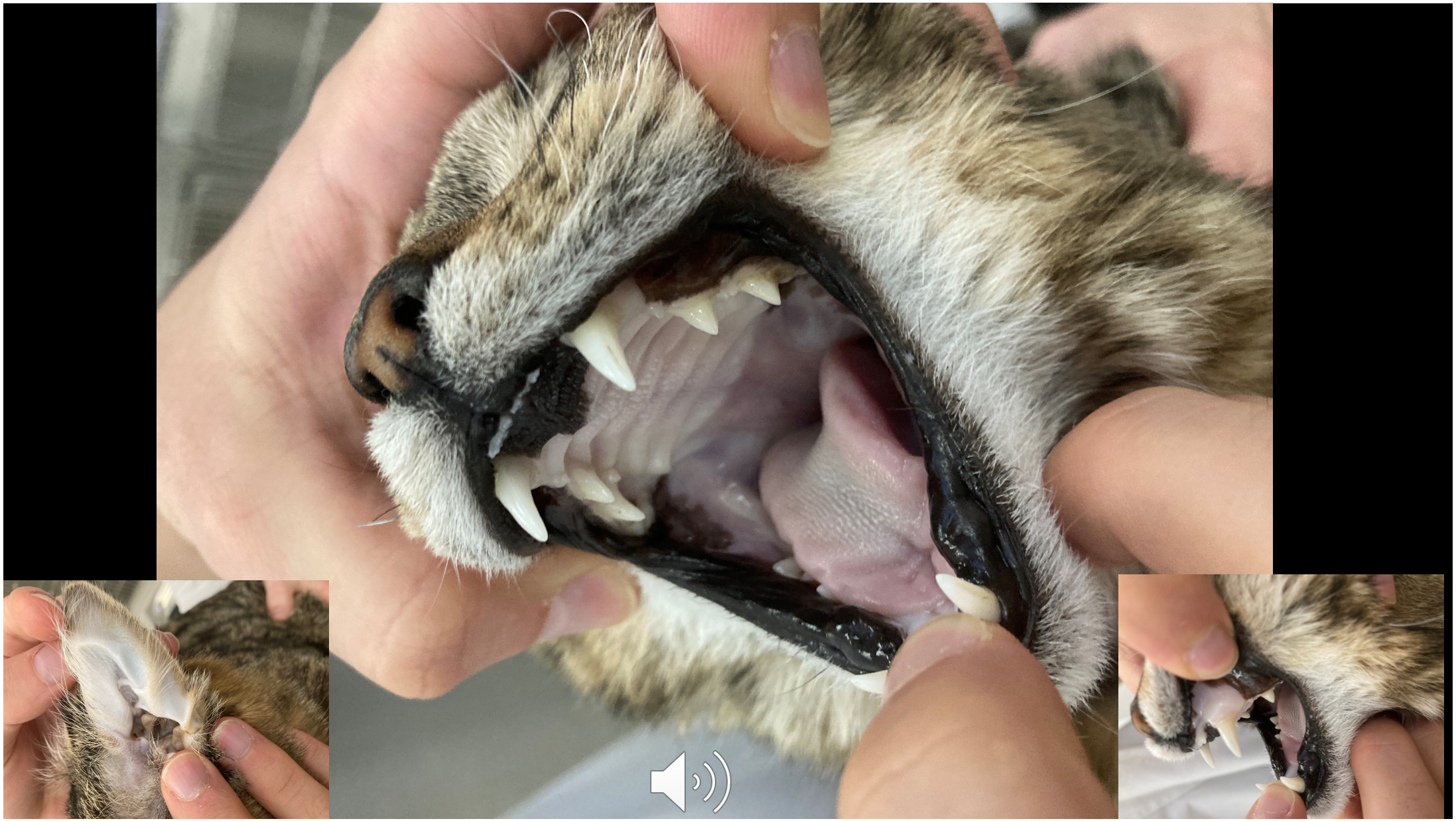
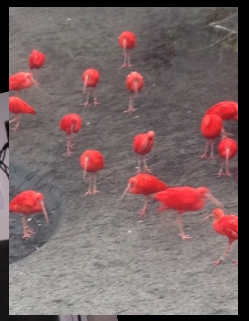
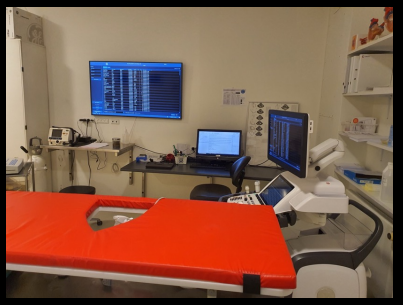
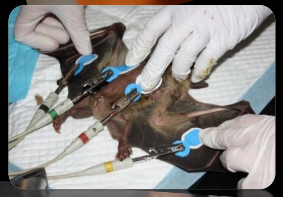
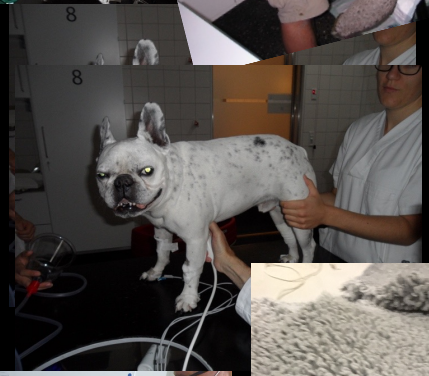


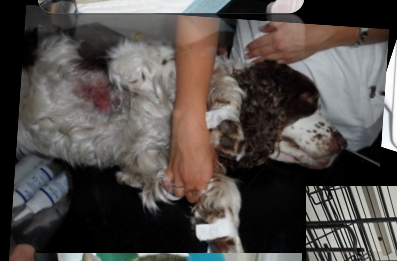
Hjerteklinikken

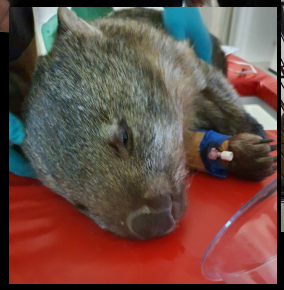


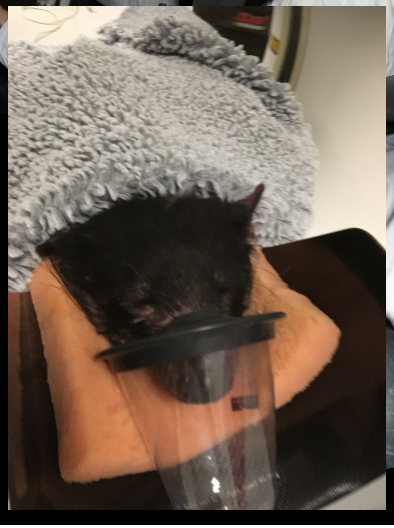
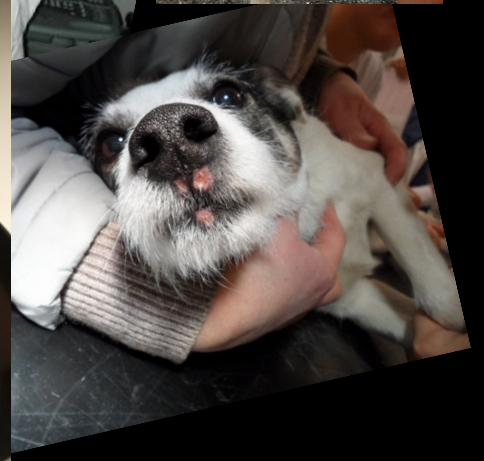








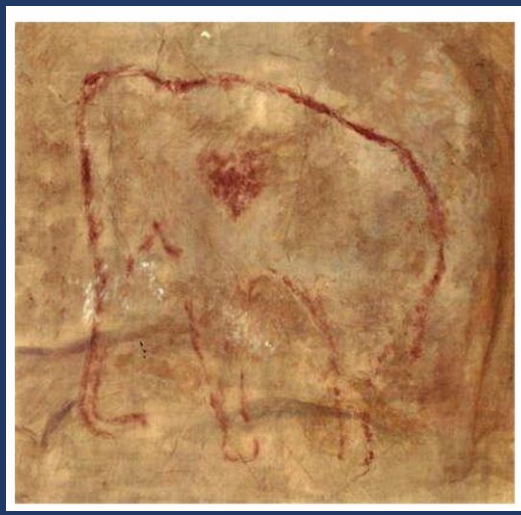
The Heart as a Symbol

ca. 510-490 BC. Cyrenian coin with a silphium seed imprinted in it

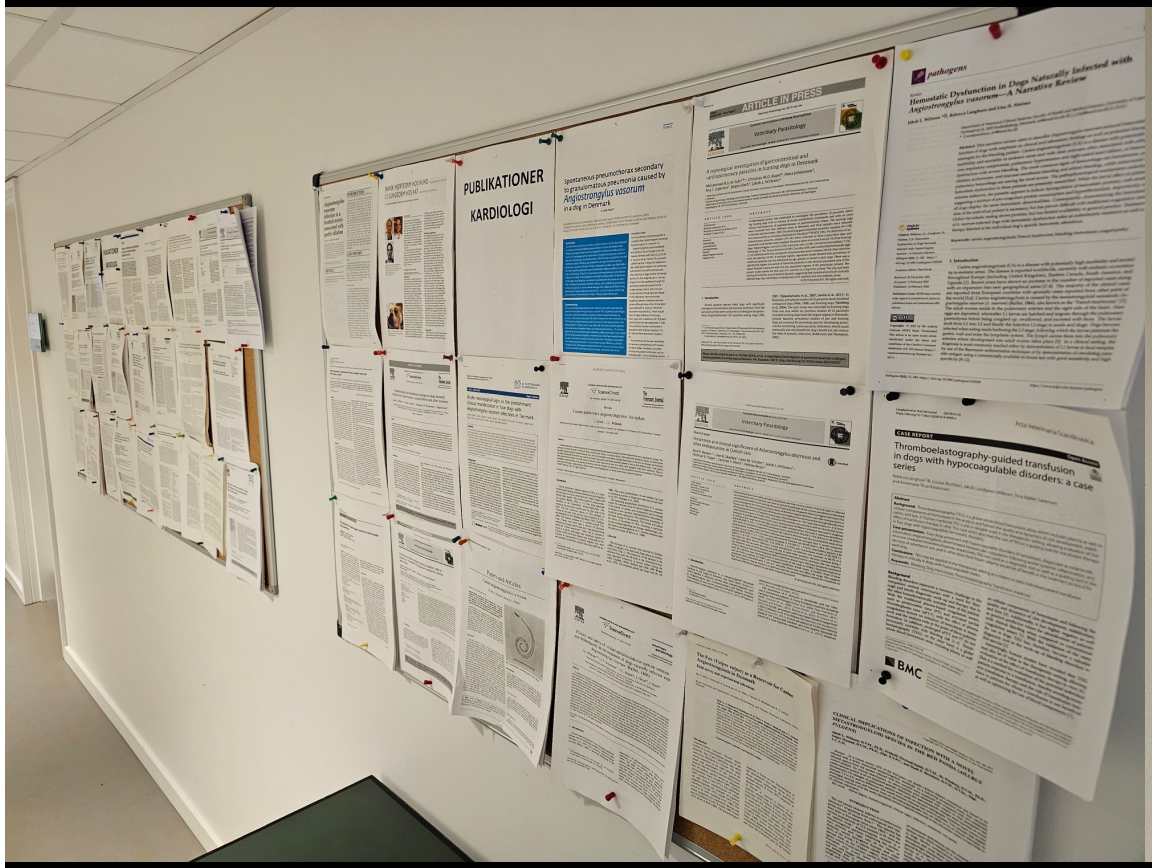
Red Heart Emoji

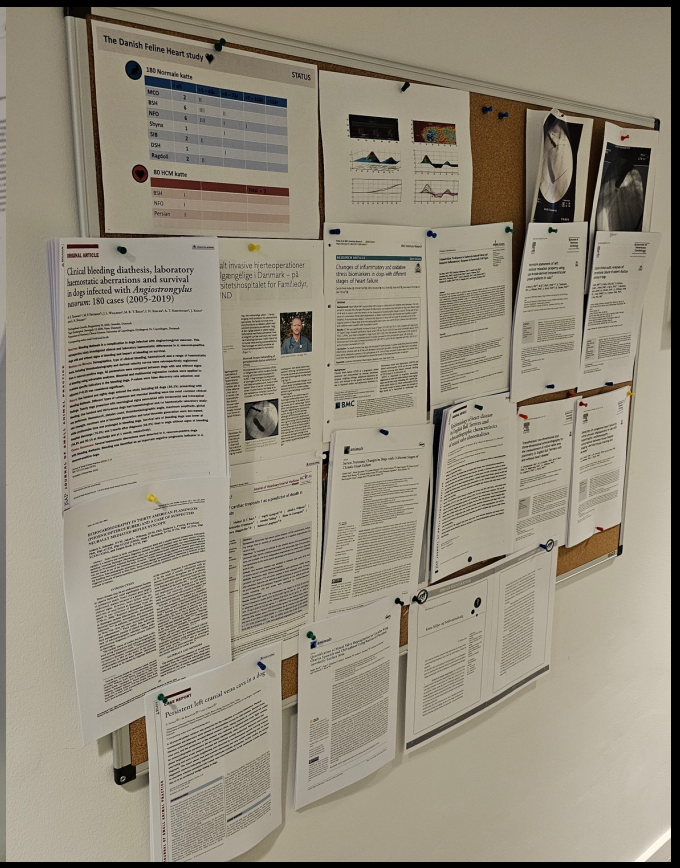
"The Heart Offering," 1338-1344

Milton Glaser, I Love New York, 1977. Trademarked logo, New York State Department of Economic Development, New York, New York



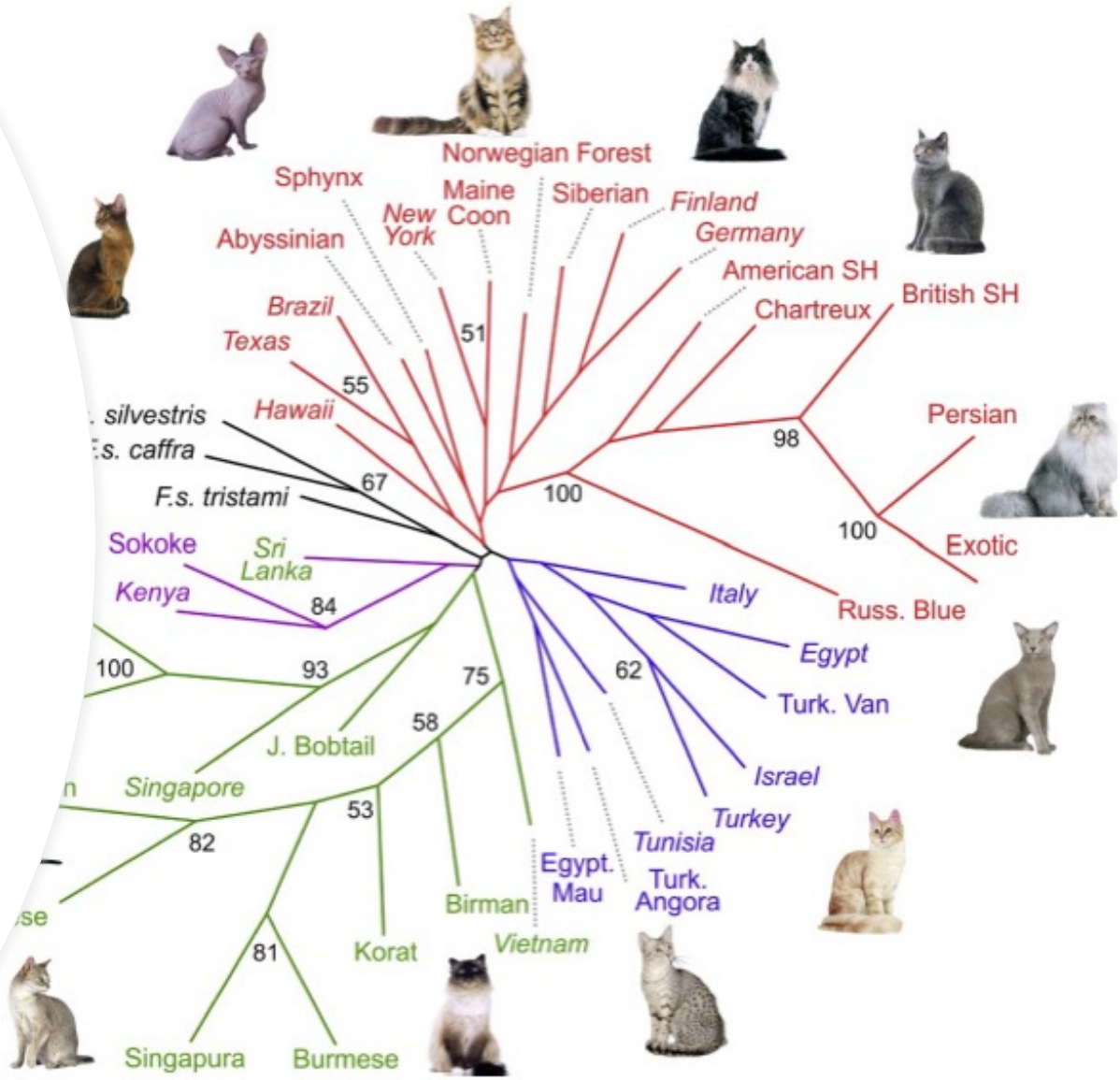
https://ideas.ted.com/how-did-the-human-heart-become-associated-with-love-and-how-did-it-turn-into-the-shape-we-know-today/


PUBLIKATIONER
KARDIOLOGI
ARTICLE IN PRESS
Veterinary Parasitology
pathogens
Hemostatic Dysfunction in Dogs Naturally Infected with Angiostrongylus vasorum—A Narrative Review
Spontaneous pneumothorax secondary to granulomatous pneumonia caused by Angiostrongylus vasorum in a dog in Denmark
Thromboelastography-guided transfusion in dogs with hypocoagulable disorders: a case series
CASE REPORT
BMC


The Danish Feline Heart study
180 Normale katte
STATUS
80 HCM katte
ORIGINAL ARTICLE
Clinical bleeding diathesis, laboratory haemostatic aberrations and survival in dogs infected with Angiostrongylus vasorum: 180 cases (2005-2019)
BMC
CASE REPORT
Peritoneal-pleural cranial vena cava shunt

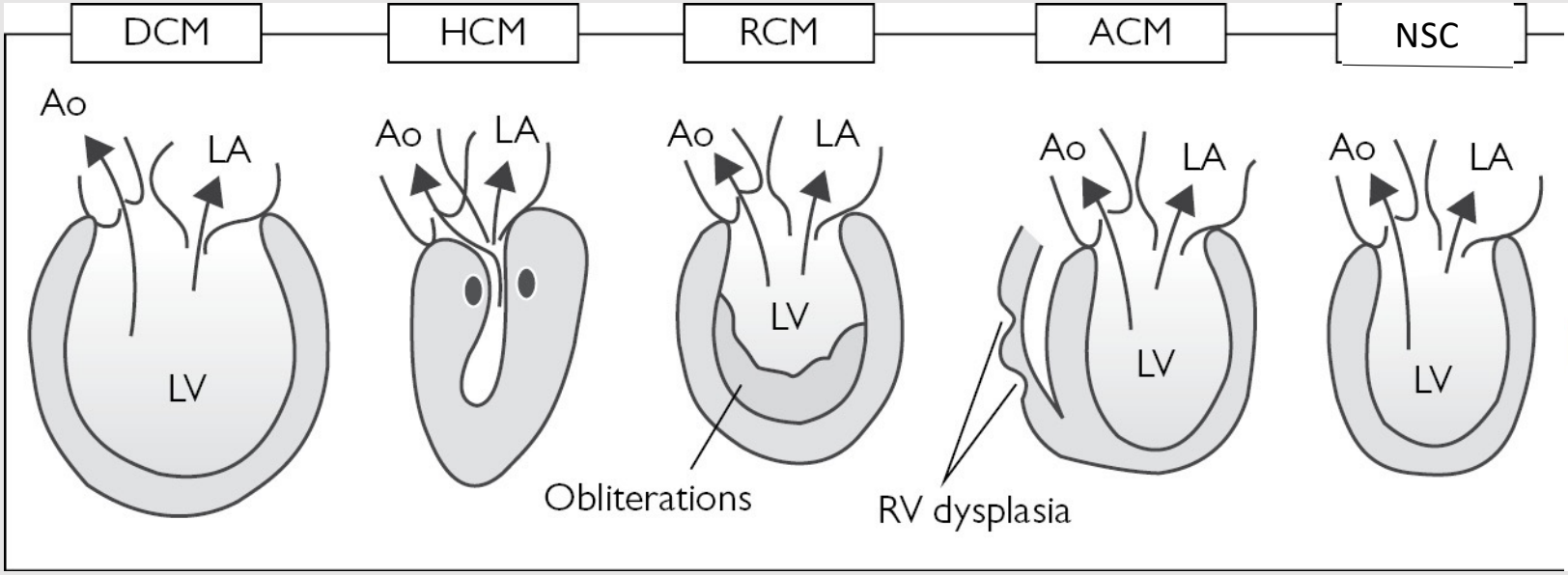
Phylogenetic Tree of Cat Breeds

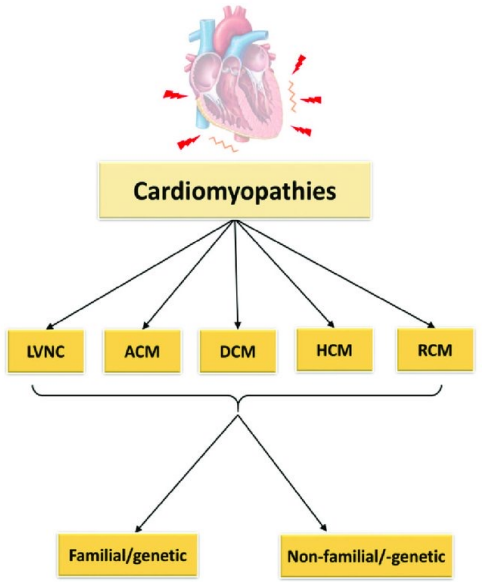
- Genomics 2008 Jan;91(1):12-21.
- doi: 10.1016/j.ygeno.2007.10.009. Epub 2007 Dec 3.



A Heart-Sick Cat

Cardiomyopathies - Classifications





Primary and Secondary Cardiomyopathies

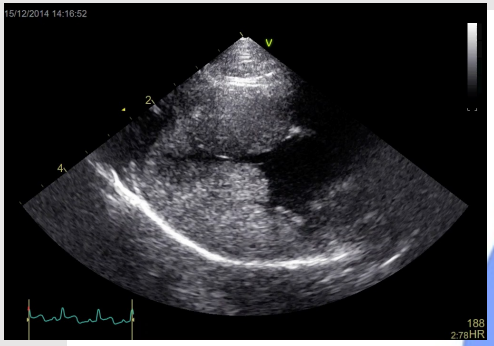
https://www.hospitalsenhedmidt.dk/patientvejledninger/hjertesygdomme/familiaer-dilateret-kardiomyopati-dcm/

Journal of Veterinary Internal Medicine

CONSENSUS STATEMENT | Open Access

ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats

Virginia Luis Fuentes, Jonathan Abbott, Valérie Chetboul, Etienne Côté, Philip R. Fox, Jens Häggström, Mark D. Kittleson, Karsten Schober, Joshua A. Stern



Journal of Veterinary Internal Medicine

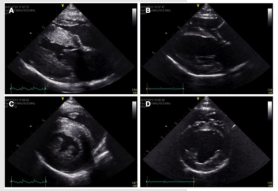
Standard Article | Open Access

Transient Myocardial Thickening in Cats Associated with Heart Failure

J. Novo Matos, N. Pereira, T. Glaus, L. Wilkie, K. Borgeat, J. Loureiro ... See all authors

First published: 15 December 2017 | https://doi.org/10.1111/jvim.14897 | Citations: 10

This study was not supported by any grant.
This work was done at the Royal Veterinary College, Hatfield, Hertfordshire, UK, Vetsuisse Faculty, Zurich, Switzerland, Highcroft Veterinary Referrals and North Downs Specialist Referrals.
The results of this study were presented as an abstract at the 26th ECVIM-CA Annual Congress 2016, Gothenburg, Sweden.



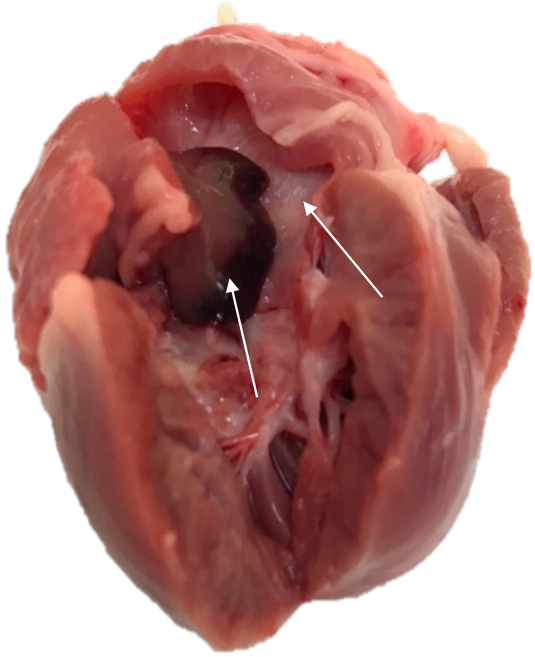
Journal of Veterinary Cardiology

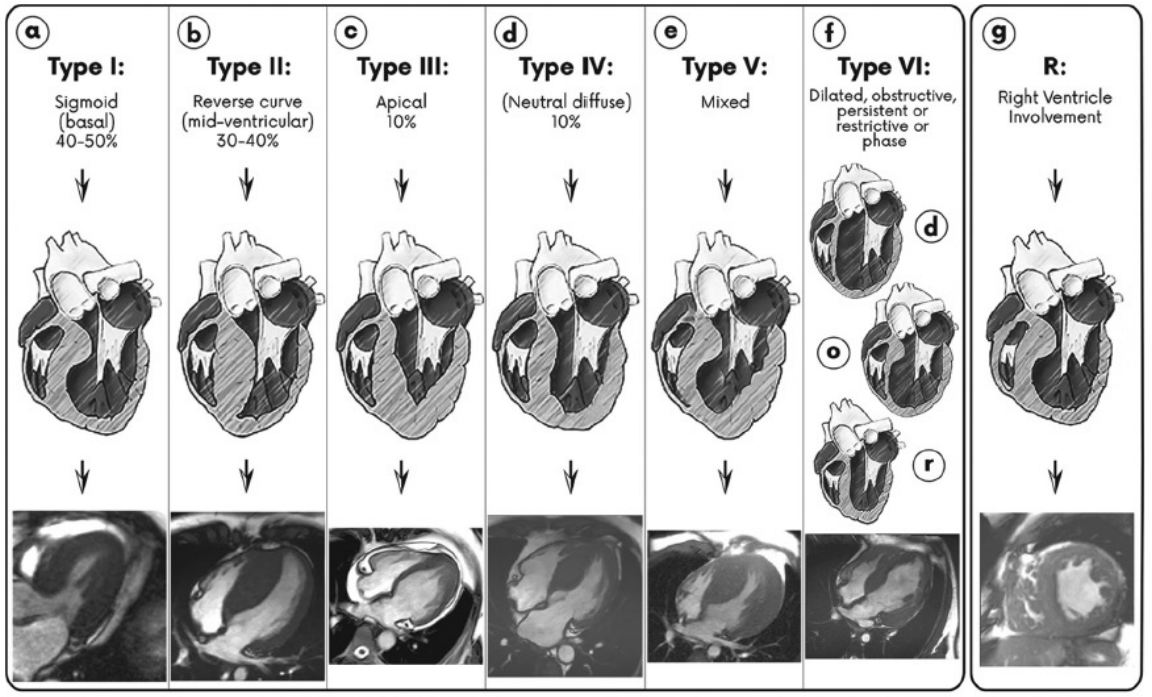
Volume 19, Issue 1, February 2017, Pages 14-23

Echocardiographic assessment of feline false tendons and their relationship with focal thickening of the left ventricle

O.A. Wolf DVM, M. Ingrund Dr. rer. nat, G. Wess DVM, PhD, Dr habil

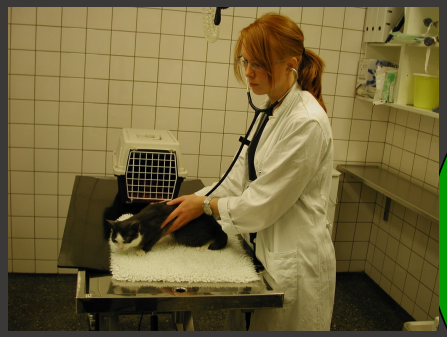
- HCM is a primary disease of the cardiac muscle that is characterized by a hypertrophied and nondilated left ventricle, normal or enhanced contractile function, and impaired ventricular relaxation in the absence of other cardiac or systemic diseases





Arch Cardiol Mex. 2022;92(3)

Hjertesygdom vs. hjertesvigt



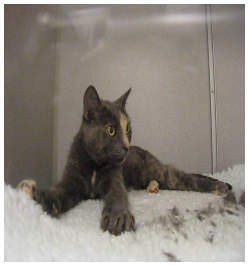
1 ud af 7 katte har en hjertelidelse

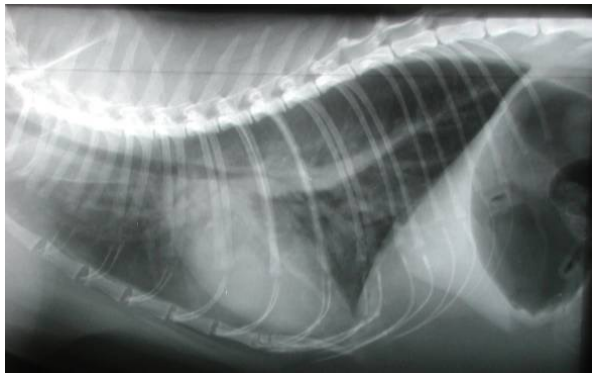
Medfødte <5%?

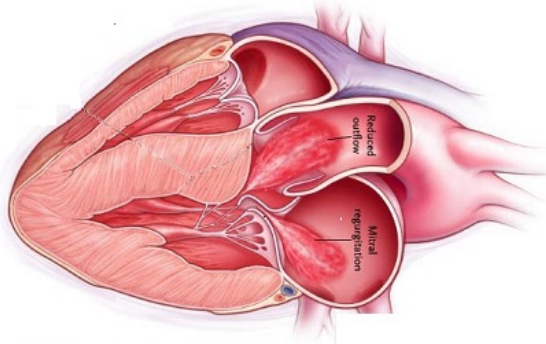
Erhvervede 95%?

Complications

- Congestive Heart Failure
- Dynamic Obstruction of the Left Ventricular Outflow Tract (SAM)
- Thromboembolism
- Arrhythmias
  - Atrial Fibrillation
  - Ventricular Tachycardia
- Sudden Death
- (Anorexia/Weight Loss)

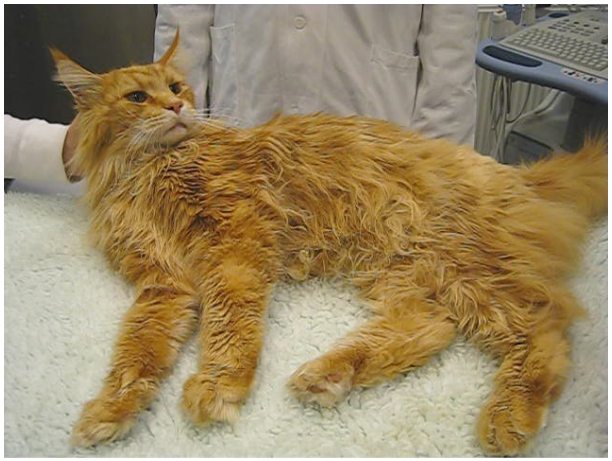


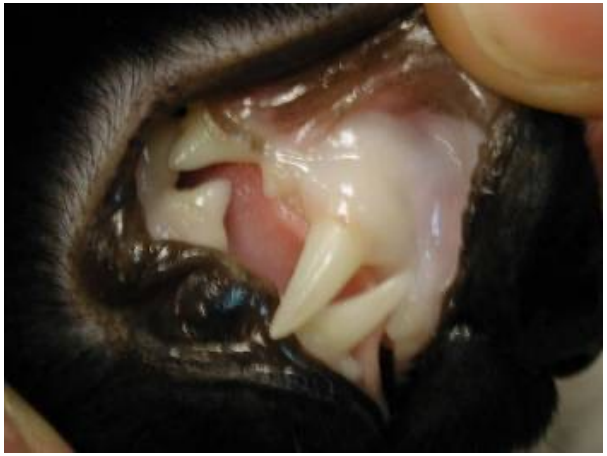




Clinical Findings

- Any age
- Murmur
  - Harsh systolic ejection murmur across entire precordium → apex & heart base
  - MR: separate murmur - severity of MR related to degree of outflow obstruction
- Gallop sounds
- Dyspnea
- Depression and anorexia
  - Weight loss!?
- Hind limb paralysis
- Syncope
- Sudden death

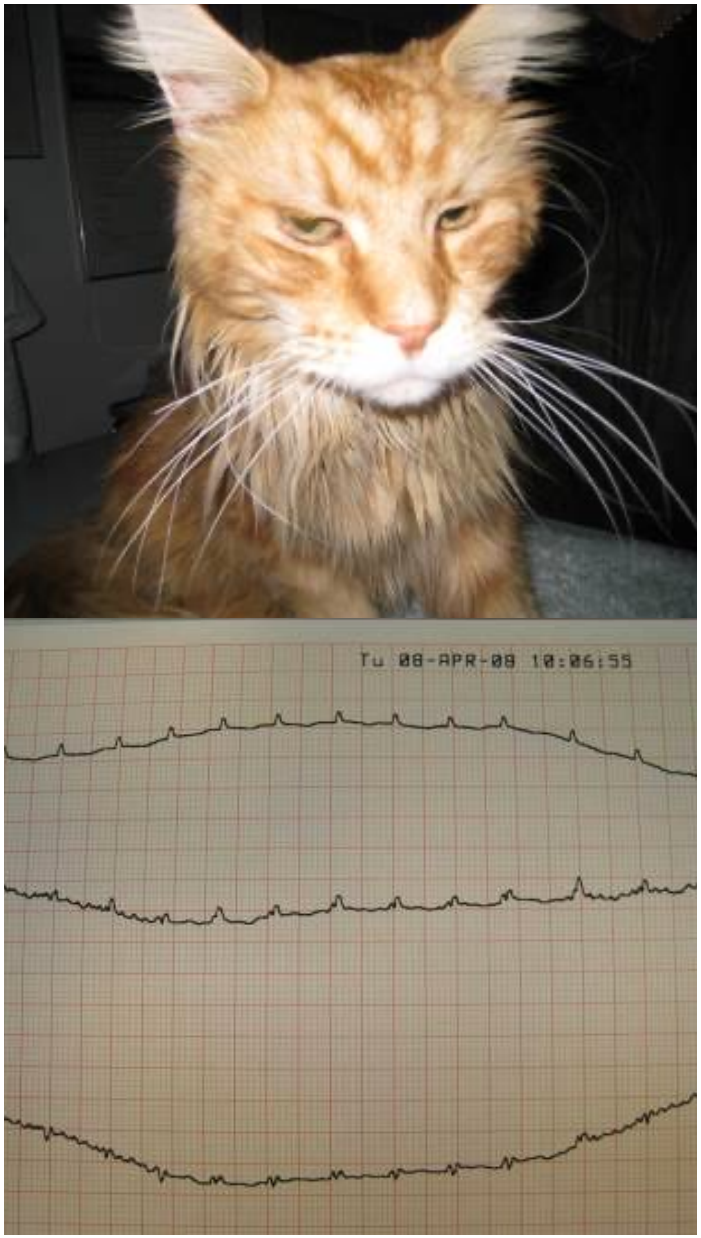




Maine Coon with Severe HCM

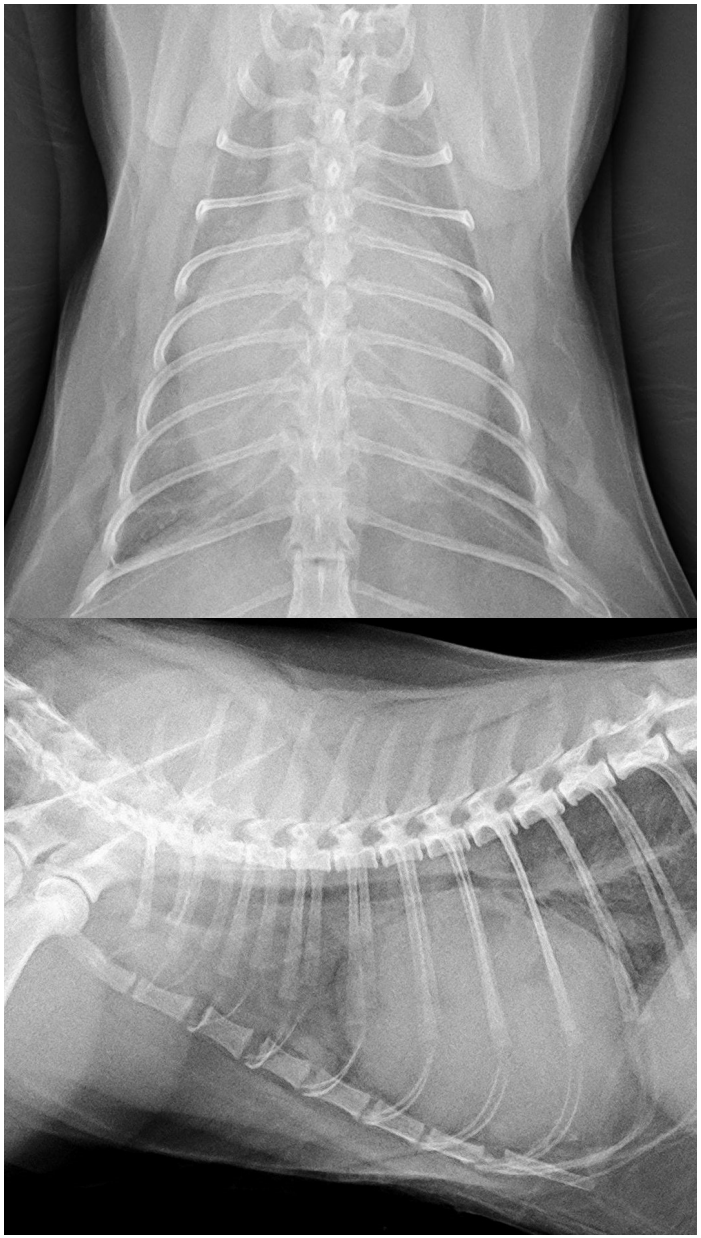
”Al Pacino” intact male, 4 years old

- Atrial Fibrillation
- Congestive Heart Failure
- Thromboembolic Disease



TREATMENT OF CONGESTIVE HEART FAILURE IN CATS

- Treatment
  - Oxygen
  - Butorphanol
  - Furosemide
  - Thoracocentesis



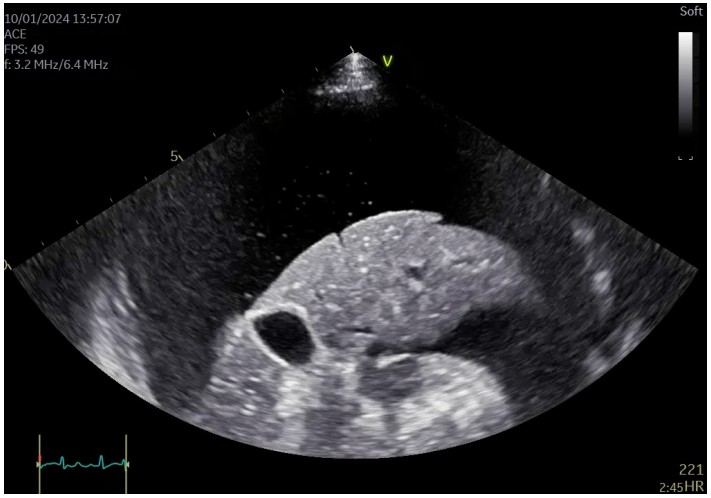
CASE – L…

Signalement:

- Norwegian Forrest cat
- 4,8 years old
- Neutered female
- 7,3 kg

- Presentation

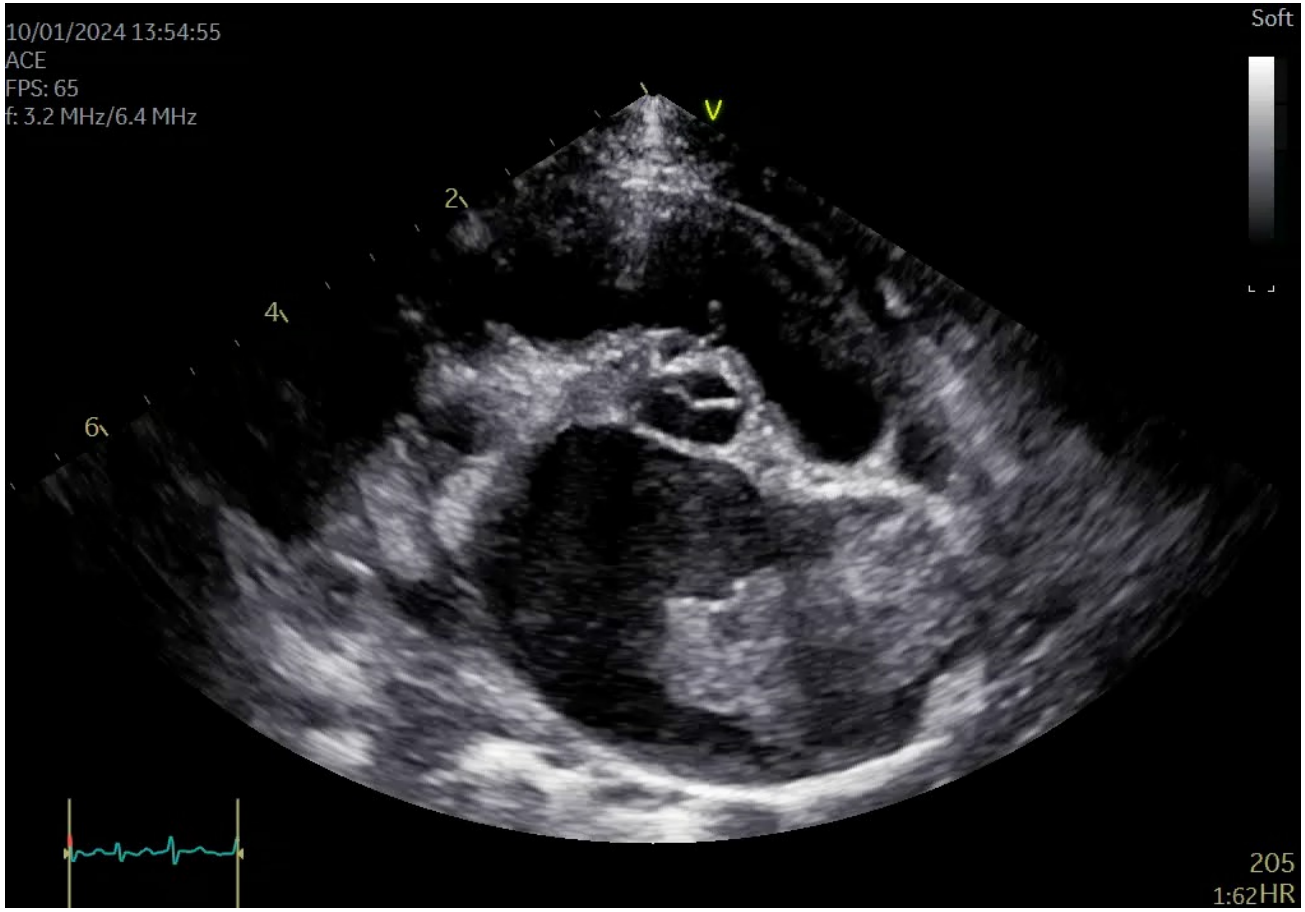
Gained weight → 0,5-1 kg the last month

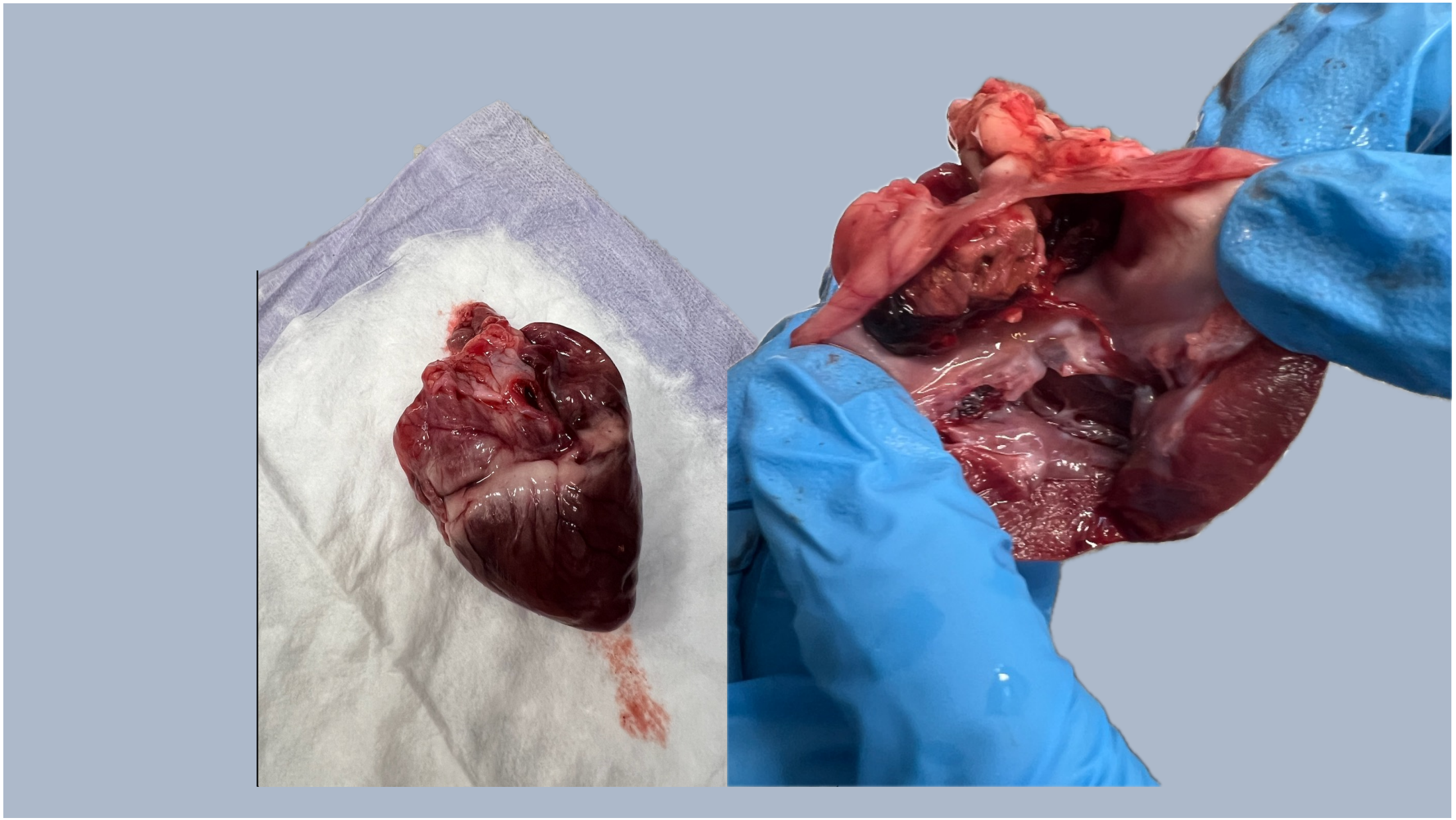


Initial Problem List

1. Weight Gain
    1. A recent increase in weight despite diet-prescription food
2. Increased Abdominal Size
    1. Possible causes: fluid accumulation, organ enlargement, or fat deposition
3. Hypertrophic Cardiomyopathy (HCM)
    1. A chronic condition requiring ongoing treatment with Clopidogrel and Atenolol
    2. Arrhythmia

ECHO SAX – heart base (RS)





Alternative treatment for cats with HCM

- Nattokinase: 3-4 capsules bid (starting with 15 Capsules) 2.000 FU (fibrinolytic units)
  Nattokinase is an enzyme derived from natto, a traditional Japanese fermented soybean dish

How it works	
Fibrinolytic Activity	Dissolves fibrin and hereby clots
Blood thinning	By promoting fibrinolysis, it can also help maintain healthy blood flow and circulation
Antihypertensive effects	Antihypertensive effects by promoting vasodilation and enhancing blood flow
nti-inflammatory properties	Inhibits the expression of certain pro-inflammatory mediators and hereby damps the inflammatory response

Alternative treatment for cats with HCM

- Rutin: 1 tablet BID (450 mg)
  Rutin is a bioflavonoid, a type of plant pigment found in certain fruits, vegetables, and herbs.

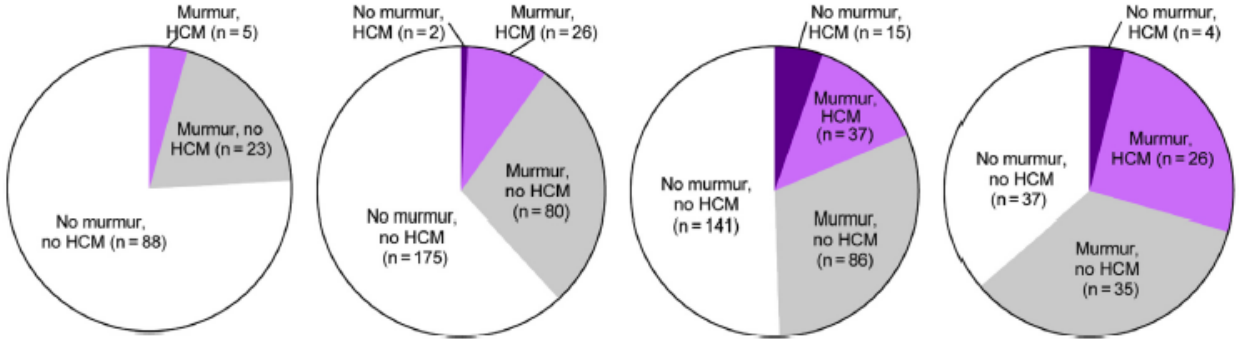
How it works	
Antioxidant activity	Can neutralize harmful free radicals in the body
Anti-inflammatory effects	Inhibiting the production and activity of pro-inflammatory molecules
Vascular health	Strengthening blood vessels and reducing permeability. This prevents leakage of fluid and nutrients into surrounding tissues
Cardiovascular protection	Lowering blood pressure, inhibiting platelet aggregation, diuresis
Anti-allergenic properties	Inhibit the release of histamine and other inflammatory mediatiors involved in allergenic reactions
Skin health	Protects against oxidative damage caused by UV radiation and environmental pollutants

Asymptomatic Hypertrophic Cardiomyopathy: Diagnosis and Therapy

Virginia Luis Fuentes, VetMB, PhD, CertVR, DVC, MRCVS[a,*],
Lois J. Wilkie, BSc, PhD, MRCVS[b]

Vet Clin Small Anim 47 (2017) 1041–1054
http://dx.doi.org/10.1016/j.cvsm.2017.05.002

Table 1
Prevalence of heart murmurs and hypertrophic cardiomyopathy in 780 apparently healthy cats from rehoming centers

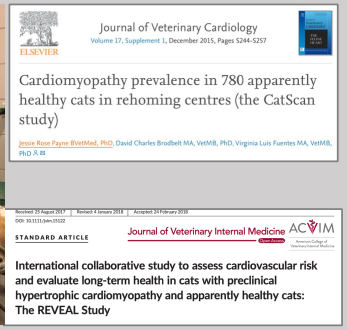


	Juvenile: 6–12 mo (n = 116)	Young Adult: 1–3 y (n = 283)	Adult: 3–9 y (n = 279)	Senior: 9 y or Older (n = 102)
Heart murmur prevalence (%)	24.1	37.5	44.1	59.8
HCM prevalence (%)	4.3	9.9	18.6	29.4

From Payne JR, Brodbelt DC, Luis Fuentes V. Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centres (the CatScan study). J Vet Cardiol 2015;17:S252; with permission.

Substantial morbidity and mortality

- HCM is common with an overall prevalence of 15% (CatScan study)
- 28% cardiac mortality at 10 years. (Reveal study)
  - No difference between HCM and OHCM in time to CHF or ATE



Journal of Veterinary Cardiology

Volume 17, Supplement 1, December 2015, Pages S244-S257

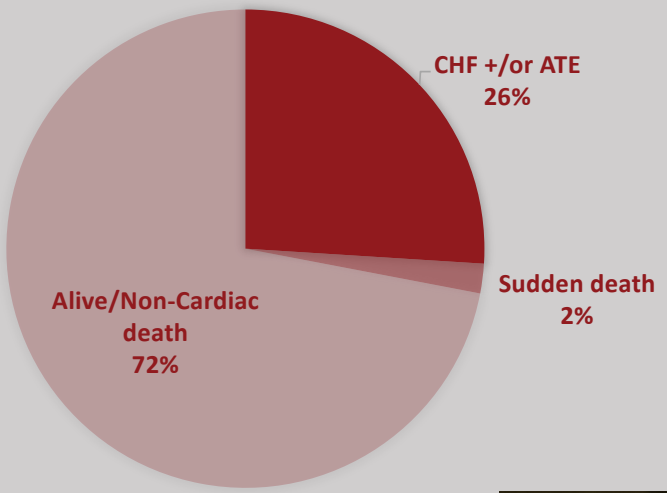
Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centres (the CatScan study)

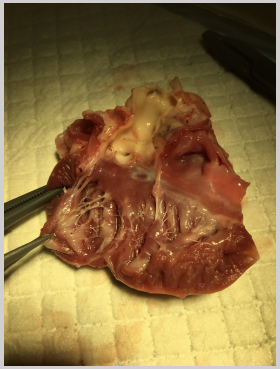
Jessie Rose Payne BVetMed, PhD, David Charles Brodbelt MA, VetMB, PhD, Virginia Luis Fuentes MA, VetMB, PhD

STANDARD ARTICLE

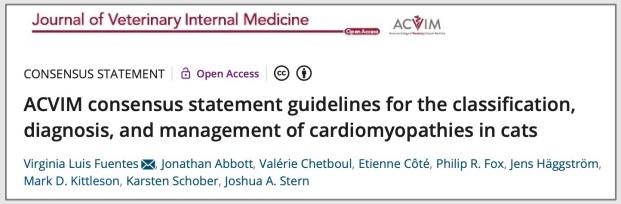
Journal of Veterinary Internal Medicine ACVIM

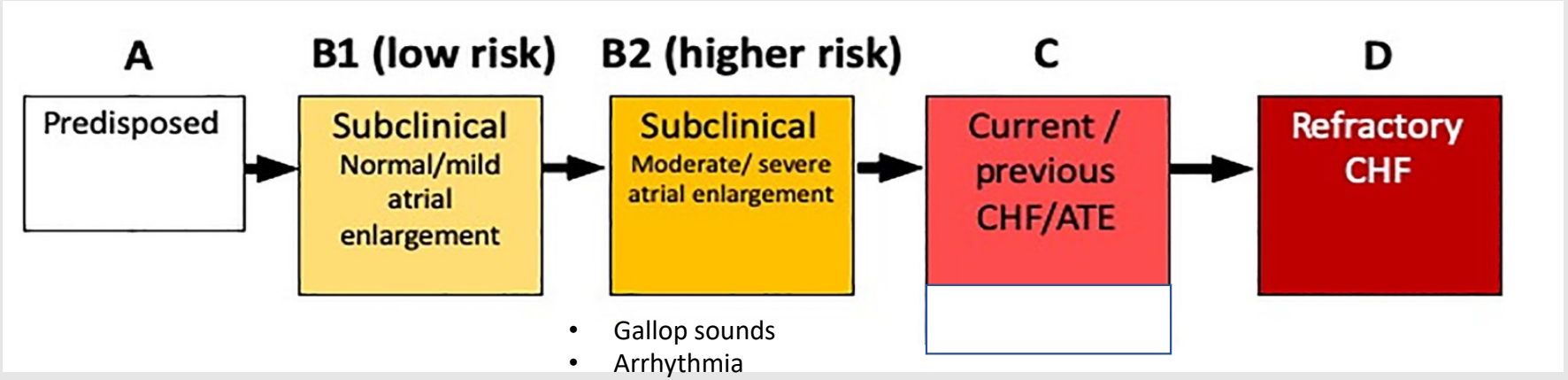
International collaborative study to assess cardiovascular risk and evaluate long-term health in cats with preclinical hypertrophic cardiomyopathy and apparently healthy cats: The REVEAL Study





Staging Feline Cardiomyopathy

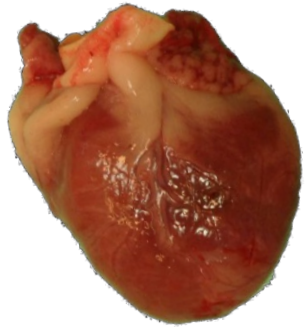


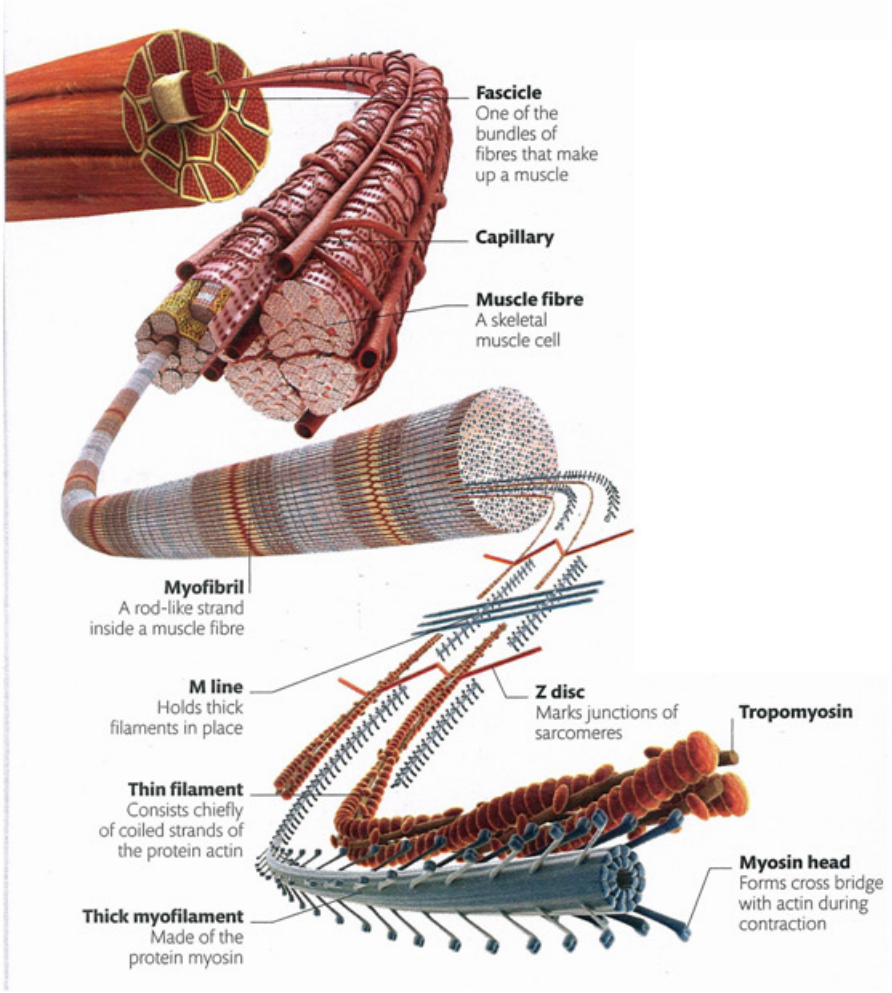


- Gallop sounds
- Arrhythmia
- Poor LA function
- Extreme LV hypertrophy
- LV systolic dysfunction
- ”Smoke” / intracardiac thrombus
- Regional LV wall thinning/hypokinesis

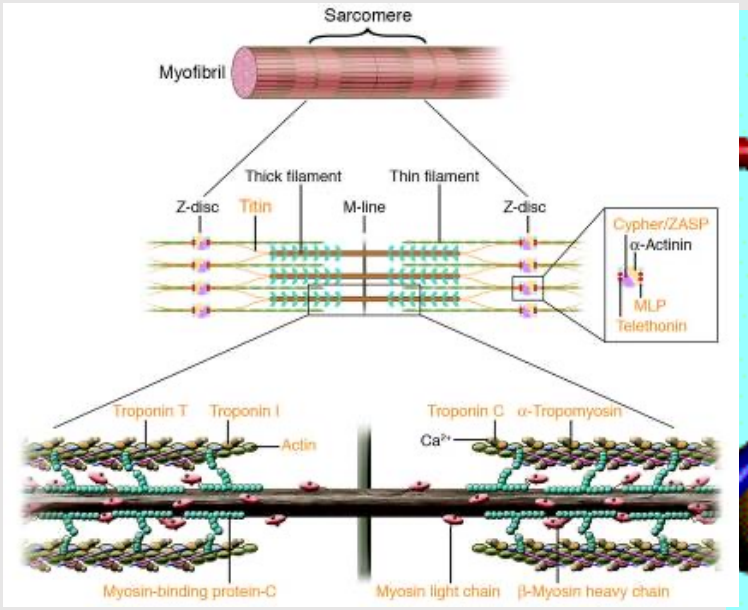
Exploration in the microworld of the myocyte

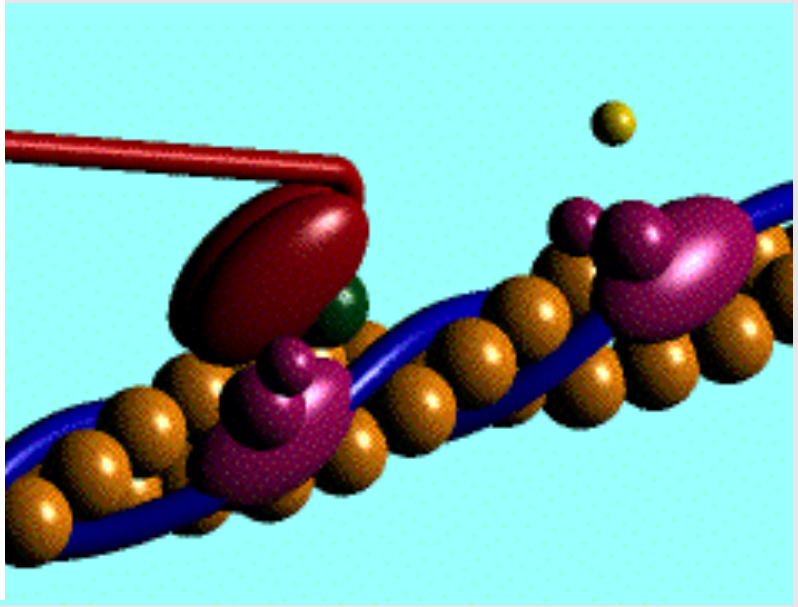
photo: Carli Hækkerup



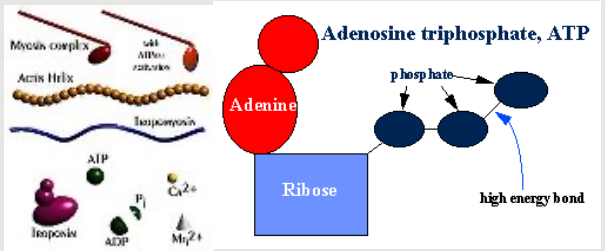


Muscle Contraction





Cardiac myosin-binding protein C,
arrayed transversely along the sarcomere,
binds myosin and, when phosphorylated,
modulates contraction:
- Not essential for cardiac development
  and function, but involved in determining
  efficiency of muscle contraction

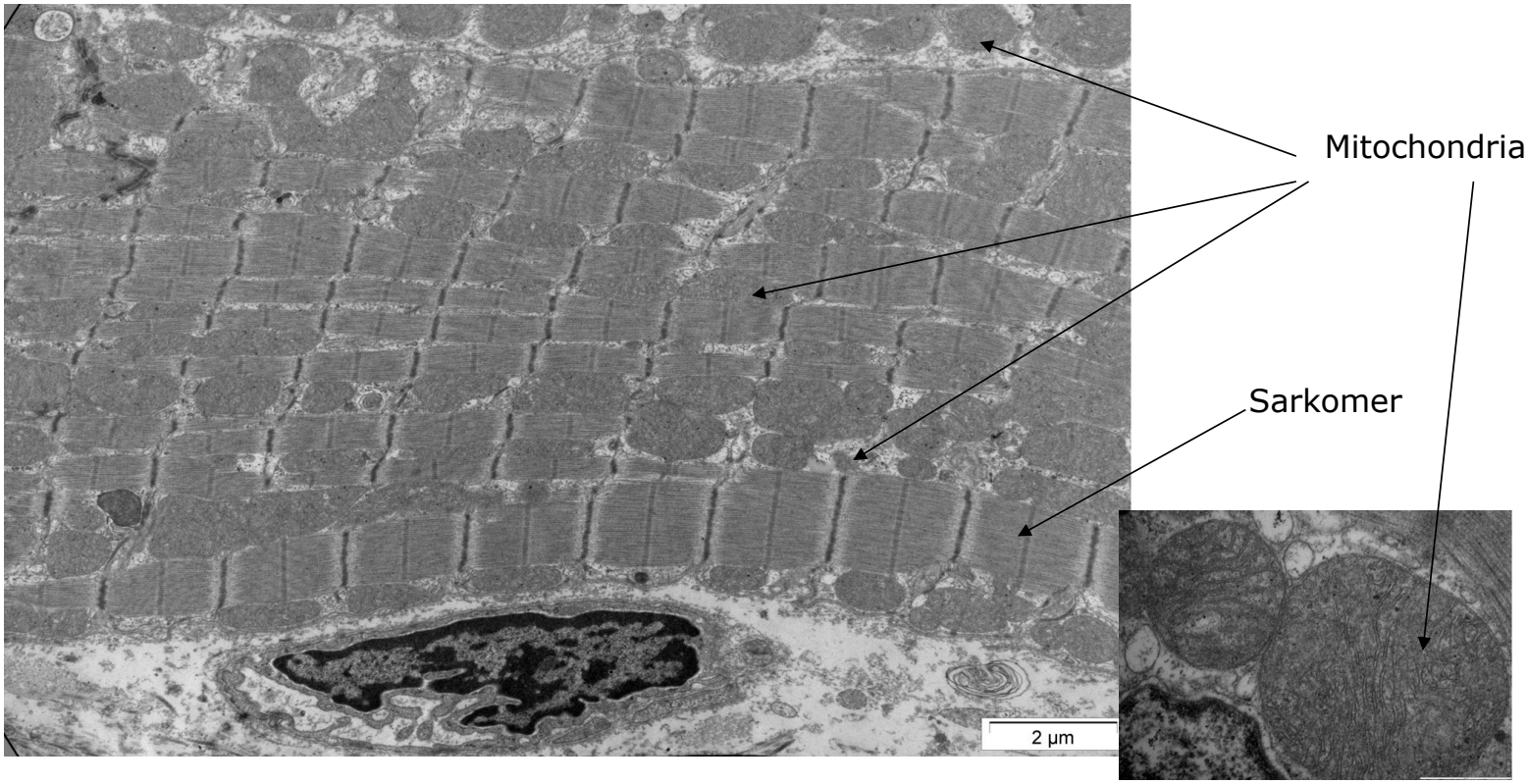


www.banyantree.org/jsale/develop.html

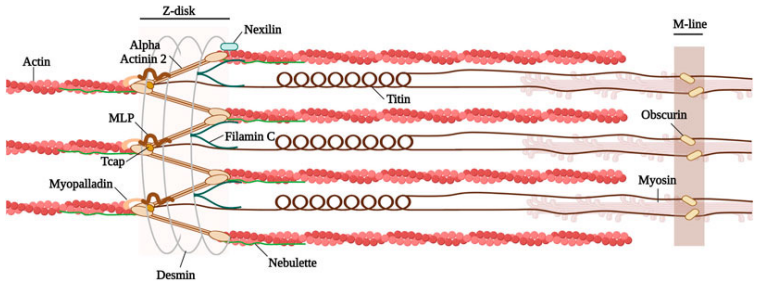
Morita et al. (2005) J. Clin. Invest. **115**:518–526

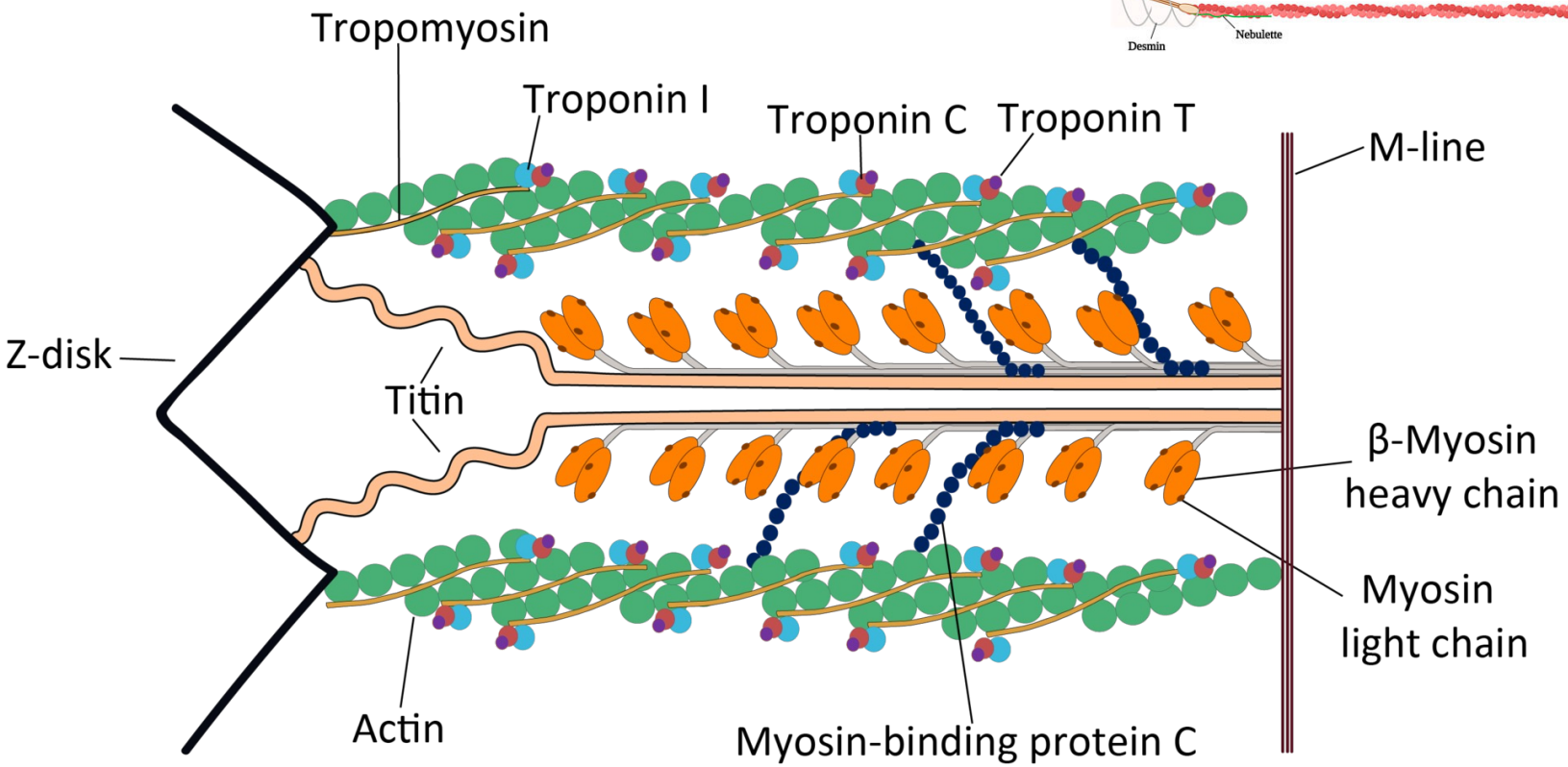
Sarcomeropathy

SEM. Venstre ventrikel, DSH, 6 år



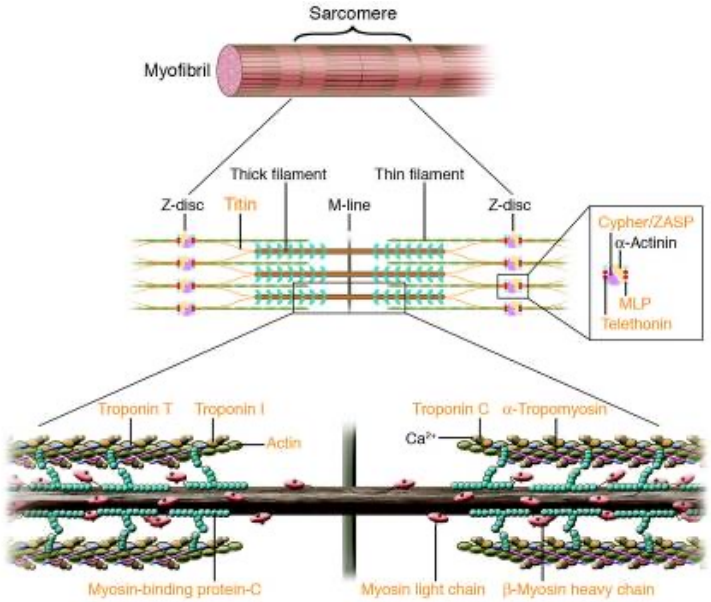
Sarcomer – multiprotein complexes

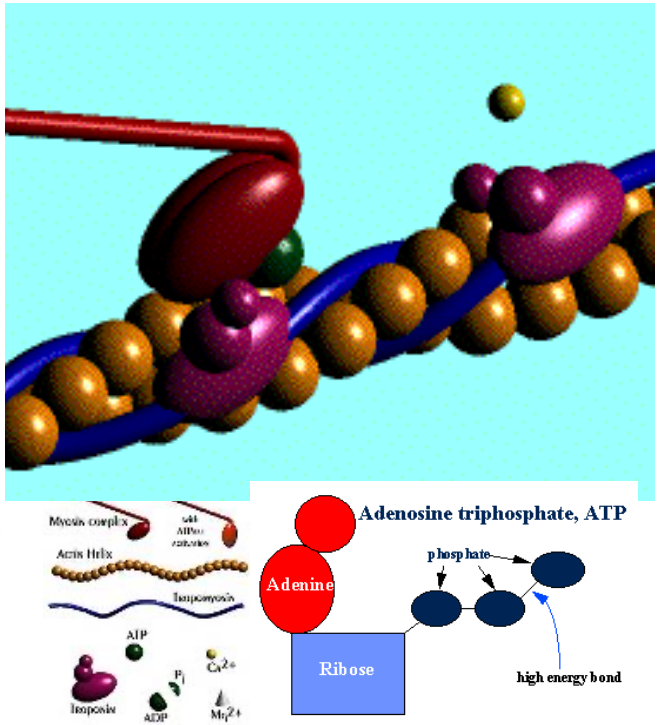




https://www.frontiersin.org/files/Articles/1143858/fphys-14-1143858-HTML/image_m/fphys-14-1143858-g001.jpg

Muscle Contraction





www.banyantree.org/jsale/develop.html

Cardiac myosin-binding protein C, arrayed transversely along the sarcomere, binds myosin and, when phosphorylated, modulates contraction:

- Not essential for cardiac development and function, but involved in determining efficiency of muscle contraction

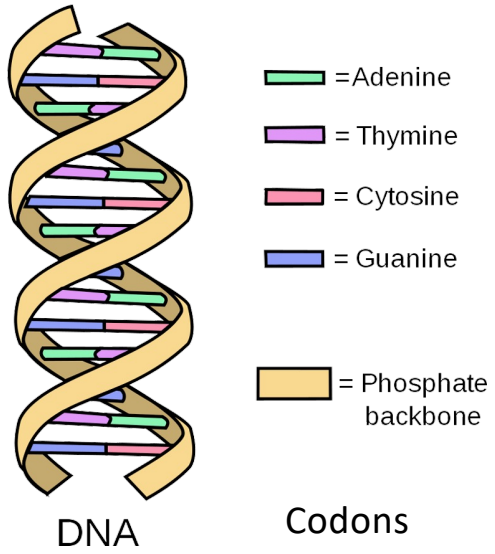
Morita et al. (2005) J. Clin. Invest. **115**:518–526

Cat (*Felix catus/i*)

- Lifespan - up to 24 years
- 38 chromosomes
- > 350 genetical diseases
- Burmese ”Cinnamon”, 4 years old
  - 2007
  - 20285 genes
  - 2.7 x 10^9 base pair
    - A,T,C,G

Our feline friends share 90% of homologous genes with us

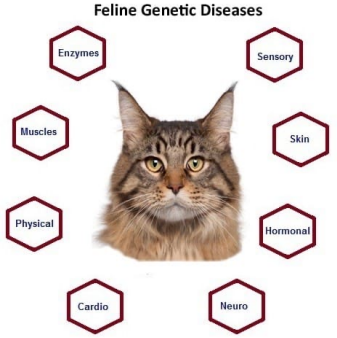
Genetic Disorders = diseases caused by mutations in one or more genes



- Genes: exons, introns, allelles
  - Transcription
  - Translation

Modes of Inheritance

- Autosomal Dominant Inheritance
- Autosomal Recessive Inheritance
- X-Linked Dominant Inheritance
- Y-Linked Inheritance
- Mitochondrial Inheritance
- Multifactorial Inheritance



https://drbillspetnutrition.com/feline-genetic-diseases/

Types of Mutations

Mutations are changes in the DNA sequence. They can occur naturally or be induced by environmental factors. Here's a breakdown of different types:

Point Mutations

Silent Mutation

Missense Mutation Nonsense Mutation

Phenotype =
Genotype x Environment

- Insertions and Deletions (Indels)
- Frameshift Mutations
- Splice Site Mutations
- Repeat Expansion

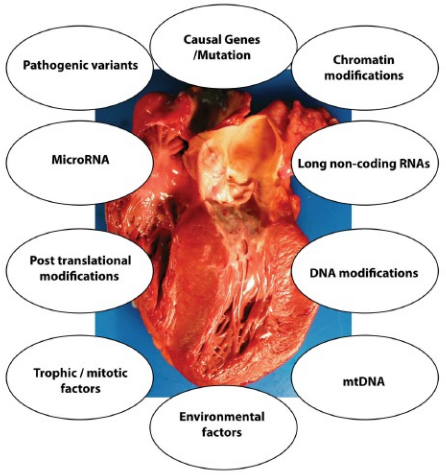


Figure 3. Determinants of phenotype in HCM
Selected factors contributing to expression of cardiac phenotype in HCM are shown. The causal mutation imparts the main effect and several others, such as other pathogenic genetic variants (modifiers), genomics (such as non-coding RNAs), proteomics (such as post-translational modifications), and environmental factors (such as isometric exercises) contributing to expression of the phenotype.

Sarcomeropathies (humans)

A. Established Causal Gene HCM (Large families)				
Gene	**Protein**	**Function**	**Tolerance to variation**	
			Missense (Z score)	**LoF (pLI)**
MYH7	β-Myosin heavy chain	ATPase activity, Force generation	6.54	0.00
MYBPC3	Myosin binding protein-C	Cardiac contraction	0.69	0.00
TNNT2	Cardiac troponin T	Regulator of acto-myosin interaction	1.54	0.01
TNNI3	Cardiac troponin I	Inhibitor of acto-myosin interaction	1.88	0.17
TPM1	α-tropomyosin	Places the troponin complex on cardiac actin	3.42	0.80
ACTC1	Cardiac α-actin	Acto-myosin interaction	5.25	0.95
MYL2	Regulatory myosin light chain	Myosin heavy chain 7 binding protein	0.86	0.02
MYL3	Essential myosin light chain	Myosin heavy chain 7 binding protein	0.75	0.89
CSRP3	Cysteine and glycine-rich protein 3	Muscle LIM protein (MLP), a Z disk protein	-0.66	0.00

B. Likely causal genes for HCM (small families)				
Gene	**Protein**	**Function**	**Tolerance to variation**	
			Missense (Z score)	**LoF (pLI)**
FHL1	Four-and-a-half LIM domains 1	Muscle development and hypertrophy	1.29	0.92
MYOZ2	Myozenin 2 (calsarcin 1)	Z disk protein	0.03	0.02
PLN	Phospholamban	Regulator of sarcoplasmic reticulum calcium	0.57	0.11
TCAP	Tcap (Telethonin)	Titin capping protein	0.45	0.08
TRIM63	Muscle ring finger protein 1	E3 ligase of proteasome ubiquitin system	0.02	0.00
TTN	Titin	Sarcomere function	−5.48	0.00

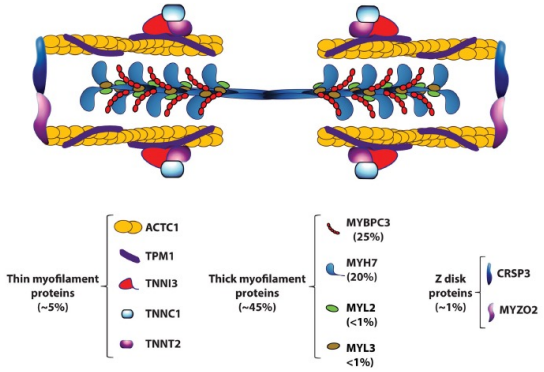


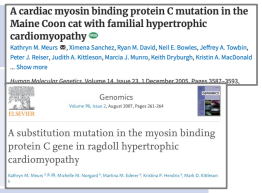
Figure 1. HCM as a disease of sarcomere proteins
A schematic structure of a sarcomere composed of thick and thin filaments and Z discs is depicted along with its protein constituents involved in HCM. Established causal genes for HCM and their population frequencies are listed.

C. Genes associated with HCM				
Gene	**Protein**	**Function**	**Tolerance to variation**	
			Missense (Z score)	**LoF (pLI)**
ACTN2	Actinin, alpha 2	Z disk protein	1.76	1.0
ANKRD1	Ankyrin repeat domain 1	A negative regulator of cardiac genes	−0.01	0.00
CASQ2	Calsequestrin 2	Calcium binding protein	−1.08	0.00
CAV3	Caveolin 3	A caveolae protein	1.19	0.34
JPH2	Junctophilin2	Intracellular calcium signaling	3.93	0.01
LDB3	Lim domain binding 3	Z disk protein	0.32	0.00
MYH6	Myosin heavy chain alpha	Sarcomere protein expressed at low levels in the adult human heart	2.87	0.00
MYLK2	Myosin light chain kinase 2	Phosphorylate myosin light chain 2	0.73	0.22
NEXN	Nexilin	Z disc protein	−1.32	0.00
TNNC1	Cardiac troponin C	Calcium sensitive regulator of myofilament function	2.22	0.51
VCL	Vinculin	Z disk protein	3.11	0.99

The Z score for each gene reflects deviation of the observed variants in the ExAC database from the expected number. A higher positive Z score indicates that the gene is intolerant to variation. Likewise, pLI indicates probability of intolerance to Loss-of-Function (LoF) variants with 1 indicating total intolerance.

Feline HCM – American College of Medical Genetics Guidelines

Protein	Mutation	Breed	Classification
MYBPC3	A31P	MCO	Pathogenic
MYBPC3	R820W	Ragdoll	Pathogenic
Alstrom syndrome protein 1	*p.G3376R* 	Sphynx	Unknown significance
TroponinT2	TNNT2 c.95-108G>A	MCO	Unknown significance
MYH7	E1883K	DSH	Likely pathogenic

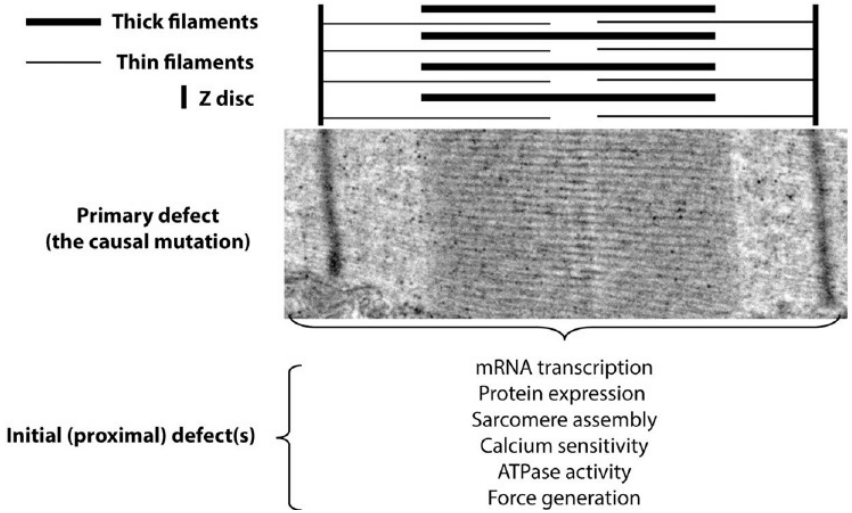

A cardiac myosin binding protein C mutation in the Maine Coon cat with familial hypertrophic cardiomyopathy

Genomics

A substitution mutation in the myosin binding protein C gene in ragdoll hypertrophic cardiomyopathy

A relatively limited number of mutations identified → The genetic architecture of feline HCM susceptibility may follow an oligogenic or polygenic mode of inheritance.

Pathogenesis – Feline HCM



Open Journal of Veterinary Medicine, 2013, 3, 95-103
doi:10.4236/ojvm.2013.32016 Published Online June 2013 (http://www.scirp.org/journal/ojvm)

Scientific Research

Feline Hypertrophic Cardiomyopathy Associated with the p.A31P Mutation in cMyBP-C Is Caused by Production of Mutated cMyBP-C with Reduced Binding to Actin

Mia T. N. Godiksen[1,2], Craig Kinnear[3], Tina Ravnsborg[4], Peter Hojrup[4], Sara Granström[1,2], Inga A. Lauvsen[1], Paula L. Hedley[1,2], Johanna C. Moolman-Smook[5], William J. McKenna[6], Jorgen Koch[1], Michael Christiansen[1*]

[1]Department of Clinical Biochemistry and Immunology, Statens Serum Institut, Copenhagen, Denmark
[2]Department of Small Animals Diseases, Faculty of Life Science, University of Copenhagen, Copenhagen, Denmark
[3]MRC Centre for Molecular and Cellular Biology, University of Stellenbosch, Cape Town, South Africa
[4]Institute of Biochemistry and Molecular Biology, University of Southern Denmark, Odense, Denmark
[5]Institute of Cardiovascular Science, The Heart Hospital, University College London, London, UK
Email: *mac@ssi.dk

Received February 7, 2013; revised March 7, 2013; accepted April 7, 2013

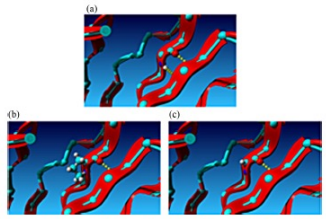


Figure 6. Schematic visualization of the effect of substituting the alanine residue with a proline residue. The proline interrupts the hydrogen bond between two beta strands. (a) Close-up of the alanine residue in the beta strand; (b) Close-up of the C0 domain with the alanine substituted by trans-proline. (c) Close-up of the C0 domain with the alanine substituted by cis-proline.

Am J Physiol Heart Circ Physiol 308: H1237–H1247, 2015.
First published March 13, 2015; doi:10.1152/ajpheart.00727.2014.

Impaired cardiac mitochondrial oxidative phosphorylation and enhanced mitochondrial oxidative stress in feline hypertrophic cardiomyopathy

Liselotte B. Christiansen,[1,2] Flemming Dela,[2] Jørgen Koch,[1] Christina N. Hansen,[2] Pall S. Leifsson,[3] and Takashi Yokota[2]

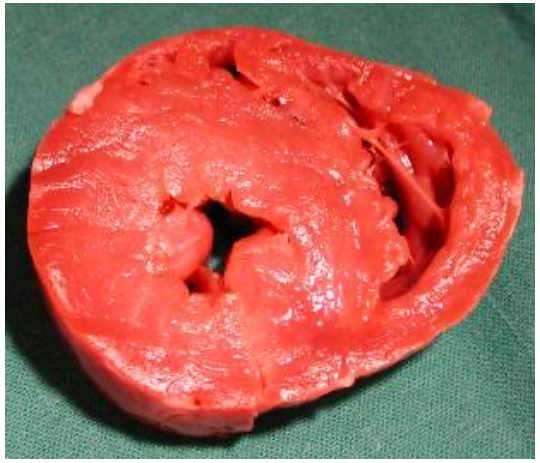
[1]Department of Veterinary Clinical and Animal Sciences, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark; [2]Department of Biomedical Sciences, Center for Healthy Aging, University of Copenhagen, Copenhagen, Denmark; and [3]Department of Veterinary Disease Biology, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark

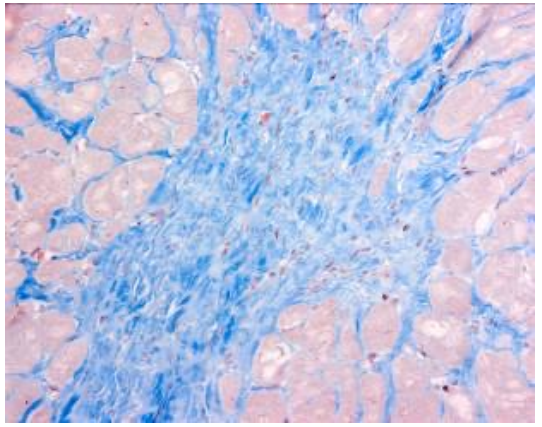
Submitted 10 October 2014; accepted in final form 10 March 2015

Pathogenesis of fHCM

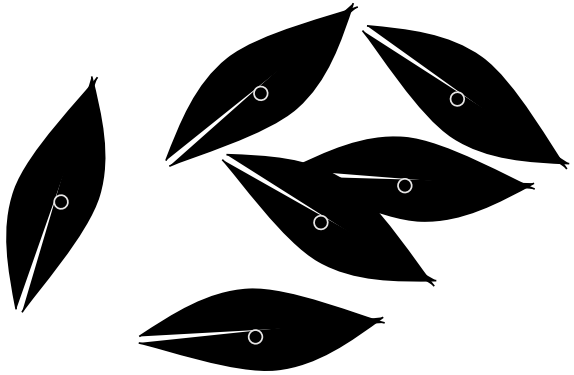
Tertiary (histological) phenotypes

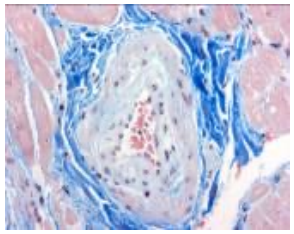
Myocyte hypertrophy
Myocyte disarray
Interestitial fibrosis
Cardiac hypertrophy





Septum, 20x, Maisson's trichrome

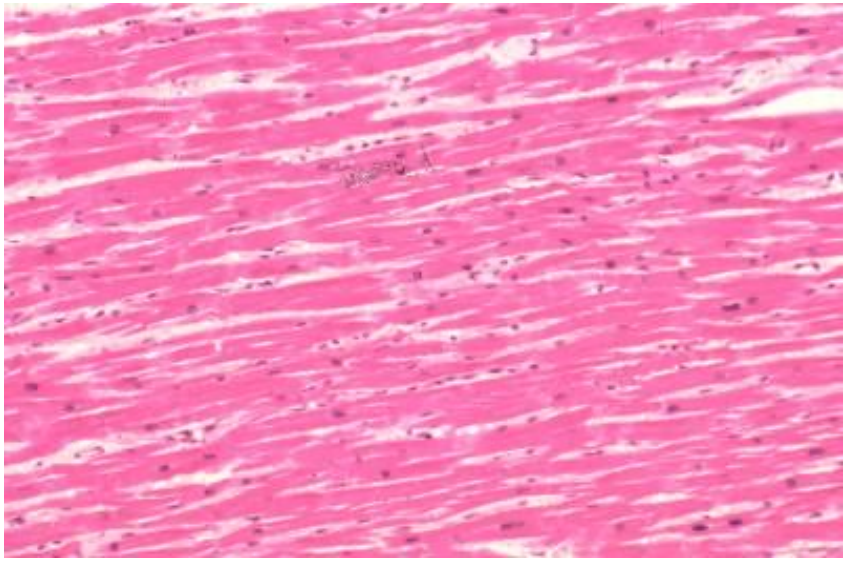




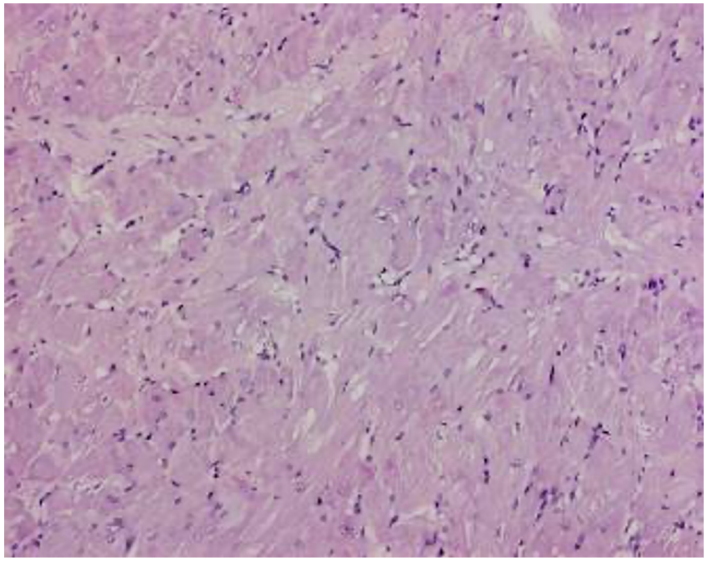
40x. Vessel

Cardiac Myocytes

Normal alignment



BSH, male, 2 years-old



HCM

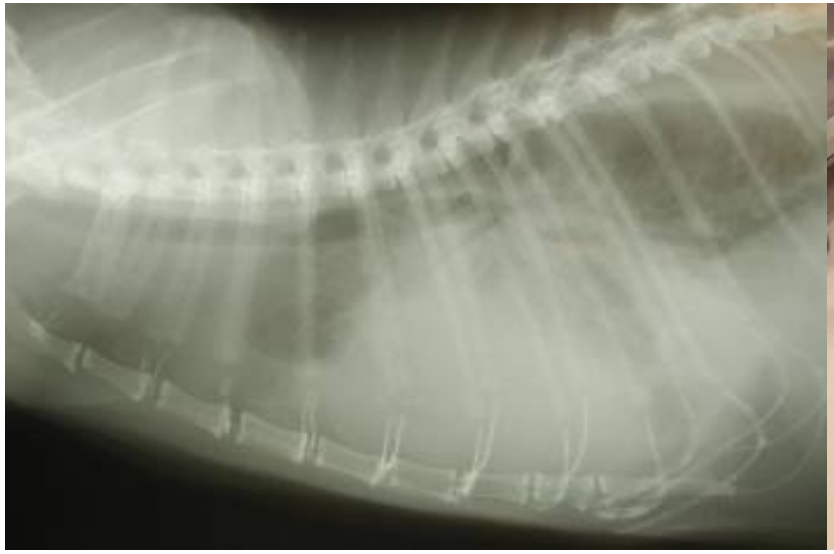
Photo: Assoc.prof. Pall S. Leifsson, IVH, KU

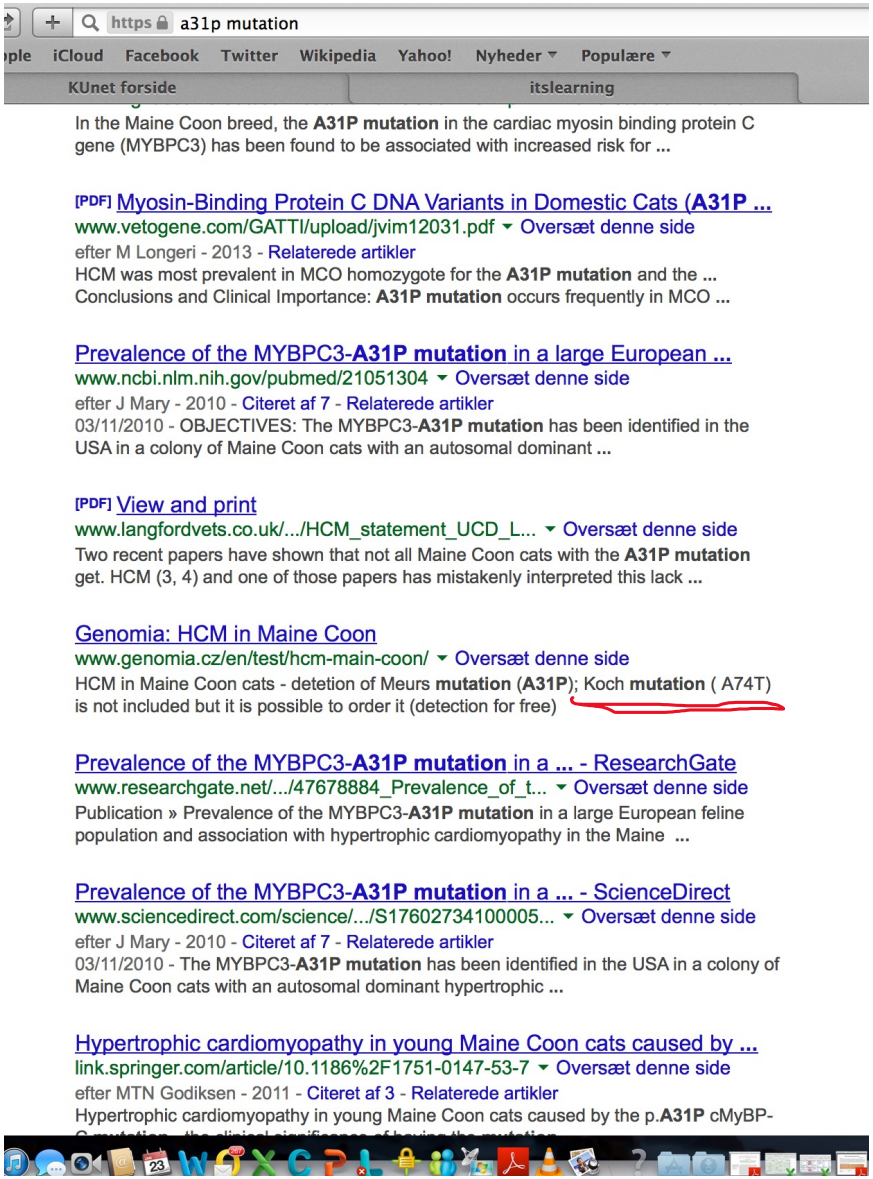
Myocyte disarray

Pathogenesis

Quaternary (clinical) phenotypes

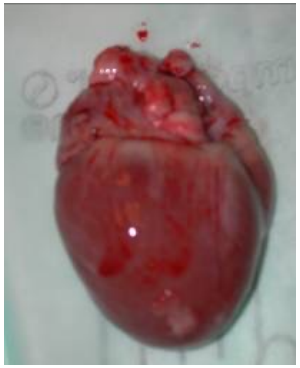
Cardiac arrhythmias
Sudden cardiac death
left ventricular outflow tract obstruction
Heart failure




https a31p mutation
iCloud Facebook Twitter Wikipedia Yahoo! Nyheder Populære
KUnet forside itslearning
In the Maine Coon breed, the A31P mutation in the cardiac myosin binding protein C gene (MYBPC3) has been found to be associated with increased risk for ...
[PDF] Myosin-Binding Protein C DNA Variants in Domestic Cats (A31P ...
www.vetogene.com/GATTI/upload/jvim12031.pdf Oversæt denne side
efter M Longeri - 2013 - Relaterede artikler
HCM was most prevalent in MCO homozygote for the A31P mutation and the ...
Conclusions and Clinical Importance: A31P mutation occurs frequently in MCO ...
Prevalence of the MYBPC3-A31P mutation in a large European ...
www.ncbi.nlm.nih.gov/pubmed/21051304 Oversæt denne side
efter J Mary - 2010 - Citeret af 7 - Relaterede artikler
03/11/2010 - OBJECTIVES: The MYBPC3-A31P mutation has been identified in the USA in a colony of Maine Coon cats with an autosomal dominant ...
[PDF] View and print
www.langfordvets.co.uk/.../HCM_statement_UCD_L... Oversæt denne side
Two recent papers have shown that not all Maine Coon cats with the A31P mutation get. HCM (3, 4) and one of those papers has mistakenly interpreted this lack ...
Genomia: HCM in Maine Coon
www.genomia.cz/en/test/hcm-main-coon/ Oversæt denne side
HCM in Maine Coon cats - detetion of Meurs mutation (A31P); Koch mutation (A74T) is not included but it is possible to order it (detection for free)
Prevalence of the MYBPC3-A31P mutation in a ... - ResearchGate
www.researchgate.net/.../47678884_Prevalence_of_t... Oversæt denne side
Publication » Prevalence of the MYBPC3-A31P mutation in a large European feline population and association with hypertrophic cardiomyopathy in the Maine ...
Prevalence of the MYBPC3-A31P mutation in a ... - ScienceDirect
www.sciencedirect.com/science/.../S17602734100005... Oversæt denne side
efter J Mary - 2010 - Citeret af 7 - Relaterede artikler
03/11/2010 - The MYBPC3-A31P mutation has been identified in the USA in a colony of Maine Coon cats with an autosomal dominant hypertrophic ...
Hypertrophic cardiomyopathy in young Maine Coon cats caused by ...
link.springer.com/article/10.1186%2F1751-0147-53-7 Oversæt denne side
efter MTN Godiksen - 2011 - Citeret af 3 - Relaterede artikler
Hypertrophic cardiomyopathy in young Maine Coon cats caused by the p.A31P cMyBP-

HCM in Cats

- Pathology:
  - Measure heart weight!
    - Normal < 20 g in a 5 kg cat
    - 0.35% of bodyweight
  - Heart weight from severe HCM affected cats often > 30-45 gram



Affected
BSH, male, 4 years-old

HCM screening within health programme

Participating clubs: Maine Coon-katten, Sällskapet Sibirisk Katt, Skogkattalingan, Rex United, Skogkattklubben Birka, Rasclub Maine Coon, Scandinavian Ragdoll Club, Birmasällskapet, SWEPEX (Svenska Perser & Exoticringen), Ragdollklubben
Visit http://www.pawpeds.com/healthprogrammes/ for more information

Patient Information

Cat's registered name

Registration number

ID number, microchip or tattoo

Race

Male Not altered
Female Altered

Born (year-month-day)

Sire

Dam

Owner's name

Address

Postcode/City/State

Country

Phone (including country code)

Email

I am aware that the results will be retained for the records of Maine Coon-katten. I authorize Maine Coon-katten to publicly release all results from this form

Signature **Date**

Examination

Examination date (year-month-day)

Sedated
Yes, with: No

Examination equipment

Weight _____ kg

Heart rate _____ bpm

Dehydrated Pregnant
Lactating Other, describe

Auscultation:
Normal Gallop
Murmur, characteristics
Grade: I II III IV V VI Dynamic Static
Timing: Systolic Diastolic Both Continuous
Location: Left apex (sternum) Left Base Other, describe

IVSd	_____ cm mm	M-mode	2-D	
LVIDd	_____	M-mode	2-D	
LVFWd	_____	M-mode	2-D	
IVSs	_____	M-mode	2-D	
LVIDs	_____	M-mode	2-D	
LVFWs	_____	M-mode	2-D	
SF	_____			
Ao	_____	M-mode	2-D	
LA	_____	M-mode	2-D	
LA/Ao	_____			

Subjective left atrial size
Normal
Mild enlargement
Moderate enlargement
Severe enlargement

Systolic anterior motion of the mitral valve yes no

If yes, LV outflow tract flow velocity (Doppler) _____

End-systolic cavity obliteration yes no

Papillary muscles
Normal
Abnormal, moderate enlargement
Abnormal, severe enlargement

Assessment (based on phenotype)

Normal
Equivocal
HCM Mild Moderate Severe
Other, describe

Comments

Veterinarian

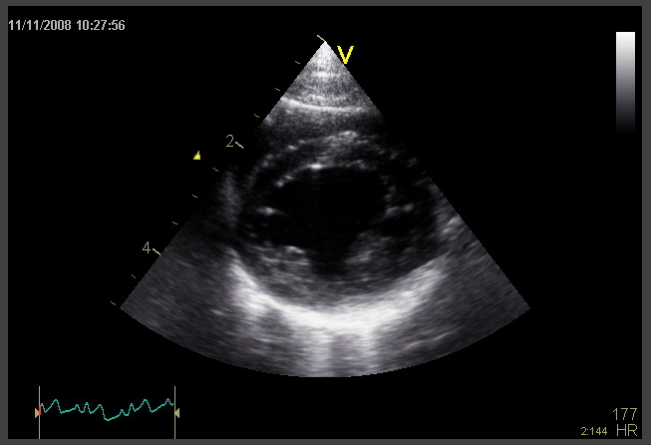
Cat's identity verified yes no, describe why not

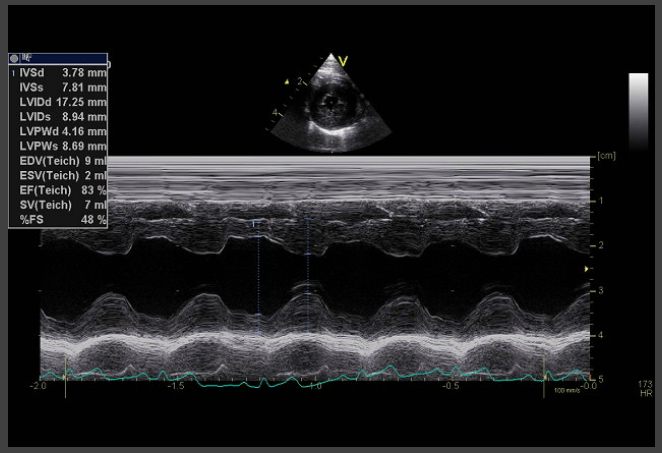
Signature **Date**

Veterinarian's name, clinic's name and address

For registration of the result, the veterinarian shall send a copy of this form to:
Maine Coon-katten, c/o Anne N. Jensen, Landsvinget 5, Nejede, 3400 Hillerød, Denmark

Rev 1.5 (en) 2007-03-04





Journal of Veterinary Internal Medicine
ACVIM
Open Access

J Vet Intern Med 2016

Effect of Body Weight on Echocardiographic Measurements in 19,866 Pure-Bred Cats with or without Heart Disease

J. Häggström, Å.O. Andersson, T. Falk, L. Nilsfors, U. OIsson, J.G. Kresken, K. Höglund, M. Rishniw, A. Tidholm, and I. Ljungvall

Background: Echocardiography is a cost-efficient method to screen cats for presence of heart disease. Current reference intervals for feline cardiac dimensions do not account for body weight (BW).

Objective: To study the effect of BW on heart rate (HR), aortic (Ao), left atrial (LA) and ventricular (LV) linear dimensions in cats, and to calculate 95% prediction intervals for these variables in normal adult pure-bred cats.

Animals: 19 866 pure-bred cats.

Methods: Clinical data from heart screens conducted between 1999 and 2014 were included. Associations between BW, HR, and cardiac dimensions were assessed using univariate linear models and allometric scaling, including all cats, and only those considered normal, respectively. Prediction intervals were created using 95% confidence intervals obtained from regression curves.

Results: Associations between BW and echocardiographic dimensions were best described by allometric scaling, and all dimensions increased with increasing BW (all $P<0.001$). Strongest associations were found between BW and Ao, LV end diastolic, LA dimensions, and thickness of LV free wall. Weak linear associations were found between BW and HR and left atrial to aortic ratio (LA:Ao), for which HR decreased with increasing BW ($P<0.001$), and LA:Ao increased with increasing BW ($P<0.001$). Marginal differences were found for prediction formulas and prediction intervals when the dataset included all cats versus only those considered normal.

Conclusions and Importance: BW had a clinically relevant effect on echocardiographic dimensions in cats, and BW based 95% prediction intervals may help in screening cats for heart disease.

Key words: Heart dimensions; M-mode; Prediction intervals; Screening.

breed, the database consisted of 5,274 Maine Coon, 3,301 Norwegian Forest, 2,663 British Shorthair, 1,832 Siberian, 1,809 Ragdoll, 1,258 Sphynx, 914 Birman, 745 Cornish Rex, 584 Bengal, 526 Devon Rex, 204 Persian, and 756 cats of other breeds. The latter group consisted of 38 different breeds of which British Longhair, European, and Ocicat were the most common (>50 cats per breed).

Table 1. Medians and interquartile ranges (IQR) for age, body weight, heart rate, and echocardiographic measurements in all cats, in cats classified as normal, according to stipulated criteria (see materials and methods), and in cats with abnormal findings.

Variable	All (n = 19,866)	Normal (n = 18,460)	Abnormal (n = 1,406)	*P*-value
Age (years)	1.8 (1.1–3.2)	1.8 (1.1–3.1)	4.5 (1.5–4.5)	<.0001
Body weight (kg)	4.2 (3.5–5.1)	4.2 (3.5–5.1)	4.6 (3.7–5.9)	<.0001
Heart rate (beats/min)	180 (160–196)	180 (160–195)	180 (160–200)	<.0001
IVSd (mm)	3.9 (3.5–4.3)	3.9 (3.5–4.3)	5.2 (4.3–5.9)	<.0001
LVIDd (mm)	15.6 (14.3–17.0)	15.7 (14.3–17.1)	15.0 (13.4–16.7)	<.0001
LVFWd (mm)	3.8 (3.4–4.3)	3.8 (3.4–4.2)	5.1 (4.2–5.8)	<.0001
IVSs (mm)	6.2 (5.5–7.0)	6.2 (5.5–7.0)	7.6 (6.4–8.6)	<.0001
LVIDs (mm)	8.5 (7.3–9.8)	8.5 (7.4–9.8)	7.5 (6.1–9)	<.0001
LVFWs (mm)	6.5 (5.8–7.2)	6.4 (5.7–7.1)	7.7 (6.7–8.8)	<.0001
FS%	45 (39–51)	45 (39–51)	49 (42–56)	<.0001
Ao (mm)	9.3 (8.5–10.2)	9.3 (8.5–10.2)	9.5 (8.8–10.5)	<.0001
LA (mm)	10.8 (9.6–12.0)	10.8 (9.6–12.0)	11.7 (10.0–13.4)	<.0001
LA:Ao	1.14 (1.06–1.24)	1.13 (1.06–1.23)	1.2 (1.10–1.35)	<.0001

IVSd, interventricular septum diastole; LVIDd, left ventricular internal diameter diastole; LVFWd, left ventricular free wall diastole; IVSs, interventricular septum systole; LVIDs, left ventricular internal diameter systole; LVFWs, left ventricular free wall systole; FS%, fractional shortening; Ao, aortic diameter; LA, left atrial diameter; and LA:Ao, left atrial-to-aortic ratio.

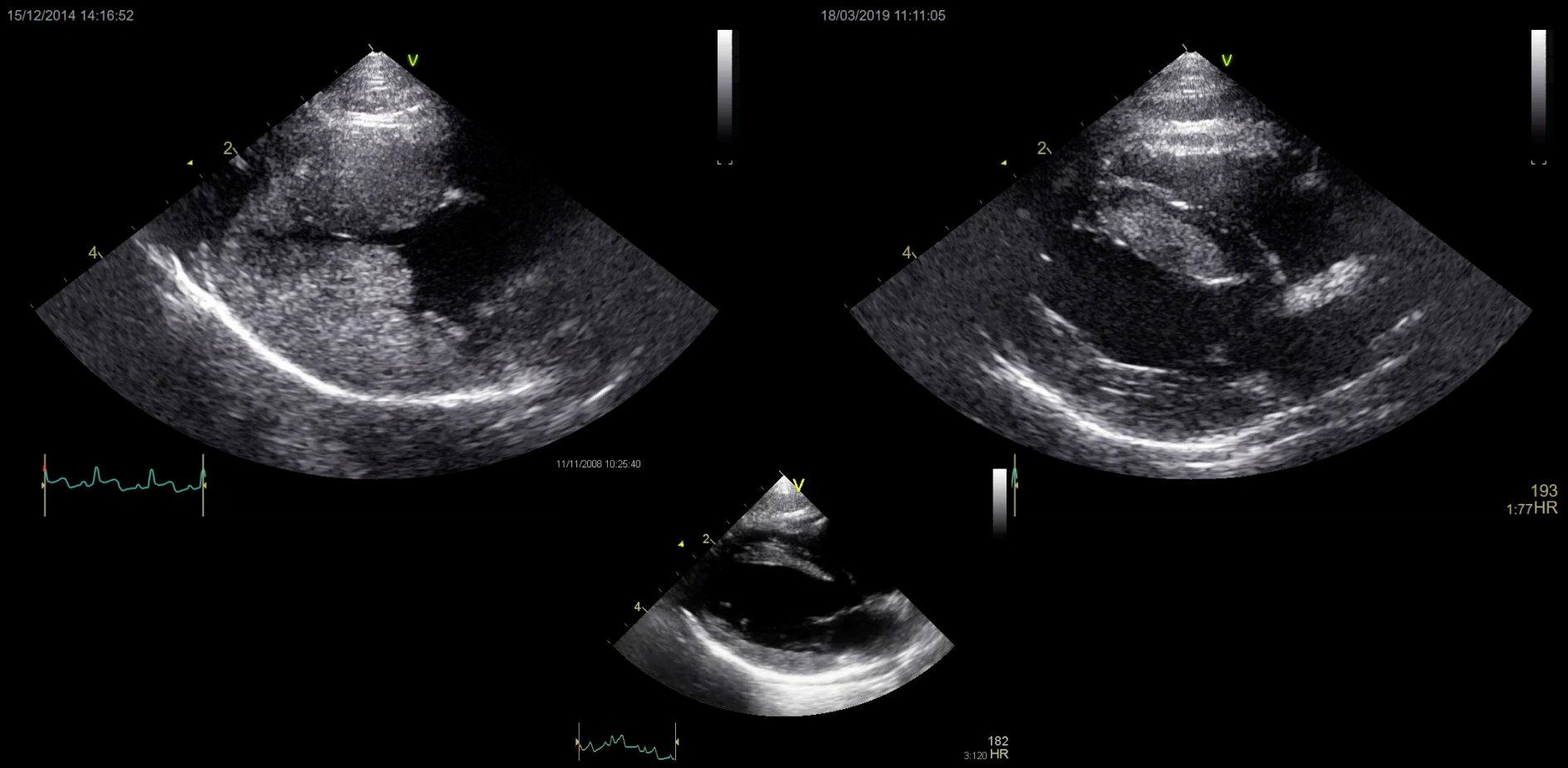
Nye grænseværdier

Table 3. Predicted cardiac dimensions and 95% prediction intervals for 18460 cats with normal echocardiograms according to specific, stated criteria (see materials and methods).

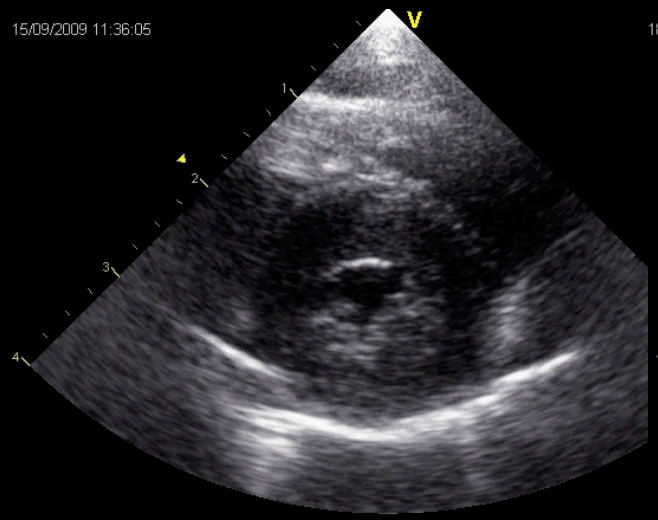
Weight (kg)	IVSd (mm)	LVIDd (mm)	LVFWd (mm)	IVSs (mm)	LVIDs (mm)	LVFWs (mm)	FS (%)	LA (mm)	Ao (mm)	LA:Ao
1.5	3.1 (2.3–4.0)	11.9 (9.5–15.0)	2.9 (2.2–3.8)	4.8 (3.5–6.7)	6.4 (4.2–9.6)	4.8 (3.6–6.5)	45 (28–62)	7.7 (5.8–10.2)	7.0 (5.5–8.8)	1.13 (0.85–1.40)
2.0	3.3 (2.5–4.3)	12.8 (10.2–16.0)	3.1 (2.4–4.1)	5.2 (3.7–7.2)	6.9 (4.6–10.5)	5.2 (3.9–7.1)	45 (28–62)	8.5 (6.3–11.2)	7.5 (6.0–9.5)	1.13 (0.85–1.40)
2.5	3.4 (2.6–4.5)	13.6 (10.9–17.0)	3.2 (2.5–4.4)	5.4 (3.9–7.6)	7.4 (4.8–11.2)	5.5 (4.1–7.5)	45 (28–62)	9.1 (6.8–12.0)	8.0 (6.3–10.1)	1.14 (0.86–1.41)
3.0	3.5 (2.7–4.7)	14.2 (11.4–17.8)	3.4 (2.6–4.5)	5.7 (4.1–7.9)	7.7 (5.1–11.7)	5.8 (4.3–7.9)	45 (28–62)	9.6 (7.2–12.7)	8.4 (6.7–10.7)	1.14 (0.86–1.42)
3.5	3.7 (2.8–4.9)	14.8 (11.9–18.5)	3.6 (2.7–4.7)	5.9 (4.2–8.2)	8.0 (5.3–12.2)	6.0 (4.5–8.2)	45 (28–62)	10.0 (7.6–13.4)	8.8 (7.0–11.1)	1.15 (0.87–1.42)
4.0	3.8 (2.8–4.9)	15.4 (12.2–19.2)	3.7 (2.8–4.8)	6.0 (4.3–8.4)	8.3 (5.5–12.6)	6.3 (4.6–8.5)	45 (28–62)	10.5 (7.9–13.9)	9.1 (7.2–11.6)	1.15 (0.88–1.43)
4.5	3.9 (2.9–5.1)	15.8 (12.7–19.8)	3.8 (2.9–5.0)	6.2 (4.4–8.7)	8.6 (5.7–13.0)	6.5 (4.8–8.7)	45 (28–62)	10.9 (8.2–14.5)	9.4 (7.5–11.9)	1.15 (0.88–1.43)
5.0	3.9 (3.0–5.2)	16.3 (13.0–20.3)	3.9 (3.0–5.1)	6.4 (4.6–8.9)	8.8 (5.8–13.4)	6.6 (4.9–9.0)	45 (28–62)	11.2 (8.4–14.9)	9.7 (7.7–12.3)	1.16 (0.88–1.43)
5.5	4.0 (3.0–5.3)	16.7 (13.4–20.9)	4.0 (3.0–5.3)	6.5 (4.7–9.1)	9.0 (6.0–13.7)	6.8 (5.0–9.2)	45 (28–62)	11.6 (8.7–15.4)	10.0 (7.9–12.6)	1.16 (0.89–1.44)
6.0	4.1 (3.1–5.4)	17.1 (13.7–21.4)	4.1 (3.1–5.4)	6.6 (4.7–9.3)	9.3 (6.1–14.1)	7.0 (5.1–9.4)	45 (28–62)	11.9 (8.9–15.8)	10.2 (8.1–12.9)	1.16 (0.89–1.44)
6.5	4.2 (3.1–5.5)	17.4 (14.0–21.8)	4.2 (3.1–5.5)	6.7 (4.8–9.4)	9.4 (6.2–14.3)	7.1 (5.3–9.6)	45 (28–62)	12.2 (9.2–16.2)	10.5 (8.3–13.2)	1.17 (0.90–1.45)
7.0	4.2 (3.2–5.6)	17.8 (14.2–22.2)	4.3 (3.2–5.6)	6.9 (4.9–9.6)	9.6 (6.3–14.6)	7.3 (5.4–9.8)	45 (28–62)	12.5 (9.4–16.6)	10.7 (8.4–13.5)	1.18 (0.90–1.46)
7.5	4.3 (3.2–5.7)	18.1 (14.5–22.6)	4.3 (3.3–5.7)	7.0 (5.0–9.7)	9.8 (6.5–14.9)	7.4 (5.5–10.0)	45 (28–62)	12.7 (9.6–16.9)	10.9 (8.6–13.8)	1.18 (0.91–1.46)
8.0	4.3 (3.3–5.8)	18.4 (14.7–23.0)	4.4 (3.3–5.8)	7.1 (5.1–9.9)	10.0 (6,6–15.1)	7.5 (5.6–10.2)	45 (28–62)	13.0 (9.8–17.3)	11.1 (8.8–14.0)	1.19 (0.91–1.47)
8.5	4.4 (3.3–5.8)	18.7 (15.0–23.4)	4.4 (3.4–5.9)	7.2 (5.1–10.0)	10.1 (6.7–15.4)	7.6 (5.6–10.3)	45 (28–62)	13.2 (10.0–17.6)	11.3 (8.9–14.3)	1.19 (0.92–1.47)
9.0	4.4 (3.3–5.9)	19.0 (15.2–23.7)	4.5 (3.4–5.9)	7.3 (5.2–10.2)	10.3 (6.8–15.6)	7.7 (5.7–10.5)	45 (28–62)	13.5 (10.1–17.9)	11.5 (9.1–14.5)	1.20 (0.92–1.47)
9.5	4.5 (3.4–6.0)	19.3 (15.4–24.0)	4.6 (3.4–5.9)	7.4 (5.3–10.3)	10.4 (6.9–15.8)	7.9 (5.8–10.6)	45 (28–63)	13.7 (10.3–18.2)	11.6 (9.1–14.7)	1.20 (0.92–1.48)
10.0	4.5 (3.4–6.0)	19.5 (15.6–24.4)	4.6 (3.5–6.1)	7.4 (5.3–10.4)	10.5 (6.9–16.0)	8.0 (5.9–10.8)	45 (28–63)	13.9 (10.5–18.5)	11.8 (9.3–14.9)	1.21 (0.92–1.48)
10.5	4.6 (3.5–6.1)	19.8 (15.8–24.7)	4.7 (3.5–6.2)	7.5 (5.4–10.5)	10.7 (7.1–16.3)	8.1 (6.0–10.9)	45 (28–63)	14.1 (10.6–18.8)	11.9 (9.5–15.1)	1.22 (0.94–1.49)
11.0	4.6 (3.5–6.1)	20.0 (16.0–25.0)	4.7 (3.5–6.2)	7.6 (5.4–10.6)	10.8 (7.2–16.5)	8.1 (6.0–11.0)	45 (28–63)	14.3 (10.8–19.1)	12.1 (9.6–15.3)	1.22 (0.94–1.50)

HR, heart rate; IVSd, interventricular septum diastole; LVIDd, left ventricular internal diameter diastole; LVFWd, left ventricular free wall diastole; IVSs, interventricular septum systole; LVIDs, left ventricular internal diameter systole; LVFWs, left ventricular free wall systole; FS%, fractional shortening; Ao, aortic diameter; LA, left atrial diameter; and LA:Ao, left atrial-to-

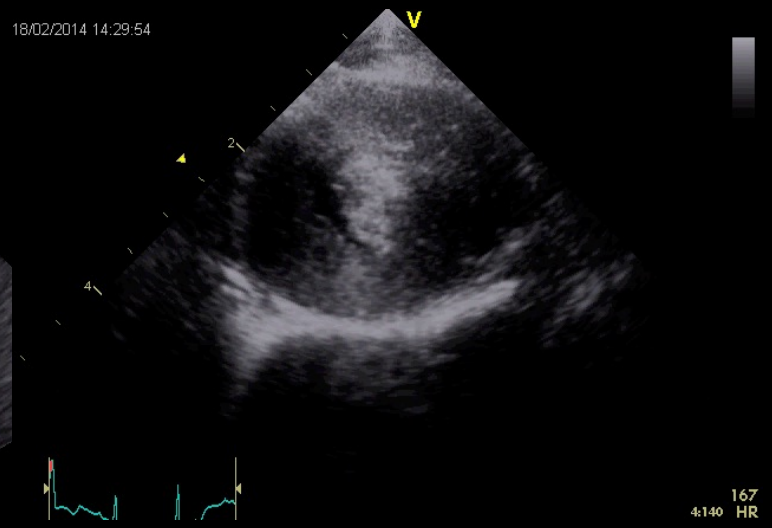
Breed-differences in Phenotype



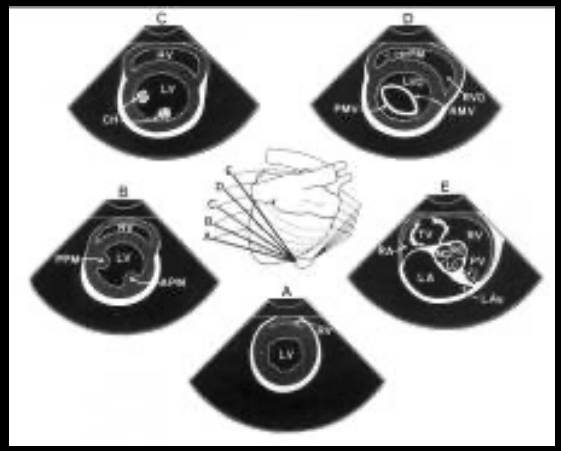
HCM in Ragdolls



Healthy Ragdoll



HCM-affected



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SPONTANEOUSLY-ARISING DISEASE

An Immunohistochemical Study of Feline Myocardial Fibrosis

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** Institute für Veterinär-Pathologie, An den Tierkliniken 33 and † Klinik für Kleintiere, An den Tierkliniken 23, Veterinärmedizinische Fakultät der Universität Leipzig, Germany*

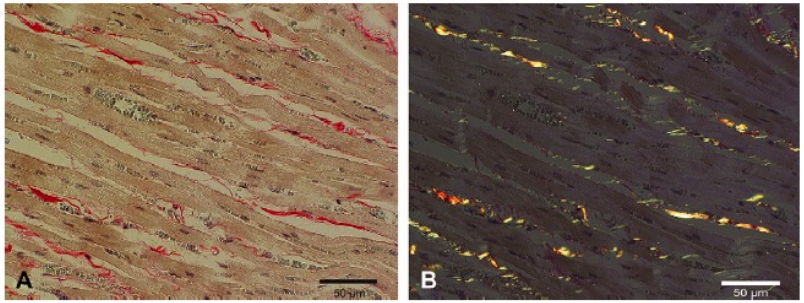


Fig. 3. Microscopical findings in the heart of an 8-year-old DSH cat. (A) Normal myocardium with few collagen fibres (red) between cardiomyocytes. Picrosirius red stain. (B) In the normal myocardium most collagen fibres are of collagen type I (yellow) and few fibres are of collagen type III (green). Picrosirius red stain with polarized light.

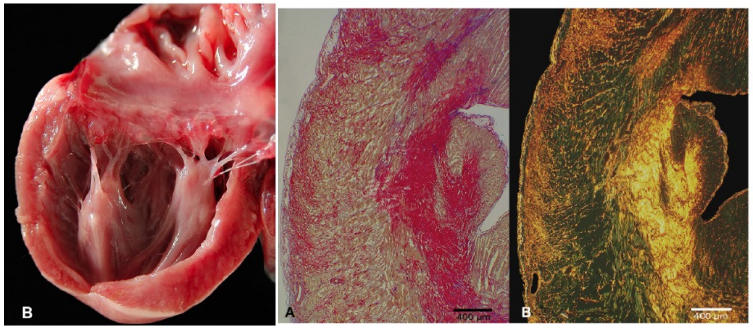
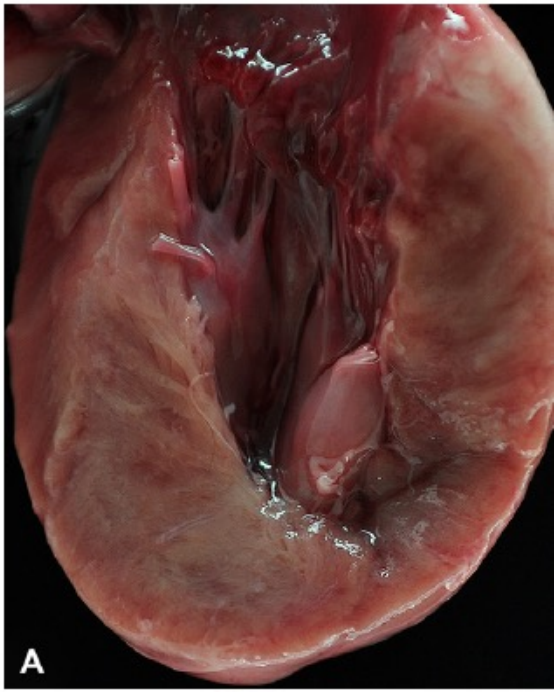
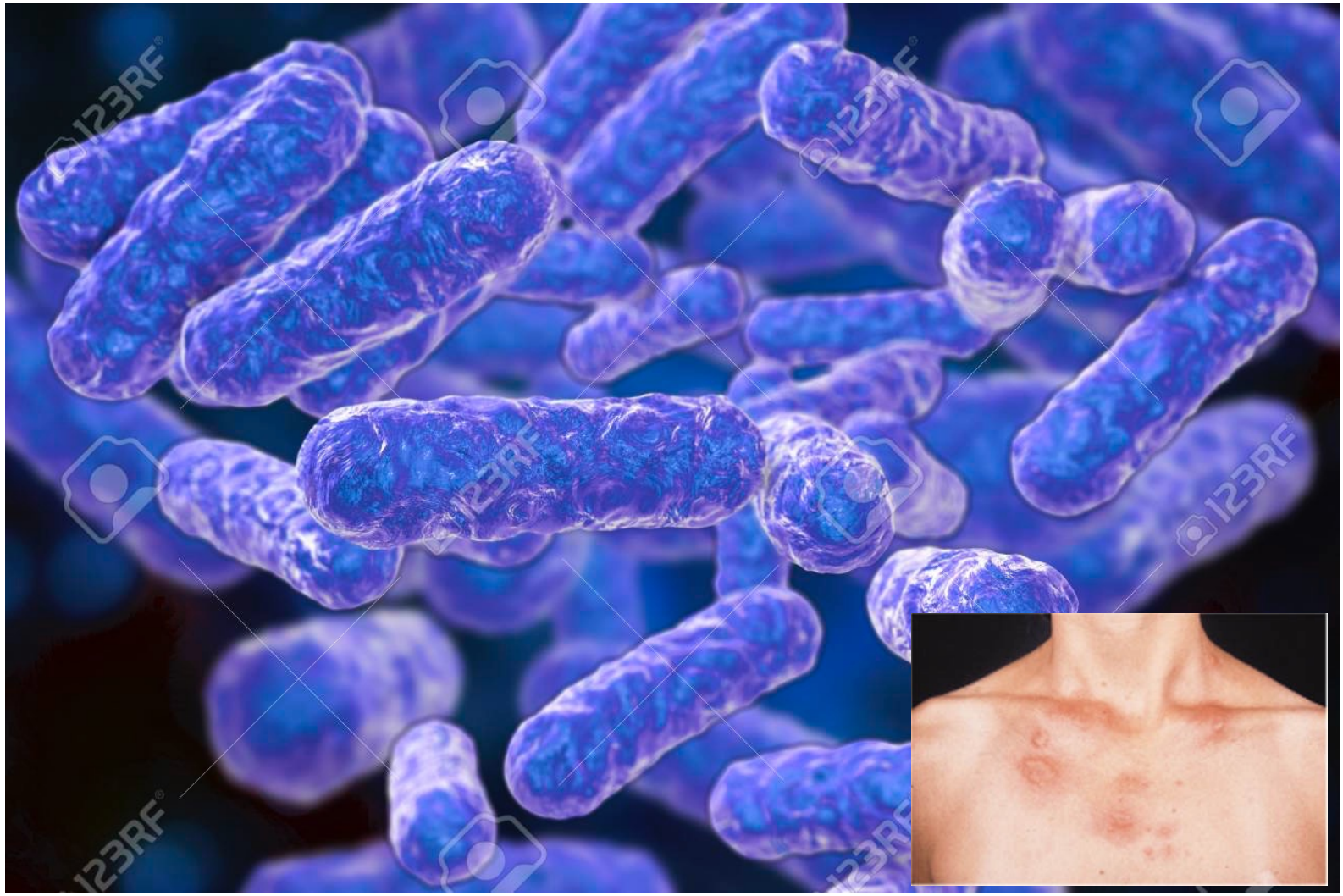


Fig. 4. Microscopical findings in the heart of a 7-year-old exotic short hair cat (see also Fig. 2B). (A) Large areas of myocardial fibrosis (red). Picrosirius red stain. (B) The fibrotic areas are mainly composed of type I collagen fibres (yellow). Picrosirius red stain with polarized light.



'ig. 2. (A) Heart from a 14-year-old DSH cat with marked hypert areas of white scar formation. (B) Heart from a 7-year-old ex to extensive myocardial fibrosis (see Fig. 4), but the papilla ened (feline endocardiosis).

Bartonella henselae



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INFECTIOUS DISEASE

Bartonella spp. as a Possible Cause or Cofactor of Feline Endomyocarditis–Left Ventricular Endocardial Fibrosis Complex

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Summary

Endomyocarditis is a commonly detected post-mortem finding in domestic cats presenting for sudden onset cardiovascular death, yet the aetiology remains unresolved. Cats are documented reservoir hosts for *Bartonella henselae*, the infectious cause of cat scratch disease in man. Various *Bartonella* spp. have been associated with culture-negative endocarditis, myocarditis and sudden death in man and animals. We hypothesized that *Bartonella* spp. DNA could be amplified more often from the hearts of cats with feline endomyocarditis–left ventricular endocardial fibrosis (FEMC–LVEF) complex compared with cats with hypertrophic cardiomyopathy (HCM) or cats with grossly and microscopically unremarkable hearts (designated non-cardiac disease controls). Formalin-fixed and paraffin wax-embedded, cardiac tissues from 60 domestic and purebred cats aged 3 months to 18 years were examined, and histological features were recorded. Cardiac tissue sections were tested for *Bartonella* DNA using multiple 16–23S intergenic transcribed spacer region polymerase chain reaction (PCR) primer sets, including two *Bartonella* genera, a *Bartonella koehlerae* species-specific and a *Bartonella vinsonii* subsp. *berkhoffii*-specific assay, followed by DNA sequence confirmation of the species or genotype. Special precautions were taken to avoid DNA cross-contamination between tissues. *Bartonella* spp. DNA was amplified by PCR and sequenced from 18 of 36 cats (50%) with FEMC–LVEF and 1/12 (8.3%) cats with HCM. *Bartonella* spp. DNA was not amplified from any non-cardiac disease control hearts. Based on PCR/DNA sequencing, one *Bartonella* spp. was amplified from 10 cats, while the remaining eight were coinfected with more than one *Bartonella* spp. To our knowledge, this study represents the first documentation of *B. vinsonii* subsp. *berkhoffii* genotype I infection in cats ($n = 11$). Fluorescence in-situ hybridization testing facilitated visualization of *Bartonella* bacteria within the myocardium of four of seven PCR-positive FEMC–LVEF hearts. Collectively, these findings support the hypothesis that *Bartonella* spp. may play a primary role or act as a cofactor in the pathogenesis of FEMC–LVEF. Studies involving cats from other geographical regions and definitive demonstration of *Bartonella* spp. within regions of inflammation are needed to confirm an association between *Bartonella* spp. and FEMC–LVEF induced morbidity and mortality in cats.

Bartonella?! (Restrictive Cardiomyopathy)

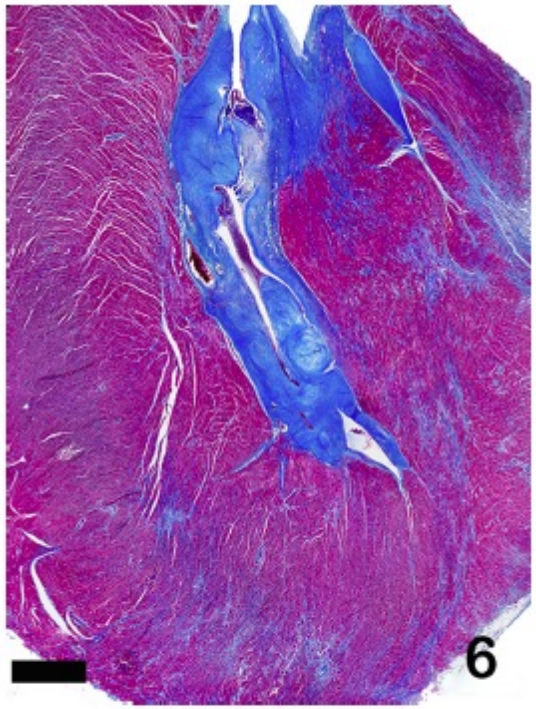


Fig. 6. Case 33, FEMC–LVEF cohort, subgross image. Left ventricular endocardial and myocardial fibrosis are highlighted by blue staining. Trichrome stain. Bar, 2 mm.

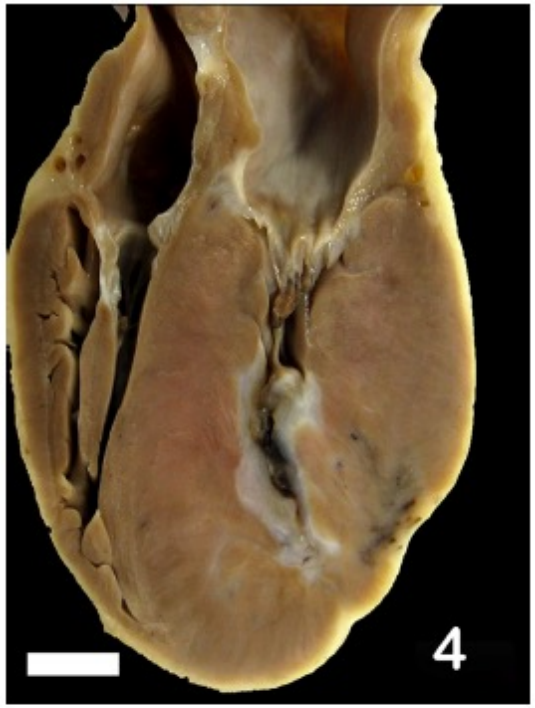
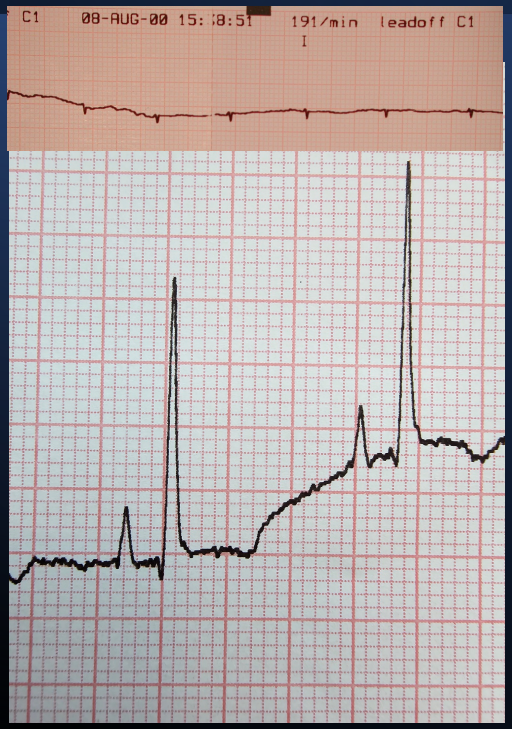


Fig. 4. Case 33, FEMC–LVEF cohort, gross longitudinal four-chamber cardiac dissection. There is severe, generalized left ventricular hypertrophy and mild right ventricular wall thickening. There is marked endocardial thickening associated with fibrosis in the mid to apical left ventricular chamber. Regions of mottling and white discolouration are present within the myocardium (fibrosis). The left atrium is markedly enlarged with diffuse endocardial fibrosis. The left atrioventricular valve is mildly thickened. Bar, 5 mm.

False Tendons in Humans and Cats



Birman cat, 6Y, Male

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Echocardiographic assessment of feline false tendons and their relationship with focal thickening of the left ventricle

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In conclusion, focal insertion is often associated with increased segmental wall thickness of the IVS in diastole. Although focal hypertrophy can occur with feline HCM, the FT-regions did not apparently change over time when compared with non-FT regions. Furthermore, there was no obvious relationship with other forms of LV hypertrophy during routine echocardiographic examinations. The mean growth at the FT-region did not significantly differ from the growth at the non-FT region at follow-up examinations, suggesting that these regions might be normal variation or of little clinical significance to non-breeding cats. It is important to carefully scrutinize focal thickening in an echocardiographic examination and review echocardiographic loops and 2D images for FT insertions. Furthermore, care must be taken to not make erroneous measurements by including an FT to the wall measurement or by erroneously filling in the space between the FT and the endocardium because of inappropriate contrast and grayscale settings. Finally, it is important to conduct off-axis imaging [6,21,31,32] to verify that true septal thickness is not being altered by an adjacent FT running parallel to the IVS leading to erroneous measurements [13–15,33].

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Anatomia, Histologia, Embryologia

SHORT COMMUNICATION

Incidence, Distribution and Morphology of Left Ventricular False Tendons in Cat Hearts

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With 3 figures

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Summary

The incidence, distribution, and macro- and microscopic structures of left ventricular false tendons (LVFTs) in the cat heart were studied using 25 normal and 57 diseased hearts. The fibrous bands were observed in the left ventricle of all 82 cat hearts examined and most commonly extended between the papillary muscles and the ventricular septum. Histologically, the LVFTs were composed of central Purkinje fibres and surrounding dense collagenous fibres covered by endothelium. There was no appreciable difference in the incidence, distribution or morphology of LVFTs between the normal and the diseased hearts, indicating that LVFTs are a common anatomic variant in the cat heart.

CLINICAL INVESTIGATION

Left Ventricular False Tendons are Associated With Left Ventricular Dilation and Impaired Systolic and Diastolic Function

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ABSTRACT

Background: Left ventricular false tendons (LVFTs) are chord-like structures that traverse the LV cavity and are generally considered to be benign. However, they have been associated with arrhythmias, LV hypertrophy and LV dilation in some small studies. We hypothesize that LVFTs are associated with LV structural and functional changes assessed by echocardiography.

Methods: We retrospectively evaluated echocardiographic and clinical parameters of 126 patients identified as having LVFTs within the past 2 years and compared them to 85 age-matched controls without LVFTs.

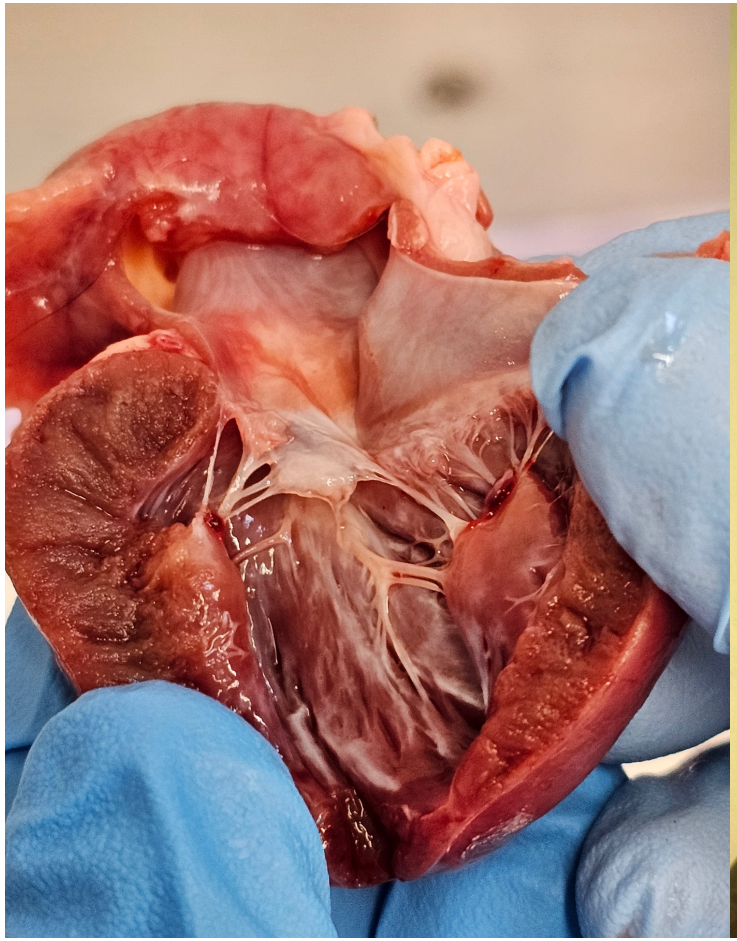
Results: There were no significant differences in age (52 ± 18 versus 54 ± 18 years, $P = 0.37$), sex (55% versus 59% men, $P = 0.49$), race (36% versus 23% white, $P = 0.07$), systolic blood pressure (131 ± 22 versus 132 ± 23 mmHg, $P = 0.76$) or body mass index (BMI, 31 ± 8 versus 29 ± 10 kg/m^2, $P = 0.07$) between controls and patients with LVFTs, respectively. Patients with LVFTs had more prevalent heart failure (43% versus 21%, $P = 0.001$). Patients with LVFTs had more LV dilation, were 2.5 times more likely to have moderate-to-severe mitral regurgitation, had more severe diastolic dysfunction and reduced LV systolic function (18% lower) compared with controls (all $P < 0.05$). After adjustment for covariates, basal and middle LVFT locations were associated with reduced LV systolic function ($P < 0.01$), and middle LVFTs were associated with LV dilation ($P < 0.01$).

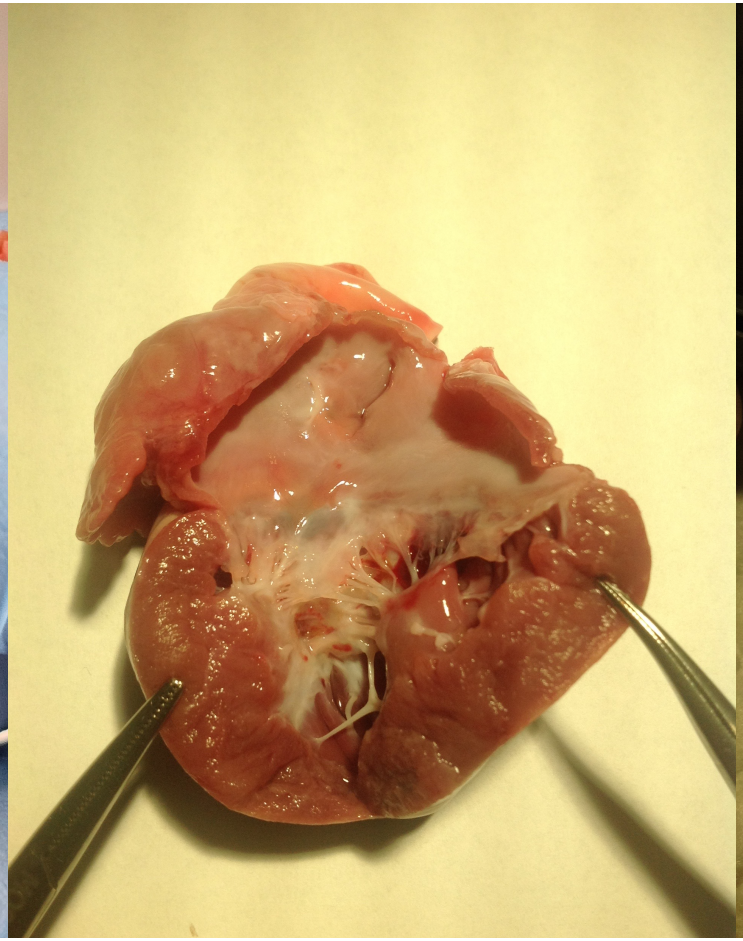
Conclusions: Our findings suggest that LVFTs may not be benign variants, and basal and middle LVFTs may have more deleterious effects. Further prospective studies should be performed to determine their pathophysiological significance and whether they play a causal role in LV dysfunction.

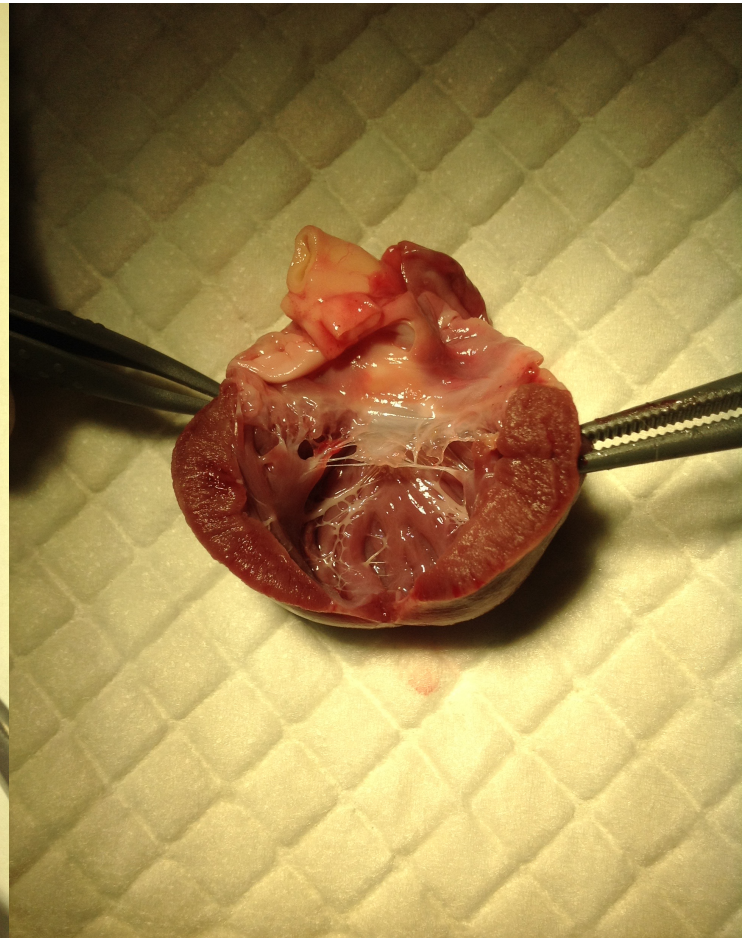
Key Indexing Terms: Myocardial band; Left ventricle; Cardiac dysfunction. [Am J Med Sci 2017;354(3):278–284.]

False Tendons – Normal Variant eller Sygdom?

3 fænotyper?!







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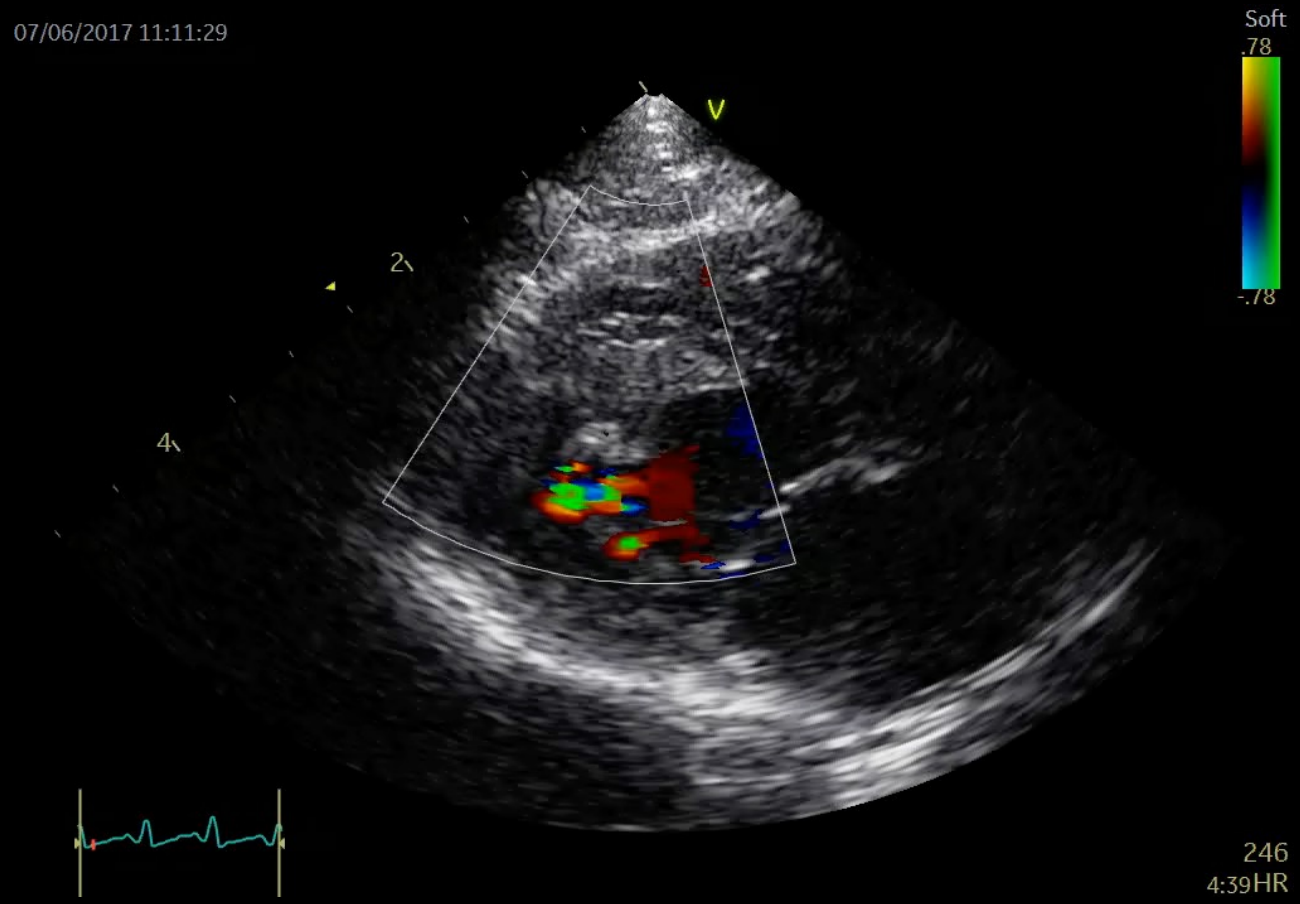
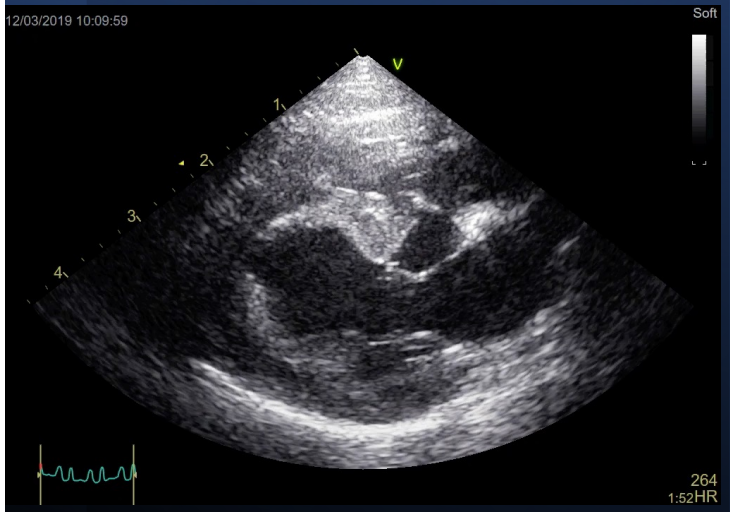
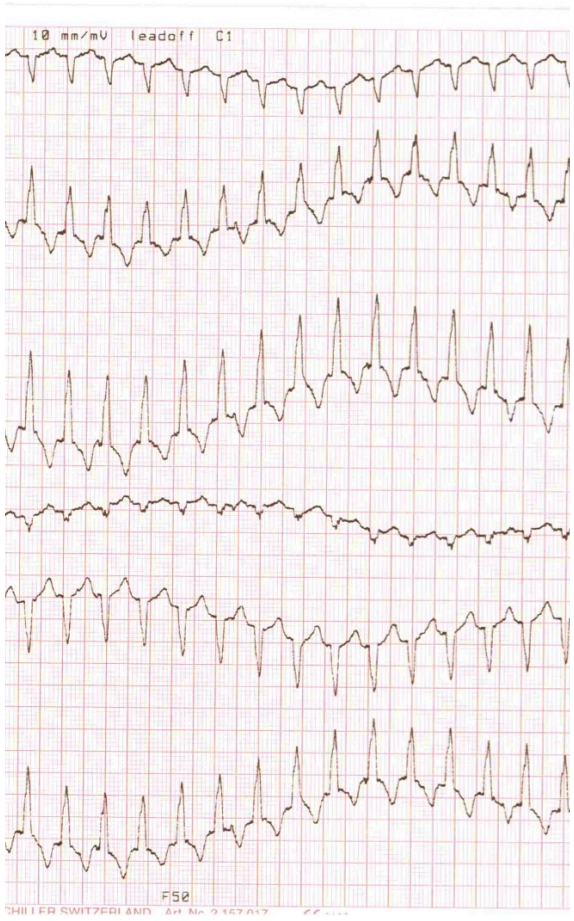
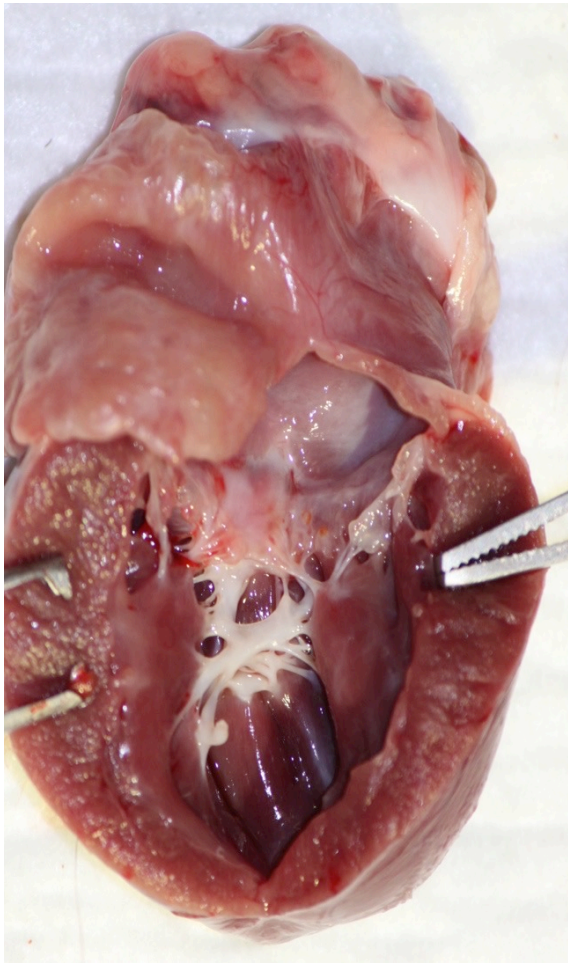


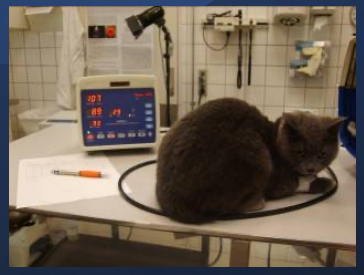
False Tendons



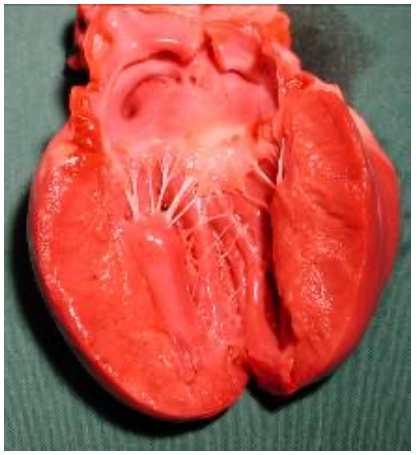


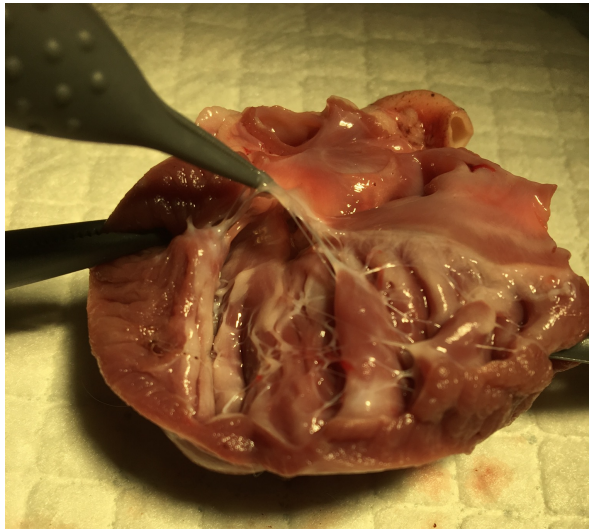


Differential Diagnosis



- Aortic stenosis
- Hyperthyroidism
- Hypertension
  - Chronic renal disease
- Papillary muscle malpositioned
- False tendons
- Transient Myocardial Thickening
- Myocarditis
- Myocardial fibrosis
- Dehydration
- Lymphoma
- Acromegaly
- Amyloidosis
- Intra- and inter-breed variation
- Observer variation





Prognosis

Population and survival characteristics of cats with hypertrophic cardiomyopathy: 260 cases (1990–1999)

John E. Rush, DVM, MS, DACVIM, DACVECC; Lisa M. Freeman, DVM, PhD, DACVN; Nathaniel K. Fenollosa, DVM; Donald J. Brown, DVM, PhD, DACVIM

Asymptomatic for years	• Median survival time 1129 days
Syncope	• MST 654 days
Congestive heart failure	• MST 563 days
Thromboembolism	• MST 183 days • Poor to guarded
Sudden death less than 5-10%?	• Genotype?
Genotype!?	• Future - gene therapy

Symptomatic – Median survival 194 days (Paine et al, 2010)

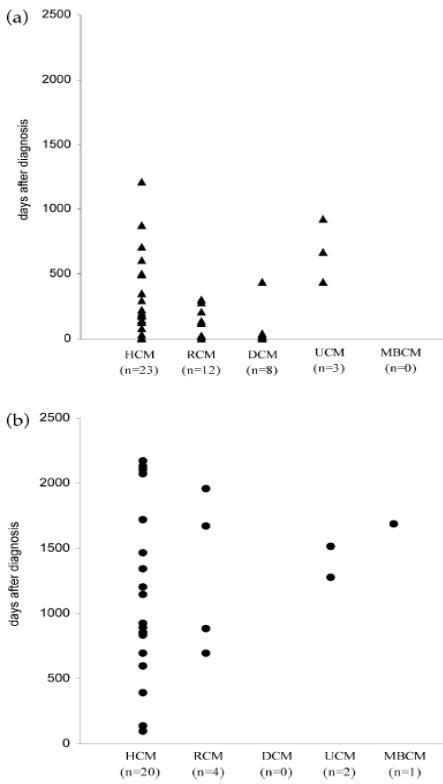


Fig 1. (a) Survival of 46 cats with cardiomyopathy that died before the time of publication. (b) Survival of 27 cats with cardiomyopathy that were still alive at the time of publication.

Journal of Feline Medicine and Surgery (2003) 5, 151–159
doi:10.1016/S1098-612X(02)00133-X

jfms

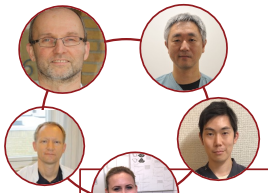
Feline idiopathic cardiomyopathy: a retrospective study of 106 cats (1994–2001)

L Ferasin*, CP Sturgess, MJ Cannon[1], SMA Caney, TJ Gruffydd-Jones, PR Wotton[2]

JAVMA, Vol 220, No. 2, January 15, 2002

Maiken Bach, Studieadjunkt i klinisk kardiologi og Ph.D studerende

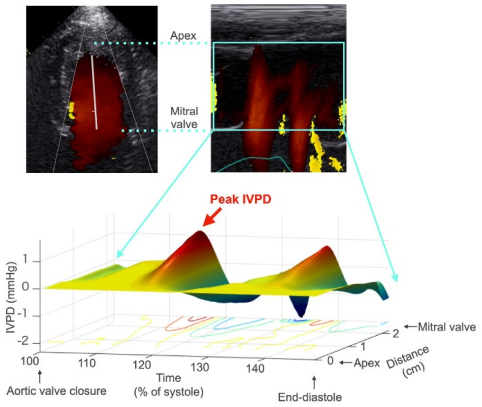
Ph.D. i avanceret ekkokardiografi 2025: Nye ekkokardiografiske markører for …



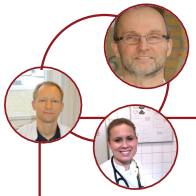
Journal of Veterinary Cardiology
Volume 41, June 2022, Pages 236-248

Non-invasive assessment of left ventricular relaxation property using color M-mode-derived intraventricular pressure gradients in cats ☆

K. Matsuura PhD [a, d], M.B.T. Bach DVM [b], [d], K. Takahashi PhD [c], J.L. Willesen PhD [b], J. Koch PhD [b, e], R. Tanaka PhD [a, e]



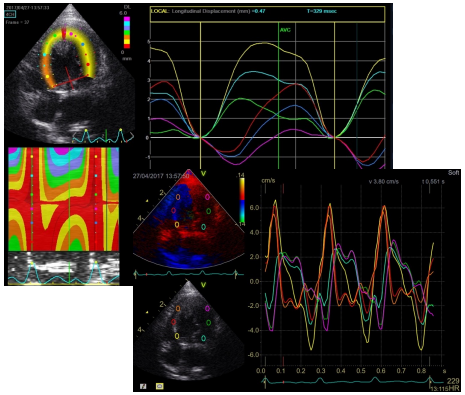
Tidlig detektion af HCM



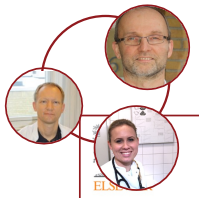
Status: Data opmåles!

Left ventricular myocardial dyssynchrony and 2D strain in cats with hypertrophic cardiomyopathy

M.B.T. Bach, J.L. Willesen, J.Koch (et al.)



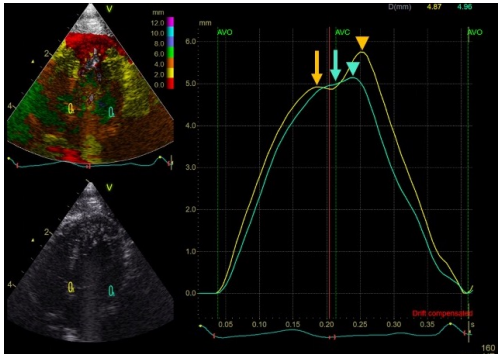
Risiko vurdering af HCM katte



Journal of Veterinary Cardiology
Volume 36, August 2021, Pages 153-168

Detection of congestive heart failure by mitral annular displacement in cats with hypertrophic cardiomyopathy – concordance between tissue Doppler imaging–derived tissue tracking and M-mode ☆

M.B.T. Bach DVM, J.R. Grevsen DVM, M.A.B. Kiely DVM, J.L. Willesen PhD, J. Koch PhD



Detektion af hjertesvigt

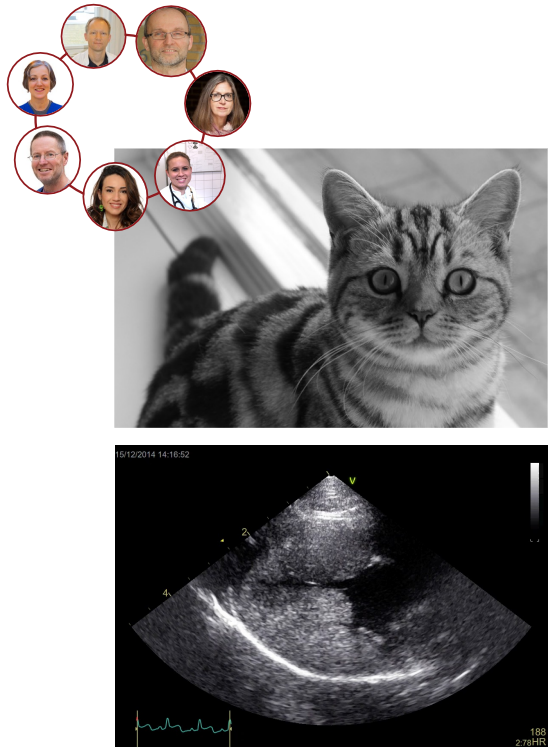
Igangværende forskningsprojekter indenfor HCM

*Der **inkluderes** NFO'er med HCM diagnosticeret før 5 års alderen!*

HCM-genetik i Norsk Skovkat

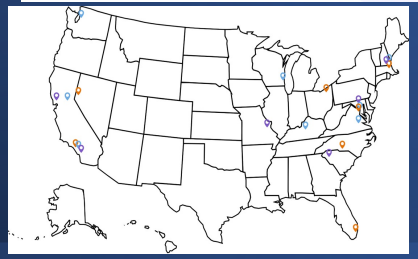
*Der **inkluderes** katte med HCM der skal aflives. Pjece for mere information.*

Myosin funktion & Behandlingsmuligheder

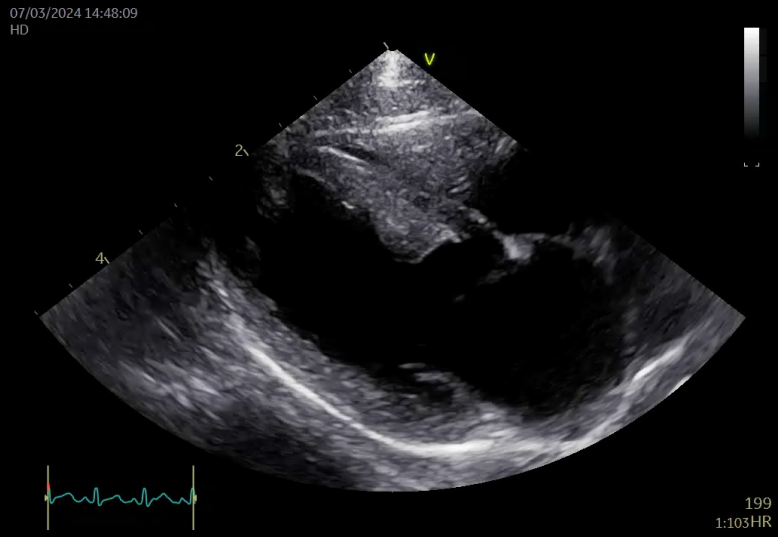


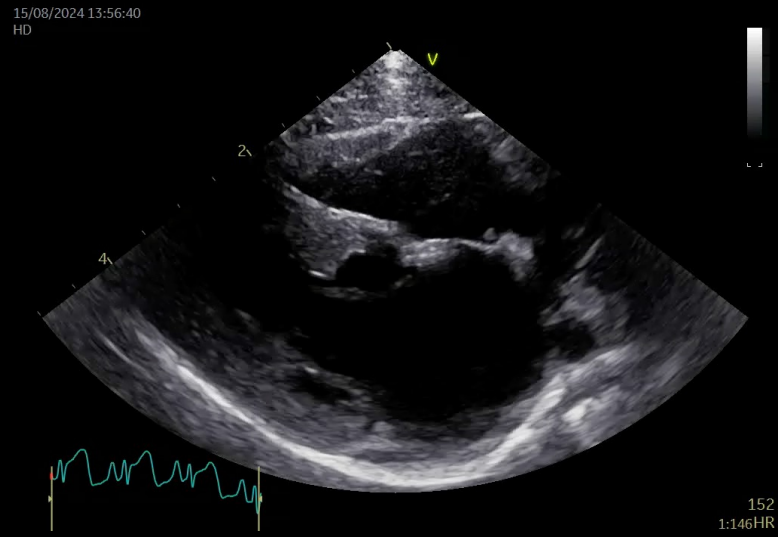
I DK, fokus på familiestudier af katte med tidlig udviklet sygdomspræsentation <3år.

HCM-genetik i British shorthair



Katte med HCM kan registreres online til behandlingsstudie …

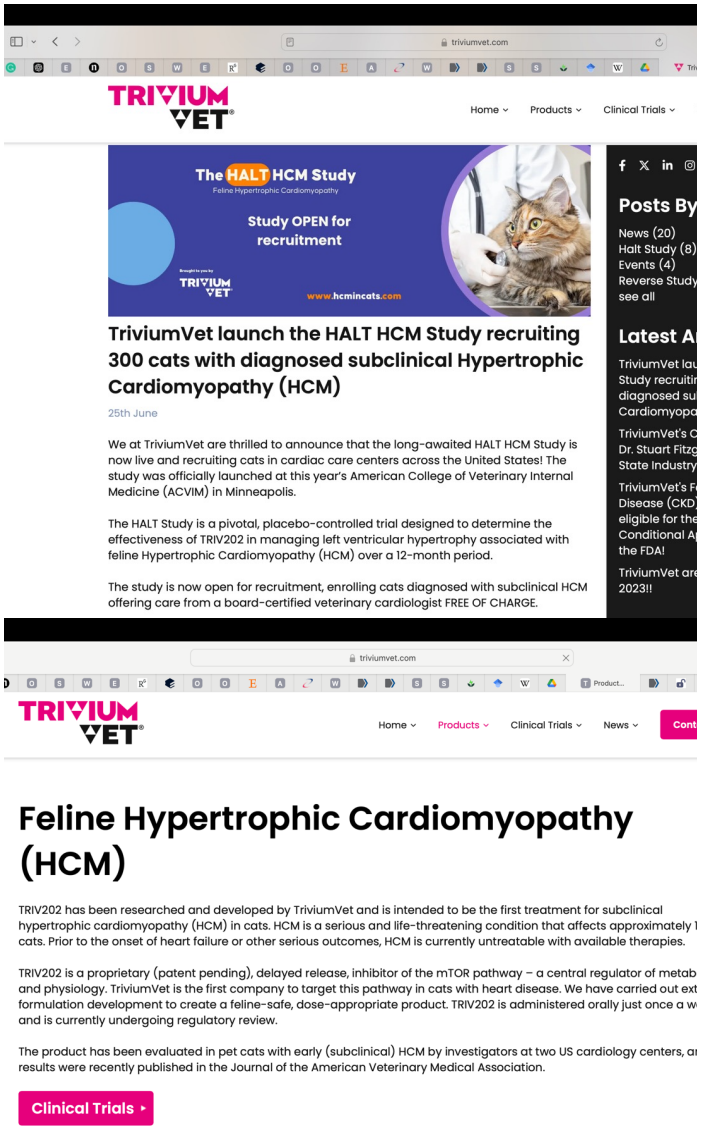




Enhedens navn

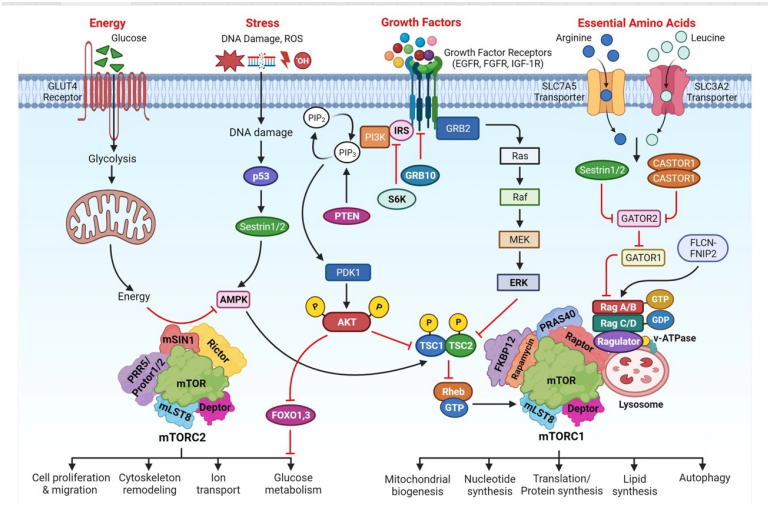
Rapamycin Inhibition of mTORC1

- Rapamycin specifically inhibits **mTORC1** by binding to an intracellular protein called **FKBP12** (FK506 binding protein 12). This complex then interacts with mTORC1, leading to its inhibition. The effects of rapamycin on mTORC1 are as follows:
- **Reduction in protein synthesis**: By inhibiting mTORC1, rapamycin reduces the synthesis of proteins involved in hypertrophy, leading to a suppression of the pathological growth of cardiomyocytes.
- **Restoration of autophagy**: Inhibition of mTORC1 allows autophagy to resume, helping cells clear out damaged proteins and organelles, improving cellular health and mitigating hypertrophic signaling.



mTORC1 & mTORC2 Effects (Multiprotein Complexes)

- Protein synthesis and cell growth
- Lipid biosynthesis
- Inhibition of autophagy
- Metabolic regulation, including enhanced glycolysis and mitochondrial biogenesis
- Inhibition of catabolic processes
- Angiogenesis regulation
- Involvement in cellular senescence and aging
- Cardiac hypertrophy (pathological remodeling in diseases like hypertrophic cardiomyopathy)
- Modulation of inflammatory responses



The major upstream regulators of mTORC1 and mTORC2. Growth factors, amino acids like arginine and leucine, energy from glucose or other sources, cell stresses including DNA damage, and ROS stimulate mTORC1 to modulate various biological processes like mitochondrial biogenesis, nucleotide synthesis, mRNA translation (protein synthesis), lipid synthesis, and autophagy. The growth factors are the main regulators of the mTORC2 to control cell proliferation, migration, cytoskeleton remodeling, ion transport, and glucose metabolism. Created with BioRender.com

Panwar, V., Singh, A., Bhatt, M. *et al.* Multifaceted role of mTOR (mammalian target of rapamycin) signaling pathway in human health and disease. *Sig Transduct Target Ther* **8**, 375 (2023). https://doi.org/10.1038/s41392-023-01608-z

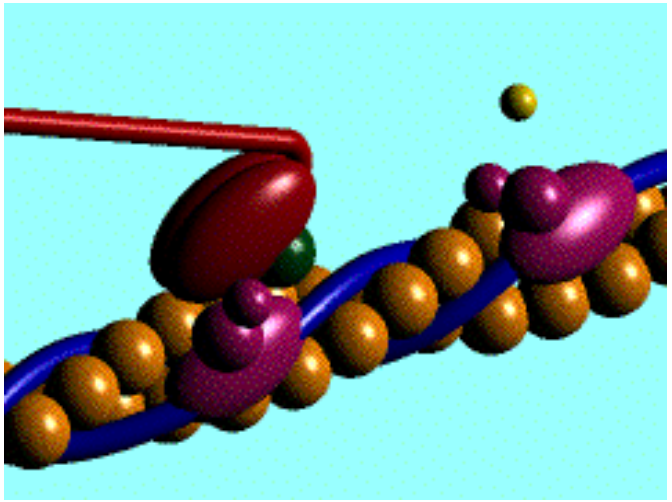
Enhedens navn

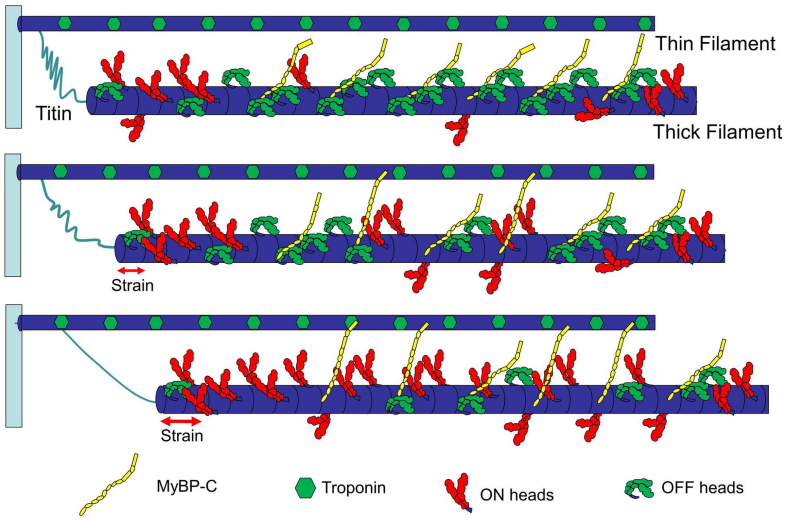
Mavacamten in HCM Treatment

- Pathophysiology: Mutations in sarcomeric proteins (e.g., myosin) lead to excessive contractility, myocardial thickening, and impaired relaxation. Overactive myosin-actin cycling increases ATP consumption and causes dysfunction

Mechanism:

- Binds to β-cardiac myosin, reducing ATPase activity
- Stabilizes myosin in a "super-relaxed" state (SRX), lowering cross-bridge formation and decreasing contractile force

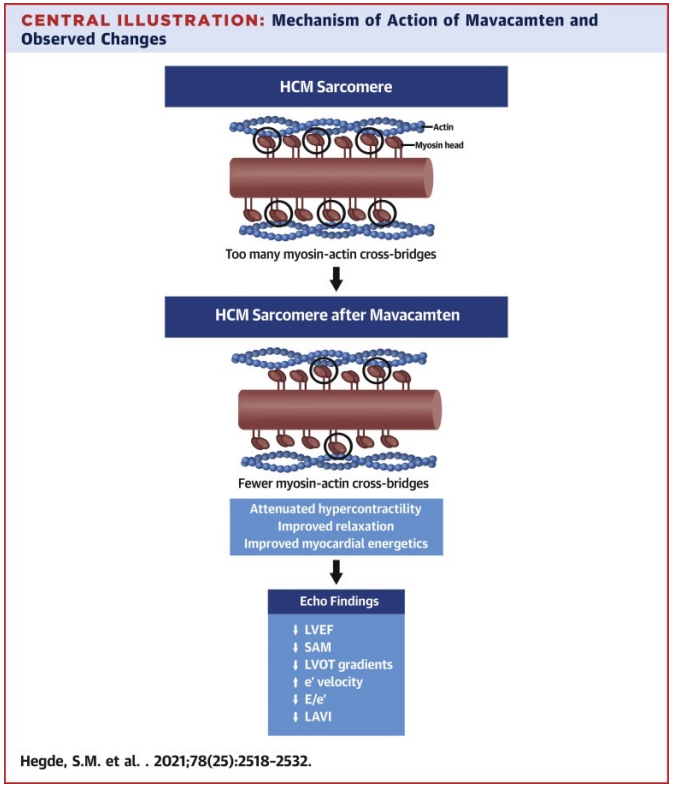




Enhedens navn

Mavacamten in HCM Treatment

- Therapeutic Effects:
- Reduces hypercontractility and ATP consumption.
- Improves diastolic function, reducing symptoms like shortness of breath.
- Relieves LVOT obstruction, improving blood flow.
- May reverse myocardial hypertrophy and reduce arrhythmia risk.
- Clinical Outcomes: Improved symptoms, cardiac function, and reduced hypertrophy over time.



Journal of the American College of Cardiology
Volume 78, Issue 25, 21–28 December 2021, Pages 2518-2532

Difficult to continue to believe that HCM is generally a disease caused by single mutations in the sarcomere (or, in the jargon of genetics, an autosomal dominant mutation). Leading researchers today are investigating broader hypotheses about the causes of HCM (Maron et al. 2019, Harper et al. 2021, Watkins 2021).

https://4hcm.org/updated-thinking-on-hcm-genetics/