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Landrace and Korean native pigs using the 60K porcine single nucleotide polymorphism (SNP) beadchip and a mixed-effects model approach accounting for familial relationships between individuals. Data used in this study included 1105 F2 offspring. After implementation of quality control performance criteria, 42,773 SNP markers were left for GWAS. In SSC14, SNP markers with associations surpassing a Bonferroni threshold of $1.17 \times 10–6$ were detected under additive model.

Key Words: GWAS, gamma-glutamyl transpeptidase, Korean native pigs

P5040 Investigating the genetic basis of a Canine Motor-Sensory Neuropathy by HD Canine 170000 SNPs array. Michele Polli[1], Stefano P. Marelli[1], Cristina Di Palma[2], Alessandra Gessi[3], Alessandra Mezzelani[4], Jacopo Riva[1,2], and Maria Longeri*[1], *[1]Università degli Studi di Milano; Dipartimento di Scienze Animali - Sez. Zootecnica Veterinaria, Milan, Italy, [2]Clinica Veterinaria C.so Concordia, Milan, Italy, [3]VEtoGene srl, spin-off Università degli Studi di Milano, Milan, Italy, [4]Institute for Biomedical Technologies - National Research Council of Italy, Segrate (MI), Italy.*

The aims of the present research is to investigate the genetic basis of a Canine Motor-Sensory Neuropathy (CMS) clinically very similar to human Charcot-Marie-Tooth (CMT) inherited disease. Recently some cases of degenerative neuropathy in Rhodesian Ridgeback (RR) dogs have been clinically studied. RR can be considered a valuable animal model for CMT and for inherited developmental disorders of Peripheral Nervous System. The 9 generations family pedigree (79 records) of 3 dogs suspected of CMS was drawn suggesting an autosomal recessive inheritance of the disease. Parentage was verified comparing the genetic profiles to ensure genealogical accuracy. The affected dogs showed fasciculations and tremors, atrophy, limb muscles degeneration plus demyelination and myelin outfolding. Clinical and histological features resulted in a description reporting signs very similar to human CMT subtypes: CMT4B-1, CMT4B-2 and CMT4H (all with autosomal recessive inheritance). MTMR2, MTMR13 and FGD4 are considered to be the causative genes for these forms of the human disease respectively. MTMR2, MTMR13 and FGD4 human genes were identified on dog genome nucleotide sequence assembly and homology and conservation were evaluated in silico. Out of the pedigree, genomic DNA of 3 healthy and 3 affected dogs were extracted and a Genome-Wide Association (GWA) was performed using high density SNP arrays (~170,000 SNPs - Illumina). By Homozigosity Mapping 17 candidate regions were significantly associated.

Key Words: Rhodesian dog, Canine Motor-Sensory Neuropathy, high density SNP array

P5041 Pedigree of Abyssinian cats with amyloidosis and polymorphism of SAA gene in several feline breeds. Maria Longeri*[1], Stefano P. Marelli[1], Paolo Valiati[1], Andrea Mapelli[1], Jacopo Riva[1,2], and Michele Polli[1], *[1]Università degli Studi di Milano; Dipartimento di Scienze Animali - Sez. Zootecnica Veterinaria, Milan, Italy, [2]VEtoGene srl, spin-off Università degli Studi di Milano, Milan, Italy.*

Amyloidosis is a rare disorders occurring in many species including, humans, chickens, and mainly domestic and wild felids. It is characterized, and the diagnosis is only possible so far, by post-mortem evidence of huge amyloid deposits in single organs or systemic. In Abyssinian/Somali and Siamese/Oriental cats a juvenile form with storage of apolipoprotein aposerum amyloid (apo-SAA) occurs most frequently than in other breeds and has been repeatedly suggested as familial. In the past SAA aminoacidic and coding sequence variations and amiloidogenic variants have been recorded on small cohorts of Abyssinians, Siameses and domestic shorthair cats. Additional amyloid associated SAA genes and predisposing factors (such as infections and inflammatory process) involved in the disease onset and development have also been suggested. The present work mainly aims to present a family pedigree (73 records) of Abyssinian cats with more than 20 subjects recording an anamnesis of death due to Amyloidosis. Moreover SAA coding region has been sequenced in the 31 available samples (both affected and healthy) out of the pedigree, in a group of 50 cats belonging to the following breeds: Siamese, Oriental, domestic shorthair cat, Bengal, Devon Rex, Chartreuse, Siberian, Thai, Ragdoll, Scottish Fold, Persian, Exotic, Birman, Norwegian Forest cat, Sphynx and in 9 samples of Panthera tigris. The pedigree/phenotype data seem to reconfirm familial predisposition and the SAA sequences present the previously suggested amiloidogenic motives. The importance of understanding the mechanisms of amyloidogenesis also for human health, encourage the constitution of an "Amyloid network" and a wide genome analysis.

Key Words: Abyssinian cat, amyloidosis, SAA polymorphism

P5042 Genetic control of swine responses to PRRSV infection: Progress of the PRRS Host Genetics Consortium. J. K. Lunney*[1], I. Choi[1], C. J. Souza[1], K. P. C. Araujo[1], S. M. Abrams[1], J. P. Steibel[2,3], M. Arceo[2], C. W. Ernst[2], J. M. Reecy[4], E. Fritz[4], J. C. M. Dekkers[4], N. J. Boddicker[4], E. H. Waide[4], X. Zhao[4], M. F. Rothschild[4], G. S. Plastow[5], R. A. Kemp[6], J. C. S. Harding[7], M. Kerrigan[8], B. Trible[8], and R. R. R. Rowland[8], *[1]USDA, ARS, BARC, APDL, Beltsville, MD, USA, [2]Dept. Animal Science, Michigan State Univ.,*