CAGH

Companion Animal Genetic Health Conference
8-9 December 2025
Cambridge, UK

CONFERENCE ORGANISERS

Cathryn Mellersh (University of Cambridge)
David Sargan (University of Cambridge)

CONFERENCE WEB DEVELOPER

Ellen Schofield (University of Cambridge)

The CAGH conference organisers would like to thank our generous sponsors, Companion Animal Health and Genetics, without whose support this meeting would not have been be possible.

Companion Animal Health and Genetics is an open access journal focused on advancing the health and welfare of domesticated animals/pets by providing new clinical, genetic, and epidemiological insights.

The cover and pages 2 and 35 of the booklet feature YoYo, the Basset Hound who achieved worldwide fame when the Master of Selwyn College secured permission from the College Council to keep her on college grounds by describing her as "a very large cat". She soon became a favourite presence at Selwyn, appearing in college publicity and winning the affection of students and staff alike.

CONTENTS

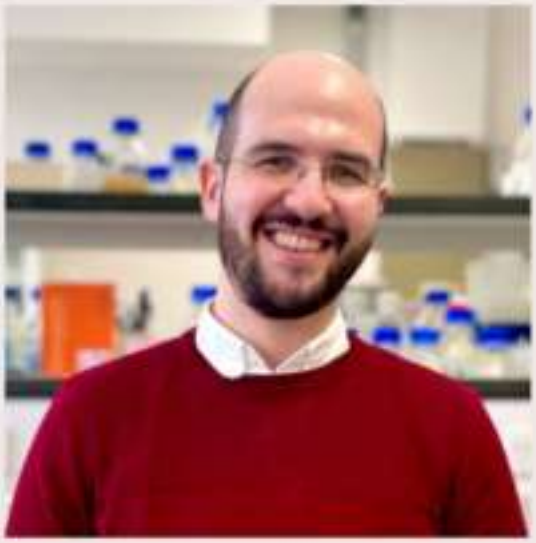
CAGH

Image: Sledge Dog Monument, Scott Polar Research Institute, Cambridge

WELCOME

Welcome to Cambridge and to the 3rd Companion Animal Genetic Health (CAGH) Conference. CAGH is a UK-based scientific meeting dedicated to genetic health issues in companion animals. The conference was created in response to the growing number of researchers across the UK who are investigating the genetics of important health conditions in dogs, cats, horses and other companion species, and the need for an opportunity to meet, present and share findings locally. We aim to bring together as broad a range of work as possible, with contributions from invited speakers, oral presentations and posters. Thank you for joining us, and we hope you find the programme stimulating, supportive and enjoyable.

KEYNOTE SPEAKER



Dr Alex Cagan is a comparative somatic evolutionary genomicist and Assistant Professor at the University of Cambridge, with joint appointments in the Departments of Genetics, Pathology and Veterinary Medicine. His research investigates how somatic mutations accumulate with age in healthy tissues across the animal kingdom, and how species with exceptional longevity or unusual life histories maintain genome integrity.

He led the first comparative study of somatic mutation rates across mammals, showing that these rates scale inversely with lifespan, and has pioneered the use of ultra-accurate sequencing, laser-capture microdissection and long-read technologies to study cellular evolution within organisms. His group now applies these approaches to companion animals, wildlife and diverse vertebrates to understand links between DNA damage, ageing, environment and disease risk.

Before establishing his laboratory in Cambridge, he was a postdoctoral fellow at the Wellcome Sanger Institute with Inigo Martincorena, and completed his PhD at the Max Planck Institute for Evolutionary Anthropology with Svante Pääbo. He is also known internationally for his science communication and illustration, combining visual storytelling with genomics to engage broad audiences.
His work has been recognised with several awards, including the Emerging Leader in Computational Oncology Award and the Walter M. Fitch Prize, and he is committed to open science, mentoring and public engagement.

CONFERENCE VENUES

West Hub

The West Hub is a modern, purpose-built centre at the heart of the University's West Cambridge site. It offers meeting rooms, open collaborative spaces and excellent facilities for teaching and research events. The conference sessions will take place on the second floor, with cafés and social areas available throughout the building.

Selwyn College - Accommodation

Founded in 1882, Selwyn College is a short walk from the West Cambridge site and offers comfortable, traditional Cambridge accommodation in a peaceful setting. The college's gardens, courts and historic architecture provide a quiet base during the conference, with easy access to both the city centre and the West Hub.

Photo: David Iliff, CC BY-SA 3.0

St John's College - Conference Dinner

St John's College, founded in 1511, is one of Cambridge's largest and most iconic colleges. Its historic courts, elegant river views and striking architecture provide a memorable setting for the conference dinner. Attendees should note that the college has several entrances, and access arrangements may vary—please check signage or instructions on the day.

Sunday 7 December

19:30 **Meet and greet at The Eagle pub, Bene't St, Cambridge CB2 3QN.**

The Eagle is one of Cambridge's oldest pubs and the place where, in 1953, Francis Crick announced that he and James Watson had "discovered the secret of life" after proposing the structure of DNA.

Scan to join the conference WhatsApp group for updates and announcements

CAGH

PROGRAMME

Monday 8 December

09:30 **Welcome and announcements**

Session 1 - chaired by Eleanor Raffan

09:45 Profiling the mutational landscape of cancer-associated genes in the domestic cat. **Bailey Francis** - student

10:05 Use of omics to understand catastrophic fracture in Thoroughbred horses. **Esther Palomino Lago**

10:25 A canine form of GAPO syndrome identified in Segugio Italiano dogs caused by a nonsense variant in ANTXR1. **Katherine Stanbury**

10:45 **Coffee break**

Session 2 - chaired by Cathryn Mellersh

11:15 Companion Animal Health and Genetics journal - A short message from our sponsor

11:30 Keynote - **Alex Cagan**
Somatic Genomics in wild and companion animals: insights into cancer and ageing across the tree of life

12:30 **Lunch**

Session 3 - chaired by Liz Murchinson

13:30 Identification of an intronic mutation in the MYO7A gene associated with congenital vestibular disease in Welsh Springer Spaniels. **Bruno Lopes** - student

14:30 A new form of inherited retinal degeneration in the Rough Collie. **Thomas Simon** - student

14:50 Poster introductions

15:30 **Posters and break**

Session 4 - chaired by Debbie Guest

16:30 Research into the genetics of atopic dermatitis: an update. **Oliver Forman**

16:50 Horizontal transfer of nuclear DNA in transmissible cancer. **Kevin Gori**

17:10 Wrap up, free time

18:00 **Dinner**

PROGRAMME

Tuesday 9 December

09:30 **Welcome and announcements**

Session 5 - chaired by Lucy Davison

09:40 Genetic Parameters and Prospects for Within-Breed Selection to Reduce BOAS in Brachycephalic Breeds
Joanna Ilska

10:00 Recurrent mitochondrial horizontal transfer in canine transmissible venereal tumours
Andrea Strakova - student

10:20 The Animal Variant Classification Guidelines: an objective and reproducible tool to assess variant pathogenicity
Bart Broeckx

10:40 **Posters and break**

Session 6 - chaired by Tomas Bergstrom

11:20 Pedigree-tracking improves population monitoring of recessive disease-causing alleles in KC-registered dogs
Ros Craddock - student

11:40 Establishment of a Doxycycline-Inducible SV40Tag System Enabling Controlled Immortalisation and Reprogramming of Canine Fibroblasts
Chandrindu Abeykoon - student

12:00 Multi-modal myocardial tissue characterization in feline patients with hypertrophic cardiomyopathy
Frank van Steenbeek

12:20 **Comfort break**

12:30 Prizes and wrap up

Packed lunch to eat on site or take away

ABSTRACTS - SESSION 1, TALK 1

Profiling the mutational landscape of cancer-associated genes in the domestic cat

Bailey A. Francis[1†], Latasha Ludwig[2,3†], Chang He[4,5†], Melanie Dobromylskyj[6], Christof A. Bertram[7], Heike Aupperle-Lellbach[8,9], Hannah Wong[10], Aiden P. Foster[11], Mark J. Arends[12], Alejandro Suárez-Bonnet[13], Simon L. Priestnall[13], Laetitia Tatiersky[14], Fernanda Castillo-Alcala[15], Angie Rupp[16], Arlene Khachadoorian[2], Eda Parlak[7], Marine Inglebert[4,5], Shevaniee Umamaheswaran[1], Saamin Cheema1, Martin Del Castillo Velasco-Herrera1, Kim Wong[1], Ian C. Vermes[1], Jamie Billington[1], Sven Rottenberg[4,17], Geoffrey A. Wood[2], David J. Adams[1], Louise van der Weyden[1]

†These authors contributed equally to this work.

Cancer is a common cause of morbidity and mortality in domestic cats. Since the mutational landscape of feline tumours remains largely uncharacterised, we performed sequencing of 493 feline tumor-normal tissue pairs from 13 tumor types, focusing on the feline orthologs of ~1,000 human cancer genes. The most frequently mutated gene was TP53, and the most recurrent copy number alterations were loss of PTEN or FAS, or gain of MYC. Through the identification of driver genes, mutational signatures, viral sequences, and tumor-predisposing germline variants, our study provides the first insight into the domestic cat oncogenome. We demonstrate key similarities with the human oncogenome, confirming the cat as a valuable model for comparative studies, and using a 'One Medicine' approach we identify potentially actionable mutations.

1 Wellcome Sanger Institute, Wellcome Genome Campus, Hinxton, Cambridge, Cambridgeshire, CB10 1SA, U.K.
2 Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, Ontario, N1G 2W1, Canada
3 Department of Population Medicine and Diagnostic Sciences, Cornell University, College of Veterinary Medicine, Ithaca, NY, 14853, USA
4 Institute of Animal Pathology, Vetsuisse Faculty, University of Bern, 3012 Bern, Switzerland
5 Graduate School for Cellular and Biomedical Sciences, University of Bern, 3012 Bern, Switzerland
6 Finn Pathologists, One-Eyed Lane, Weybread, Diss, Norfolk, IP21 5TT, UK
7 Institute of Pathology, University of Veterinary Medicine Vienna, Wien, Austria
8 Laboklin GmbH & Co. KG, Laboratory for Clinical Diagnostics, 97688 Bad Kissingen, Germany
9 Institute of Pathology, School of Medicine, Technical University of Munich, Trogerstrasse 18, 81675 Munich, Germany
10 Department of Veterinary Medicine, University of Cambridge, Cambridge, Cambridgeshire, CB3 0ES, UK
11 Bristol Veterinary School, University of Bristol, Langford House, Langford, North Somerset, BS40 5DU, UK
12 University of Edinburgh, Edinburgh Pathology, Centre for Comparative Pathology, CRUK Scotland Centre, Institute of Genetics and Cancer, Crewe Road South, Edinburgh, EH4 2XR, Scotland
13 Department of Pathobiology and Population Sciences, Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield AL9 7TA, UK
14 VETPATH Canada, Guelph, Ontario, N1G 2W1, Canada
15 Tāwharau Ora-School of Veterinary Science, Massey University, Palmerston North 4474, New Zealand
16 School of Biodiversity, One Health & Veterinary Medicine, University of Glasgow, Glasgow, G61 1QH, Scotland
17 Bern Center for Precision Medicine and Cancer Therapy Resistance Cluster, Department for BioMedical Research, University of Bern, 3008 Bern, Switzerland

ABSTRACTS - SESSION 1, TALK 2

Use of omics to understand catastrophic fracture in Thoroughbred horses

Esther Palomino Lago[1], Debbie Guest[1]

Catastrophic fractures are the most common type of injury are the main cause of euthanasia on the racecourse. Bone fracture is a complex condition caused by environmental and genetic factors.
Using a polygenic risk score (PRS) for fracture we generated induced pluripotent stem cells (iPSCs) from horses at high and low genetic risk and differentiated them into osteoblasts-like cells. RNA sequencing performed on the osteoblasts revealed 112 differentially expressed genes (DEGs).
Whole Genome Sequencing (WGS) on 7 catastrophic fracture cases and 7 controls identified 12,224,941 DNA variants across all genomes. These were filtered based on their segregation between cases and controls, predicted consequence, location in the genome, and minor allele frequency (MAF) across breeds. 474 candidate variants were then genotyped using a custom-designed MassARRAY (Agena Bioscience) in 155 fracture cases and 206 controls. This identified 25 variants significantly associated with fracture, 11 were upstream of six different genes and 14 were mis-sense in 10 different genes. 64% of variants were located on chromosome 18, which was previously found to be associated with fracture, but no variants associated with the DEGs. Four of the variants were protective and 21 conferred risk. Four of the mis-sense variants were in ICA1L and epistatic analysis revealed that they interact with all the significant upstream variants. Three of these variants are predicted to have a deleterious effect on the ICA1L protein.
Primary osteoblasts from Thoroughbred were isolated and used to generate immortalized osteoblast by constitutive expression of human telomerase reverse transcriptase (hTERT) and Simian Vacuolating Virus 40 large T antigen (SV40LT) to provide an in vitro tool to study gene and variant function. Knockdown of ICA1L reduced cell viability and bone formation demonstrating a novel role for this gene in osteoblasts. Luciferase assays performed in immortalized osteoblasts revealed significant differences in reporter gene expression between alleles when analysing small regions (<120bp) flanking some of the variants.
Assay for Transposase-Accessible Chromatin (ATAC) sequencing on iPSC-derived osteoblasts from horses at high and low genetic risk and in immortalized osteoblasts during osteogenic culture is currently being performed to combine with our RNA seq and WGS to help to prioritize further variants for follow up. This will enable us to refine our PRS to increase its accuracy in the identification of high and low risk horses to allow informed breeding and targeted monitoring of bone health.

1 Royal Veterinary College, UK

ABSTRACTS - SESSION 1, TALK 3

A canine form of GAPO syndrome identified in Segugio Italiano dogs caused by a nonsense variant in ANTXR1

Katherine Stanbury [1], Maria Paola Cassarani [2], Ellen Schofield [1], Bryan McLaughlin [1], Cathryn Mellersh [1]

GAPO syndrome is a rare autosomal recessive condition that to date has only been reported in humans. GAPO is the synonym that represents the four primary features of the condition; Growth retardation, Alopecia, Pseudoanodontia and Ocular atrophy. It is a severe syndrome which reduces the life expectancy of affected individuals, and is caused by pathogenic variants in the Anthrax Toxin Receptor 1 (ANTXR1) gene. This study reports a canine form of GAPO syndrome observed in a litter of smooth hair Segugio Italiano (SI) dogs. Affected dogs, examined at 21 months old by a veterinary ophthalmologist, presented with the core GAPO phenotype, short-limbed dwarfism, craniofacial abnormalities, alopecia with mantle lichenization, pseudoanodontia, and ocular abnormalities that included open angle glaucoma. Whole genome sequence analysis of two affected puppies and the dam identified a nonsense variant c.505C>T in ANTXR1 which introduces an early stop codon in exon 7 and is predicted to truncate the protein by 395 amino acids. All affected SI (n=3) were homozygous and the dam and unaffected littermate heterozygous for the variant. It was absent from the Dog10K VCF, our internal genome data bank of >450 samples and 24 unaffected and unrelated SI. ANTXR1 gene expression in corneal and retinal tissues of a GAPO-affected SI was significantly reduced compared to control samples suggesting that the aberrant transcript (or transcripts) undergo partial degradation via nonsense-mediated mRNA decay.

1 Canine Genetics Centre, Department of Veterinary Medicine, University of Cambridge, UK
2 Centro Veterinario Oculistico Argo, Ancona, Italy

ABSTRACTS - SESSION 2

A short message from our sponsor

BMC *Companion Animal Health and Genetics*

COMPANION ANIMAL GENETIC HEALTH CONFERENCE (CAGH), CAMBRIDGE, 2025

Call for Collection Ideas

The 3rd biennial CAGH conference will be running a Collection.

- We invite contributions related to the conference.
- This is an invite-only Collection.
- Topics can include outside the scope of conference.

What are the benefits from a Collection?

Journal articles are more **cited and downloaded** on average than standard journal articles

Collections are a **unique, timely** opportunity for authors to contribute on a specific topic

Build **relationships** with the research community

Guest Editors of collections gain publishing experience by handling manuscripts and collaborating with the journal

Why Publish with Companion Animal Health and Genetics journal?

Rapid peer review and publication of your research

Open access publication ensures articles can be easily discovered, accessed, and shared

Has waivers (~10) available to offer based on first come basis

What is a collection?

A Collection is a timely grouping of articles on specific areas of research within the scope of a journal, handled by Guest Editor(s). Articles published in a Collection goes through rigorous ethics & integrity checks to ensure high quality.

Journal Metrics

Speed
128 days submission to accept (Median days, 2024)

Downloads
223.1k (2024)

What are the roles of a Guest Editor?

- **Suggest the Theme:** Propose/define the scope of the collection, highlighting timely and significant issues in [subject area].
- **Manage the Review Process:** Work with authors and peer reviewers to ensure the highest quality of published work.
- **Share:** Where possible, help to share the collection with relevant scientists in your network.

Guest Editors are supported by the Editor-in-Chief and the Collections team.

ABSTRACTS - SESSION 3, TALK 1

Identification of an intronic mutation in the MYO7A gene associated with congenital vestibular disease in Welsh Springer Spaniels

Bruno Lopes[1,2], Ellen Schofield[1], Katherine Stanbury[1], Louise Pettitt[1], Paul Freeman[3], Cathryn Mellersh[1]

Congenital peripheral vestibular disorder is rare in dogs, and has only previously been described in Dobermann Pinscher, English Cocker Spaniel, and German Shepherd breeds. Genetic mutations in the MYO7A and PTPRQ genes have been previously reported as associated.

Two puppies in a litter of nine Welsh Springer Spaniels (WSS) showed circling and rolling from one week of age. Examination of both puppies revealed peripheral vestibular signs, and investigations with magnetic resonance imaging at two months old did not reveal any structural abnormality. Additionally, one other puppy from a litter of five, which was two years old and closely related to the affected puppies, was reported to have had similar signs which stabilised over time. An autosomal recessive genetic causal variant was hypothesised.

DNA Samples were obtained from both affected families (parents and affected and unaffected progeny) and whole genome sequencing (WGS) was undertaken. Pipeline variant filtering, profiling and parentage analysis, analysis for runs of homozygosity, genome-wide association studies, and structural variant detection were performed.

Following thorough analysis, including filtering for correct segregation, an intronic, single nucleotide variant (SNV) in the MYO7A gene was identified. Splice site analysis (SpliceAI) was suggestive of a possible new donor splice site gain.

A novel intronic SNP mutation associated with canine congenital vestibular disease has been identified. Further in-silico and RNA analysis is necessary to confirm the RNA expression of the MYO7A gene in affected and non-affected dogs as well as possible protein stability.

1 The Canine Genetics Centre, University of Cambridge, Cambridge, UK
2 Southfields Veterinary Specialists, Basildon, UK
3 Queens Veterinary School Hospital, University of Cambridge, Cambridge, UK

ABSTRACTS - SESSION 3, TALK 2

A new form of inherited retinal degeneration in the Rough Collie

Thomas Simon[1], Suvi Mäkeläinen[1], Daniel Goncalves[2], Björn Ekesten[2], Tomas F. Bergström[1]

Inherited retinal degenerations (IRDs) are a diverse group of progressive diseases that cause visual impairment and blindness in both humans and dogs. They represent a heterogeneous group of genetic disorders, characterized by variable ages of onset, rates of progression, and modes of inheritance. Most of the IRDs are inherited in an autosomal recessive manner, although autosomal dominant and X-linked forms have also been described. To date, 367 causative genes have been identified in humans, while only about 30 have been reported in dogs.

A novel form of IRD has recently been observed in Rough Collies. A genetic variant (22 bp insertion) in the gene RD3 regulator of GUCY2D (RD3) has previously been described to cause rod–cone dysplasia type 2 (rcd2) in the breed. However, the novel IRD cannot be explained by the RD3 insertion or by any of the known genetic variants in dogs. To investigate the genetic cause, we performed family-sequencing of five dogs. This included both the unaffected parents as well as three offspring, of which two were affected males and one unaffected female.

After variant calling and annotation, conditional filtering was conducted under the assumption of either an autosomal recessive or an X-linked mode of inheritance. To further restrict the number of candidate variants, we filtered away common variation using publicly available data. The possible impact of the identified candidate variants in the coding sequence was predicted using SIFT and PolyPhen-2. In addition, variants were further investigated using PhyloP conservation scores and manual curation based on gene function. The results of this ongoing study will be presented.

1 Department of Animal Biosciences, Swedish University of Agricultural Sciences, Uppsala, Sweden
2 Department of Clinical Sciences, Swedish University of Agricultural Sciences, Uppsala, Sweden

ABSTRACTS - SESSION 4, TALK 1

Research into the genetics of atopic dermatitis: an update

Oliver Forman[1], Abigail Kerr[2], Jamie Freyer[1], Jason Huff[1], Luke Bawazer[1], Michelle Daya[1], Rebecca Chodroff Foran[1]

Canine atopic dermatitis (CAD) is a common inflammatory skin condition in dogs. It is a lifelong issue that poses a significant welfare concern due to the chronic skin discomfort and pruritus (itching) experienced by affected animals. Access to a large population of dogs genotyped on a medium-density single-nucleotide polymorphism array through commercial Wisdom Panel testing, along with their linked clinical records, allows large-scale, highly powered genome-wide association studies (GWAS) to be performed. In a large CAD GWAS using over 28,000 dogs, a signal on canine chromosome 38 was identified, with subsequent whole-genome resequencing revealing a compelling splice donor variant in signaling lymphocytic activation molecule 1 (SLAMF1), a transmembrane receptor with important functions in immune cells. The SLAMF1 discovery and study approach will be discussed, along with insights into more recent analyses at Wisdom.

1 Wisdom Panel, Mars Science and Diagnostics, Fountain Valley, CA, US
2 Veterinary Specialists Scotland, Linnaeus Veterinary Limited, Mars Veterinary Health, Livingston, UK

ABSTRACTS - SESSION 4, TALK 2

Horizontal transfer of nuclear DNA in transmissible cancer

Kevin Gori[1]

To date, numerous infectious cancers have been discovered in the wild. The oldest known, Canine Transmissible Venereal Tumour (CTVT), is a disease that infects primarily free roaming dogs.
Originating as a tumour in an Asian dog over six thousand years ago, for millennia CTVT has spread among dog populations as a contagious allograft. The CTVT cancer cells that infect modern dogs are direct descendants of the transformed cells of the progenitor animal and carry clonal copies of its genome. However, it is possible that a tumour cell can acquire DNA from normal cells non-clonally, through a process of horizontal gene transfer.
By exploiting the increased genetic diversity between cancer and host that is characteristic of transmissible cancers, we have recently identified the signature of horizontal gene transfer from host to tumour in a lineage of modern tumour samples. Using genomic sequence analysis, cytology and population genetics, we trace the source of this signal to a transfer of a highly rearranged fragment of DNA that was incorporated by CTVT from a host dog that lived in the Middle East over two thousand years ago.

1 University of Cambridge, UK

ABSTRACTS - SESSION 5, TALK 1

Genetic Parameters and Prospects for Within-Breed Selection to Reduce BOAS in Brachycephalic Breeds

Joanna Ilska[1], Fern McDonnell[1], Jane F. Ladlow[2,3]

Brachycephalic Obstructive Airway Syndrome (BOAS) is among the most serious welfare concerns affecting companion dogs. Owners often underestimate the severity of clinical signs, while the same morphological traits that predispose brachycephalic breeds to BOAS are also those most valued by prospective puppy buyers. As many owners and breeders remain committed to these breeds, and given the limited phenotypic variation in craniofacial morphology within the most extreme populations, there has been continuing debate about whether meaningful within-breed improvement in BOAS is achievable. To date, only limited numbers of phenotyped dogs have been available, constraining accurate estimation of genetic parameters.

We analysed data from 4,301 dogs across three extreme brachycephalic breeds assessed under the Respiratory Function Grading Scheme (RFGS), where clinical grades reflect the severity of BOAS. Genetic parameters were estimated using univariate animal models incorporating full pedigree relationships to partition phenotypic variance into additive genetic and environmental components. Estimated breeding values (EBVs) and their accuracies were subsequently derived for all individuals in pedigree. Population structure was explored within each breed using principal component analysis (PCA) based on pedigree-defined relationships.

In this largest dataset to date, the prevalence of BOAS was estimated at 15–20%, substantially lower than previously reported. Significant additive genetic variance was detected for RFGS grade, with heritability estimates ranging from 0.21 to 0.45 across breeds, indicating that BOAS susceptibility is moderately heritable and therefore amenable to selection. However, EBV accuracies were generally low, reflecting limited uptake of RFGS testing. PCA revealed evidence of population substructure within breeds. Dogs bred outside the official breed standard, here defined as those with non-standard coat colours, were under-represented among tested animals, suggesting sampling bias and uneven distribution of selection opportunities across subpopulations.

These findings demonstrate that genetic progress to improve respiratory health is feasible through within-breed selection, provided participation in health testing is expanded. Greater engagement across all breeding lines will be essential to enhance EBV accuracy and ensure that selection for improved airway function delivers meaningful welfare gains in these popular but high-risk breeds.

1 The Kennel Club, UK
2 Department of Veterinary Medicine, University of Cambridge, Cambridge, UK
3 Granta Veterinary Specialists Referrals, Linton, UK

ABSTRACTS - SESSION 5, TALK 2

Recurrent mitochondrial horizontal transfer in canine transmissible venereal tumours

Andrea Strakova[1], Adrian Baez-Ortega[1,2], Thomas J. Nicholls[3,4], Máire Ní Leathlobhair[1], Kevin Gori[1], Jinhong Wang[1], Patrick F. Chinnery[5], Maria Falkenberg[3], Claes M. Gustafsson[3], Elizabeth P. Murchison[1]

One unusual but particularly valuable natural model for studying mitochondrial DNA (mtDNA) dynamics is the canine transmissible venereal tumour (CTVT). CTVT is a contagious cancer affecting dogs, which spreads by the transfer of living cancer cells during mating, causing genital tumours. Although the CTVT nuclear genome is clonal and represents the DNA of the founder dog that lived several thousand years ago, CTVT mtDNAs are polyclonal and were acquired periodically by horizontal transfer from transient hosts. Capture of mtDNAs by CTVT cells results in a natural competition assay, whereby the relative fitness of diverse pairs of canine mtDNA haplotypes is assessed in vivo. We aimed to characterise mtDNA horizontal transfer dynamics within the CTVT population. We used low coverage whole genome DNA sequencing to survey mtDNAs in over one thousand CTVT tumours and matched hosts. The genetic analysis highlighted a single canine mtDNA haplotype, which has repeatedly colonised CTVT cells due to 'selfish' positive selection. Although CTVT is considered a biological oddity, its periodic uptake and juxtaposition of mitochondrial haplotypes provide broad and unexpected insights into mammalian mitochondrial dynamics.

1 Transmissible Cancer Group, Department of Veterinary Medicine, University of Cambridge, Cambridge, UK
2 Bivalve Transmissible Neoplasia Group, Department of Zoology, University of Cambridge, UK
3 Department of Medical Biochemistry and Cell Biology, University of Gothenburg, Gothenburg, Sweden
4 Wellcome Centre for Mitochondrial Research, Newcastle University, Newcastle Upon Tyne, UK
5 MRC-Mitochondrial Biology Unit & Department of Clinical Neurosciences, University of Cambridge, Cambridge, UK

ABSTRACTS - SESSION 5, TALK 3

The Animal Variant Classification Guidelines: an objective and reproducible tool to assess variant pathogenicity

Iris Casselman[1], Carlotta Ferrari[2], Marie Abitbol[3], Danika Bannasch[4], Jerold Bell[5], Caroline Dufaure de Citres[6], Carrie J. Finno[7], Jessica J. Hayward[8], Jens Häggström[9], Jason T. Huff[10], Tosso Leeb[11], Ingrid Ljungvall[9], Maria Longeri[2], Leslie A. Lyons[12], Marcela Martinez[13], Cathryn Mellersh[14], Frank W. Nicholas[15], Åsa Ohlsson[16], Pascale Smets[17], Maria G. Strillacci[2], Imke Tammen[15], Frank G. van Steenbeek[18], **Bart J.G. Broeckx[1,19]**

Until recently, due to the lack of standardized guidelines tailored for veterinary use, the evaluation of genetic variant pathogenicity for single-gene diseases was based on a subjective personal interpretation of the presented evidence, which has led to ambiguous interpretations. Assessing the pathogenicity is however vital, both on a population and individual scale. At the population level, breeding decisions based on invalid DNA tests can lead to the incorrect inclusion or exclusion of animals and compromise the long-term health of a population, and at the level of the individual animal, lead to incorrect treatment and even life-ending decisions. With the publication of the animal variant classification guidelines (AVCG), a more objective approach became available. Variants are evaluated based on twenty-three criteria and labeled as pathogenic, likely pathogenic, variant of uncertain significance, likely benign or benign. In two subsequent studies, we have evaluated the accuracy of these new guidelines, as well as the reproducibility of decisions on the scope and the final label.

To assess the accuracy of the guidelines, methods were developed to produce a "true" disease-causing variant dataset (n = 53), as well as a "benign" variant dataset (n = 47). This led to the first dataset where classifications by panelists were compared with the "truth". For reproducibility, a second set of 150 published likely causal variants for single-gene diseases from three species (dog, cat, horse) was independently and blindly assessed by three different reviewers, each applying the same AVCG. To evaluate agreement, the classifications of each individual reviewer were compared pairwise, leading to a total of 450 pairwise comparisons. In the first study, 92% of the pathogenic variants were accurately classified with AVCG. In the second study, there was an overall agreement of 93% for decisions on the scope, i.e. whether they fit the inclusion criteria to allow evaluation with AVCG. More importantly, the exact reproducibility was 65% for the pathogenicity classification and this increased further to 83% clinically important agreement. While a direct comparison of the reproducibility with human literature is not possible for the scope, the reproducibility on pathogenicity classification is in line with reports using the human American College of Medical Genetics and Genomics and Association for Molecular Pathology guidelines for human variants. Factors that might improve reproducibility include automated label calculation to avoid tabulation errors, and additional clarification of criteria.

Overall, with the AVCG, there is now a tool tailored for variant classification in domestic animals that has been demonstrated to be highly accurate and reproducible within current expectations. To ensure easy access and quick updating of labels whenever necessary, the labels will also be included in the Online Mendelian Inheritance in Animals variant table.

ABSTRACTS - SESSION 5, TALK 3

Continued...

1 Laboratory of Animal Genetics, Department of Veterinary and Biosciences, Faculty of Veterinary Medicine, Ghent University, Merelbeke-Melle, Belgium
2 Department of Veterinary Medicine and Animal Science, University of Milan, Lodi, Italy
3 Univ Lyon, VetAgro Sup, Marcy-l'Etoile, France and Institut NeuroMyoGène INMG-PGNM, CNRS UMR5261, INSERM U1315, Faculté de Médecine, Rockefeller, Université Claude Bernard, Lyon, France
4 Department of Population Health and Reproduction, School of Veterinary Medicine, University of California Davis, Davis, CA, USA
5 Department of Clinical Sciences, Cummings School of Veterinary Medicine, Tufts University, N. Grafton, MA, USA
6 Antagene - Animal Genomics Laboratory, La Tour de Salvagny, France
7 Department of Population Health and Reproduction, University of California Davis School of Veterinary Medicine, Davis, USA
8 Department of Biomedical Sciences and Cornell Veterinary Biobank, College of Veterinary Medicine, Cornell University, Ithaca, USA
9 Department of Clinical Sciences, Faculty of Veterinary Medicine and Animal Science, Swedish University of Agricultural Sciences, Uppsala, Sweden
10 Wisdom Panel, Mars Petcare Science & Diagnostics, Fountain Valley, USA
11 Institute of Genetics, Vetsuisse Faculty, University of Bern, Bern, Switzerland
12 Department of Veterinary Medicine and Surgery, College of Veterinary Medicine, University of Missouri, Columbia, USA
13 Applied Genetics Laboratory, Sociedad Rural Argentina, Buenos Aires, Argentina
14 Canine Genetics Centre, Department of Veterinary Medicine, University of Cambridge, Cambridge, UK
15 Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, Camden, Australia
16 Department of Animal Biosciences, Faculty of Veterinary Medicine and Animal Science, Swedish University of Agricultural Sciences, Uppsala, Sweden
17 Small Animal Department, Faculty of Veterinary Medicine, Ghent University, Merelbeke-Melle, Belgium
18 Department of Clinical Sciences, Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands
19 Centre for Clinical Genetics of Companion Animals, Faculty of Veterinary Medicine, Ghent University, Merelbeke-Melle, Belgium

ABSTRACTS - SESSION 6, TALK 1

Pedigree-tracking improves population monitoring of recessive disease-causing alleles in KC-registered dogs

Rosalind Craddock[1], Mateja Janes[1], Cathryn Mellersh[2], Joanna Ilska[3], Pamela Wiener[1], Steph Smith[4], Gregor Gorjanc[1]

Most monogenic diseases in dogs are recessive. Where possible, dog owners use genetic tests to identify dogs that carry specific disease-causing alleles and the Kennel Club, UK (KC) records the result against the dog's individual pedigree as either "clear" (homozygous wild), "carrier" (heterozygous), or "affected" (homozygous mutant). These results, along with "hereditary status" (an assigned offspring genotype where both parents are homozygous for either the normal or disease-causing allele), are used to monitor changes in allele frequencies over time. However, this only considers a proportion of the pedigree population since not all dogs have genetic test information. This work used probabilistic inference for pedigree-based tracking of alleles for two monogenic recessive eye diseases in five KC-registered breeds. With this method we (1) estimated genotype probabilities for all individuals in the pedigree using available genetic test information and family relationships, (2) estimated allele frequencies by year of birth, and (3) examined the quality of the estimated genotype probabilities using leave-one-out cross-validation and simulations.

Results show that disease-causing allele frequencies were underestimated after the introduction of a genetic test when considering only the proportion of the pedigree with either genetic test information or hereditary status. With the estimated genotype probabilities, the allele frequencies were up to 0.092 higher per year after genetic test introduction. This reflects probabilistic inference's ability to take into account the entire pedigree, thereby diluting the reporting bias toward clear dogs.

The quality of the estimated genotype probabilities varied across breeds, largely depending on population allele frequency, with accuracies ranging from moderate (0.47) to high (0.67) as measured by Pearson correlation. Simulations indicated a tendency of the probabilistic inference to underestimate the disease-causing allele frequency prior to the 2000s due to only recent availability of genetic tests.

Overall, this study shows improved population monitoring of recessive disease-causing alleles by estimating genotype probabilities for all pedigree individuals, aiding better breeding decisions to reduce disease incidence in KC-registered dogs. Nonetheless, estimates were limited by the uptake of genetic testing, highlighting the need for continued genetic testing of dogs.

1 The Roslin Institute, University of Edinburgh
2 The Canine Genetics Centre, University of Cambridge
3 The Kennel Club, UK
4 Global Agriculture and Food Systems, University of Edinburgh

ABSTRACTS - SESSION 6, TALK 2

Establishment of a Doxycycline-Inducible SV40Tag System Enabling Controlled Immortalisation and Reprogramming of Canine Fibroblasts

Chandrindu Abeykoon, Melany Jackson, Louise Adamson, Tom Watson, Stephen Meek, Amy Findlay, Gura Bergkvist, Tom Burdon, Jeffrey J. Schoenebeck

Induced pluripotent stem cells (iPSCs) are multifaceted tools used in regenerative medicine, drug discovery and disease modelling. However, the generation of canine iPSCs has not been efficient compared with their human counterparts. Ethical constraints in obtaining embryonic material and technical challenges in maintaining adult somatic cells in culture complicate canine iPSC generation. Furthermore, compared with well-established human iPSC systems, canine iPSC generation often shows lower reprogramming efficiency, incomplete pluripotency, continual dependence on reprogramming transgenes, and poor directed differentiation outcomes.

To enable efficient reprogramming of canine somatic cells while reducing dependence on limited primary material, we immortalised canine fibroblasts using SV40 Large T antigen (SV40Tag). Previous studies on other species show that SV40Tag enhances reprogramming efficiency by antagonising p53 and Rb pathways, suppressing stress-induced apoptosis, and extending the proliferative lifespan of somatic cells.

In our study, a doxycycline-inducible SV40Tag system enhanced the proliferative capacity of canine fibroblasts, allowing them to be maintained for extended passages in continuous culture. Two immortalised fibroblast cell lines were established, and their transfection efficiencies were evaluated using a GFP reporter plasmid, yielding an average efficiency of approximately 88%.

To assess the dependence of SV40Tag-mediated immortalisation, doxycycline was withdrawn from the culture medium, and cells were monitored over 96 hours. A significant reduction in proliferation was observed as early as 12 hours after doxycycline removal. Moreover, a decline in SV40Tag expression and a gradual increase in the p53 downstream marker MDM2 gene expression were observed. These findings confirm that SV40Tag activity can be modulated in this system.

Abstract continued on next page...

The Roslin Institute and The Royal (Dick) School of Veterinary Studies, University of Edinburgh, UK

ABSTRACTS - SESSION 6, TALK 2

Continued...

To explore the transcriptomic landscape of reprogrammed cells and evaluate the effects of SV40Tag in propagated canine iPSCs, an immortalised cell line was reprogrammed in parallel with primary fibroblasts using a doxycycline-inducible PiggyBac system carrying eight transcription factors, each with three technical replicates. Following transfection, iPSC-like colonies emerged by Day 11. Cell pellets from untransfected (Day 0) and transfected cells (Days 2, 4, 6, 8 and 11) were collected and processed for bulk RNA sequencing, and data were analysed using the nf-core RNA-seq and differential abundance workflows. Preliminary analyses revealed a clear and consistent pattern highlighting the strong transcriptional impact of SV40Tag-mediated immortalisation. Principal component analysis (PCA) showed that PC1 (76.4%) accounted for most variance, distinguishing SV40Tag-immortalised cells from primary fibroblasts. PC2 (8.1%) captured shifts linked to reprogramming, separating earlier from later stages in both cell lines. The heatmap of top variable genes showed two distinct clusters, with SV40Tag-immortalised samples displaying upregulation of genes involved in cell-cycle regulation and DNA replication, while primary fibroblasts exhibited higher expression of extracellular matrix associated genes.
To further investigate these differences, datasets from Day 0 and Day 2 are under analysis to investigate the molecular pathways influenced by SV40Tag during early reprogramming, focusing on p53 downstream targets, apoptotic and cell cycle regulators, and the expression of transgenes and endogenous pluripotency markers. Data from a second biological replicate are currently being generated to strengthen these analyses.

ABSTRACTS - SESSION 6, TALK 3

Multi-modal myocardial tissue characterization in feline patients with hypertrophic cardiomyopathy

Talitha C F Spanjersberg[1,2,5*], Alma H Hulsman[7*], Guy C M Grinwis[4], Babette Janssen[2,3], C Nina van der Wilt[3,4], Rogier J A Veltrop[2,3,5,6] Christian Snijders Blok[2,3], Claudia Rozendom[1], Paul Besseling[2,7], Jolanda van der Velden[8], Pim van der Harst[3], Magdalena Harakalova[2,3,5,6#], **Frank G van Steenbeek[1,2,3#]**

Hypertrophic cardiomyopathy (HCM) is a naturally occurring disease in domestic cats displaying genetic, clinical, and pathological similarities to humans, highlighting their value as a biologically pertinent but as of yet underutilized model. While feline HCM affects 14.7% of cats compared to 0.2% of humans, systematic studies linking cardiac remodeling with clinical outcomes are lacking. We aimed to comprehensively characterize feline HCM in a cross-sectional cohort using advanced histological and transcriptomic approaches to bridge this gap.

We analysed midventricular cardiac sections from 37 cats with arterial thromboembolism (ATE), congestive heart failure (CHF) without ATE, or no documented heart disease. Semi-automated machine and deep learning image analysis quantified fibrosis, adipocyte infiltration, vascular morphology, and nuclear shape. Additionally, we performed Nanopore long-read direct RNA sequencing to link histological features with transcriptomic profiles. Cats with ATE showed increased myocardial fibrosis, whereas CHF cats did not differ significantly from controls, suggesting distinct remodeling pathways. Nuclear hypertrophy was prominent in CHF but not in cats with ATE. Fibrosis correlated with suppressed mitochondrial and contractile gene expression. Adipocyte infiltration mirrored patterns seen in human hearts and inversely correlated with immune-related gene expression, indicating potential anti-inflammatory roles.

This study establishes a high-resolution, multimodal framework for investigating hypertrophic cardiomyopathy in the domestic cat. By combining scalable histological quantification with transcriptomic and targeted gene expression analysis, we demonstrate the translational relevance of this model and its potential to translate basic research into clinical applications.

1 Department Clinical Sciences, Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands
2 Regenerative Medicine Center Utrecht (RMCU), UMC Utrecht, University of Utrecht, The Netherlands
3 Department of Cardiology, Division Heart & Lungs, UMC Utrecht, University of Utrecht, Utrecht, The Netherlands
4 Department Biomolecular Health Sciences, Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands
5 Department of Biochemistry, CARIM, Maastricht University, Maastricht, The Netherlands
6 Netherlands Heart Institute, Holland Heart House, Utrecht, The Netherlands
7 Department of Nephrology and Hypertension, UMC Utrecht, University of Utrecht, 3584 CX Utrecht, The Netherlands
8 Department of Physiology, Amsterdam Cardiovascular Sciences, Amsterdam University Medical Center, Amsterdam, The Netherlands
* and # equal contribution

POSTER PRESENTATION

Amplification of ribosomal DNA in the Canine Transmissible Venereal Tumour

Freddie McNay, Kevin Gori, Elizabeth Murchison

Transmissible cancers spread between individuals via direct transfer of cancer cells, enabling persistence beyond the lifetime of their original host. The Canine Transmissible Venereal Tumour (CTVT), which arose approximately 6000 years ago, is the oldest known somatic cell lineage and provides a powerful model for studying somatic evolution over extended timescales. Since diverging from their last common ancestor, CTVT has diversified into multiple clades, each with distinct genetic features.

Tandem repeat arrays are ubiquitous but unstable genomic regions that represent one of the largest sources of variation in eukaryotic genomes. Repeat instability increases in both cancer and aging, making CTVT an ideal model to investigate long-term tandem repeat dynamics during somatic evolution, offering insight into how repeats accumulate, stabilize, or diversify over extended time periods.

One striking repeat expansion in CTVT is the Homogeneously Staining Region (HSR), a large negatively heteropyknotic region visible cytogenetically as a paler, lightly staining area along the long arm of the second largest submetacentric chromosome. Oshimura et al. (1973) identified the HSR as a massive 45S ribosomal DNA (rDNA) expansion, but its precise structure and function remained unclear.

Using modern long-read sequencing, we have elucidated the HSR's repeat unit structure, revealing that the expansion primarily involves the intergenic spacer region, with modest amplification of rDNA genes themselves. Fluorescent in-situ hybridization validates this structure. The HSR is highly methylated, suggesting epigenetic silencing of expanded rDNA copies, and spans approximately 10Mb, though this size varies considerably across CTVT cases and clades.

This comprehensive characterization of an ancient, massive rDNA expansion in a naturally occurring transmissible cancer provides further evidence of the high rDNA instability documented in cancer and aging.

Department of Veterinary Medicine, University of Cambridge, UK

POSTER PRESENTATION

A Common MC4R Variant with a Small but Significant Effect on Adiposity in Over 700,000 Dogs

Jade Scardham[1], Alyce McClellan[1], Michelle Daya[1], Michael Denyer[2], Rebecca Chodroff Foran[2], Oliver Forman[2], Eleanor Raffan[1]

The melanocortin-4 receptor (MC4R) regulates appetite and energy homeostasis across species. Using genotype and veterinary phenotype data from a Wisdom Panel™ cohort of more than 700,000 genotyped dogs, we performed genome-wide association analyses of body condition score (BCS). Linear mixed models controlling for sex, neuter status, age, and population structure identified a highly significant association between BCS and the T allele at CanFam4 chr1:16,215,057 ($\beta = +0.0338 \pm 0.0018$; $p = 4.8 \times 10^{-80}$), corresponding to MC4R p.V213F (c.G637T, rs852614811). Effects were directionally concordant across multiple breeds ($\beta \approx +0.03–0.15$) and slightly stronger in neutered than entire dogs. In-vitro characterisation indicates partial loss-of-function for p.V213F, driven by reduced β-arrestin recruitment, a modest reduction in cAMP Emax, and lower surface expression, consistent with an obesity-increasing allele. Despite its small individual effect, the variant's population-level significance highlights the value of large-scale canine cohorts for quantifying subtle genetic contributions to adiposity. This work demonstrates how integrating direct-to-consumer genotypes with electronic health phenotypes refines effect estimates at biologically validated loci.

Acknowledgments: We are grateful to the Banfield clinicians whose detailed and consistent medical records made this research possible, and to the Mars data and curation teams supporting the Wisdom Panel™ dataset. We thank the many dog owners who contributed samples and phenotypic information for research use.

1 Department of Physiology, Development and Neuroscience, University of Cambridge, UK
2 Wisdom Panel™, MARS Petcare Science & Diagnostics

POSTER PRESENTATION

Whole-genome sequencing identifies a candidate pathogenic variant for osteogenesis imperfecta in a Miniature Schnauzer

Ellen Schofield[1], Louise Pettitt[1], Becca Foran[2], Jamie Freyer[2], Amy Rossi[3], Cathryn Mellersh[1], Oliver Forman[2]

Osteogenesis imperfecta (OI), or brittle bone disease, is a genetic disorder characterised by fragile bones and loose joints due to poor collagen formation, leading to spontaneous fractures and other health issues. In humans, mutations in genes involved in the collagen synthesis pathway have been shown to cause OI and there are currently 19 recognised types. OI has been shown to have autosomal recessive, dominant and X-linked patterns of inheritance in both human and dogs.
A single adult male Miniature Schnauzer (MS) presented with clinical signs consistent with osteogenesis imperfecta (OI) to a veterinary referral centre. Other potential known genetic causes were excluded so whole genome sequencing (WGS) was undertaken by Wisdom and the data sent over to the Canine Genetics Centre (CGC) for further analysis. Due to limited case history, all possible inheritance patterns were hypothesised.
The data from the individual case were incorporated into a cross-breed joint-called dataset of 53 Kennel Club (KC) toy group breeds, including 15 additional MS. Following filtering and segregation, including comparisons with the Dog10K dataset of ~1900 samples, eight variants remained that were consistent with an autosomal-recessive mode of inheritance.
All eight identified variants are located in genes not directly implicated in the collagen synthesis pathway; however, one gene, SPARC, has previously been associated with recessive forms of human osteogenesis imperfecta (OI). The predicted high-impact single-base insertion within exon 7 of SPARC introduces a frameshift, resulting in a premature termination codon and consequent truncation of the mature protein.
A novel single nucleotide variant (SNV) associated with canine OI was identified. Further screening of MS is being undertaken to investigate the variant prevalence in the wider population.

1 Canine Genetics Centre, Department of Veterinary Medicine, University of Cambridge, UK
2 Wisdom Panel, Science and Diagnostics, Mars Petcare, Fountain Valley, CA, USA
3 BluePearl Tacoma, 2510 S 84th St, Lakewood, WA 98499

POSTER PRESENTATION

Tracheal Cartilage Abnormalities in Brachycephalic Dog Breeds

Francesca Tomlinson[1], David Sargan[1], Jane Ladlow[1,2]

Introduction: Pathological tracheal cartilage phenotypes are common in certain toy and brachycephalic canine breeds. These tracheal abnormalities can contribute to airway narrowing in brachycephalic obstructive airway syndrome (BOAS), leading to difficulty breathing and exercise intolerance. Tracheal hypoplasia is commonly noted in the English Bulldog narrowing their airways, whilst in the Pug, tracheomalacia contributes to dynamic airway collapse. This study aimed to investigate tracheal morphology and its association with BOAS in fourteen other brachycephalic breeds.

Methods: In this study, a large sample population of brachycephalic dogs underwent respiratory function grading (n = 898) to assess BOAS severity on a scale of 0 to 3. Clinical signs of tracheal collapse such as 'goose-honking' or a positive tracheal pinch test were noted. A computed tomography (CT) study was performed in a smaller sample of brachycephalic dogs (n = 190) and morphometric measurements were taken of the trachea at the level of mid-body of cervical vertebra C4. Tracheal collapse ratio (TrC-R) was calculated from the height and width, and tracheal cross-sectional area was normalised to body surface area (TrCSA-BSA). Descriptive statistics were calculated for each breed. One-way ANOVA was used to compared BOAS Grades to TrCSA-BSA.

Results: A reduced TrCSA-BSA was found to be significantly associated with increasing BOAS grade severity ($p < 0.0001$). Clinical signs of tracheal collapse were particularly common in the Affenpinscher (26%, n = 69) and were only noted in eight dogs of other breeds (Boston Terrier, Chihuahua, Japanese Chin, Pomeranian and Staffordshire Bull Terrier). In the CT study, Affenpinschers were found to have marked tracheal collapse with a mean height to width ratio of 0.45 (SD = 0.21, n = 3). Boston Terriers were noted to have a comparatively smaller trachea for their weight (mean TrCSA-BSA = 162, SD = 31, n = 14), compared to the other breeds (whole sample mean TrCSA-BSA = 260, SD = 90).

Discussion/Conclusion: Tracheal collapse and tracheal hypoplasia are distinct pathological phenotypes in toy and brachycephalic dog breeds. Tracheal hypoplasia was found to be associated with BOAS status. Certain breeds such as the Boston Terrier were found to have comparatively smaller tracheas in relation their body size. Whilst tracheal collapse is common in small and toy breed dogs, compared to similar populations, the Affenpinscher is particularly prone to this condition. Underlying genetic mutations causing chondromalacia that result in tracheal collapse have yet to be identified in the dog. The Affenpinscher could represent a valuable model for identifying genetic variants associated with tracheal collapse.

1 Department of Veterinary Medicine, University of Cambridge, UK
2 Granta Veterinary Specialists, Cambridge, UK

CONFERENCE REGISTRATION LIST

Name	Surname	Email address	Organisation / Institution
Chandrindu	Abeykoon	c.abeykoon@sms.ed.ac.uk	University of Edinburgh
Tomas	Bergstrom	tomas.bergstrom@slu.se	Swedish University of Agricultural Sciences
Bart	Broeckx	bart.broeckx@ugent.be	Ghent University
Alex	Cagan	atjc2@cam.ac.uk	University of Cambridge
Lucie	Chevallier	lucie.chevallier@vet-alfort.fr	École Nationale Vétérinaire d'Alfort
Rosalind	Craddock	ros.craddock@ed.ac.uk	University of Edinburgh
Lucy	Davison	ldavison@rvc.ac.uk	Royal Veterinary College
Katy	Evans	KEvans@seeingeye.org	The Seeing Eye
Oliver	Forman	oliver.forman@effem.com	Wisdom Panel
Bailey	Francis	bf14@sanger.ac.uk	The Wellcome Sanger Institute
Kevin	Gori	kcg25@cam.ac.uk	University of Cambridge
Debbie	Guest	djguest@rvc.ac.uk	Royal Veterinary College
Joanna	Ilska	Joanna.Ilska@thekennelclub.org.uk	The Kennel Club
Sebastian	Jaehne	sebastian.jaehne@springernature.com	Springer Nature
Hannah	James	hannah.james@thekennelclub.org.uk	The Kennel Club
Chris	Jenkins	cj452@cam.ac.uk	University of Cambridge
Arielle	Johnson-Pitt	ldavison@rvc.ac.uk	Royal Veterinary College
Tom	Lewis	tom.lewis@guidedogs.org.uk	Guide Dogs
Bruno	Lopes	basl2@cam.ac.uk	University of Cambridge
Freddie	McNay	fghm2@cam.ac.uk	University of Cambridge
Cathryn	Mellersh	cm935@cam.ac.uk	University of Cambridge
Elizabeth	Murchinson	epm@cam.ac.uk	University of Cambridge
Esther	Palomino Lago	epalominolago@rvc.ac.uk	Royal Veterinary College
Eleanor	Raffan	er311@cam.ac.uk	University of Cambridge
David	Sargan	drs20@cam.ac.uk	University of Cambridge
Jade	Scardham	js2868@cam.ac.uk	University of Cambridge
Ellen	Schofield	es904@cam.ac.uk	University of Cambridge
Thomas	Simon	thomas.simon@slu.se	Swedish University of Agricultural Sciences
Katherine	Stanbury	ks2017@cam.ac.uk	University of Cambridge
Andrea	Strakova	as2112@cam.ac.uk	University of Cambridge
Fran	Tomlinson	ft270@cam.ac.uk	University of Cambridge
Frank	van Steenbeek	f.g.vansteenbeek@uu.nl	Utrecht University
Marsha	Wallace	ldavison@rvc.ac.uk	Royal Veterinary College
Pam	Wiener	pam.wiener@roslin.ed.ac.uk	The Roslin Institute

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TRANSPORT

Trains

Cambridge Station

Cambridge station lies to the south of the city. It is around a 50-minute walk from the conference venue, and is well served by buses and taxis.

- A taxi from the station to the West Hub takes around 10 minutes (£10–12).
- The Universal (U) Bus runs frequently from Stop 8 at the station to West Cambridge:
  - ~30 minutes to the West Hub
  - ~20 minutes to Selwyn College

(See Buses section below for fare information)

Cambridge North Station

Cambridge North is less convenient for the conference, with no direct bus route to West Cambridge and fewer taxis available.

Driving/Parking

Cambridge is easily reached via the M11 from the south, the A1/A14 from the north, and the A14 from the east and west.

There is no parking available at Selwyn College or the West Hub.

The recommended option is the Madingley Road Park & Ride, a 10-minute walk from the West Hub.

- The site is also served by the Universal (U) Bus, which runs to Selwyn College and the city centre.
- Vehicles may be left for extended periods; parking for 20–48 hours costs £20.

Buses

The Universal Bus (U) provides a frequent service linking West Cambridge, the historic city centre and Cambridge railway station. Buses run every 10–15 minutes throughout the day.

The U bus is the most convenient public transport route to the West Hub and Selwyn College, and connects directly with Cambridge station (Stop 8).

Fares

The Universal Bus offers discounted fares for students and staff:

- University of Cambridge ID: £1.50 per single
- Other educational institutions: £2.50 per single; £3.30 all-day
- Standard fare: £2.80 per single; £4.40 all-day

Discount bundles for Cambridge University members are available via the Universal Bus app.

WEST CAMBRIDGE

GETTING AROUND

A Selwyn College
B Madingley Road Park and Ride
C West Hub

CITY CENTRE - WEST

A Selwyn College
B St John's College (multiple entrances, please check which are open)
C The Eagle pub

NOTES

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END NOTES

Designed and compiled by Jim Johnson. The CAGH website was designed by Ellen Schofield. Both are based at the Canine Genetics Centre, Cambridge.

Thank you to Minuteman Press in Cambridge for printing the brochure, and to everyone who contributed abstracts, content, and logistical support.

Cambridge photographs by Jim Johnson unless otherwise stated. The image above is Byron's Bear pub, commemorating the famous tale of Lord Byron keeping a pet bear at Cambridge to evade college bans on dogs — a story relevant to a conference on companion animals and one that also echoes YoYo's place in Selwyn folklore.

Left: The Old Cavendish Laboratory, where Watson and Crick carried out the research that led to their discovery of the double-helix structure of DNA.

And of course, many thanks to Roger Mosey for YoYo's story and photographs — a lovely reminder of how companion animals enrich our lives, and a fitting link to Cambridge and its distinctive character.

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