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Genetic testing in domestic cats

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Abstract

Varieties of genetic tests are currently available for the domestic cat that support veterinary health care, breed management, species identification, and forensic investigations. Approximately thirty-five genes contain over fifty mutations that cause feline health problems or alterations in the cat's appearance. Specific genes, such as sweet and drug receptors, have been knocked-out of Felidae during evolution and can be used along with mtDNA markers for species identification. Both STR and SNP panels differentiate cat race, breed, and individual identity, as well as gender-specific markers to determine sex of an individual. Cat genetic tests are common offerings for commercial laboratories, allowing both the veterinary clinician and the private owner to obtain DNA test results. This article will review the genetic tests for the domestic cat, and their various applications in different fields of science. Highlighted are genetic tests specific to the individual cat, which are a part of the cat's genome.

Keywords

Domestic cat; Feline; Genetic testing; Identification; Mutations; Parentage

1. Introduction

Genetic testing has been available in the domestic cat since the 1960's, but as like other species, over the past 50 years, the level of resolution has improved from the chromosome level to the sequence level. Knowing the direct causative mutation for a trait or disease assist cat breeders with the breeding programs and can help clinicians determine heritable presentations versus idiopathic versions of a health concern. Genetic tests cover all the various forms of DNA variants, including chromosomal abnormalities, mtDNA variation, gene loss, translocations, large inversions, small insertions and deletions and the simple nucleotide substitutions. Higher throughput technologies have made genetic testing cheaper, simpler and faster, thereby making cat genetic testing affordable to the lay public and small animal practice clinicians. The genetic resources for cats and other animal species have also opened the doors for animal evidence to be supportive in criminal investigations. This review will highlight the various tests available for the domestic cat and their specific capabilities and role's in cat health and management.

2. Domestic cat genetic testing

2.1. Cytogenetic testing

Some of the earliest genetic testing for any species was the examination of the chromosomes to determine the presence of the normal and complete genomic complement. Early studies of

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mitotic chromosomes of the domestic cat revealed an easily distinguishable karyotype consisting of 18 autosomal chromosomes and the XY sex chromosome pair, resulting in a 2N complement of 38 chromosomes for the cat genome [1]. Cat chromosomes are clearly defined by size; centromere position; distinctive giemsa banding patterns of the short (*p*) and long (*q*) arms of each chromosome; and the presence of only a few small acrocentric chromosomes. Various cytogenetic techniques, such as R-, RBG-banding and fragile site studies, have also helped distinguish and characterize the cat chromosomes [2–5]. Although a sequential numbering of the chromosomes has been suggested [6], the historical classification of chromosomes into morphologic groups has been retained in the cat. Hence cats have three large metacentric chromosomes (A1 to A3), four large subtelomeric chromosomes (B1 to B4), two medium-size metacentrics (C1 to C2), four small subtelomerics (D1 to D4), three small metacentrics (E1 to E3), and two small acrocentrics (F1 and F2). The X chromosome is midsize and subtelomeric, similar to chromosome B4.

Although the cat genome is conserved to humans, certain well-known chromosomal abnormalities are not found. For example, cats do not have a significant fragile X site on the X chromosome that is associated with mental retardation [5]. An analog to Down's syndrome is not present in the cat since the genes found on human chromosome 21 are represented on the mid-sized metacenteric chromosome C2, which also has genes from human chromosome 3. However, Turner's Syndrome (XO), Klinefelter's Syndrome (XXY) and chimerism has been documented in the domestic cat. Sex chromosome aneuploidies and trisomies of small acrocentric chromosomes were typically associated with cases of decreased fertility and syndromes that displayed distinct morphological presentations. Because cat has a highly recognizable X-linked trait [7–10], *Orange*, and the X-inactivation process was recognized [11], tortoiseshell and calico male cats were the first feline suspects of chromosomal abnormalities, particularly sex chromosome aberrations. Karyotypic and now gene-based assays are common methods to determine if a cat with ambiguous genitalia [12] or a poor reproductive history has a chromosomal abnormality. Karyotypic studies of male tortoiseshell cats have shown that they are often mosaics, or chimeras, being XX/XY in all or some tissues [10,13–20]. The minor chromosomal differences that are cytogenetically detectable between a domestic cat and an Asian leopard cat are likely the cause of fertility problems in the Bengal cat breed, which is a hybrid between these two species [21]. Other significant chromosomal abnormalities causing common "syndromes" are not well documented in the cat. Several research and commercial laboratories can perform cat chromosomal analyses when provided a living tissue, such as a fibroblast biopsy or whole blood for the analysis of white blood cells.

2.2. Inherited disease tests

The candidate gene approach has been fruitful in domestic cat investigations for the identification of many diseases and trait mutations. The first mutations identified were for a gangliosidosis and muscular dystrophy, discovered in the early and mid-1990's [22,23], as these diseases have well defined phenotypes and known genes with mutations that were as found in humans. Most of the common diseases, coat colors, and coat types were deciphered in the cat following the same candidate gene approach. To date, other than the muscular dystrophy mutation [22], all other mutations in the cat are autosomal.

Most of the identified disease tests in cats that are very specific to breeds and populations are available as commercial genetic tests (Table 1). Typically, diseases are identified in cat breeds, which are a small percentage of the cat population of the world, perhaps at most 10–15% in the USA [24]. However, some mutations that were found in a specific breed, such as mucopolysaccharidosis in the Siamese [25,26], were found in a specific individual and the mutation is not of significant prevalence in the breed (Table 2). These genetic mutations should not be part of routine screening by cat breeders and registries, but clinicians should

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know that genetic tests are available for diagnostic purposes, especially from research groups with specialized expertise, such as at the University of Pennsylvania (http://research.vet.upenn.edu/penngen). Other diseases, such as polycystic kidney disease (PKD), are prevalent, PKD in Persians is estimated at 30–38% worldwide [27–29]. Because of cross breeding with Persians, many other breeds, such as British Shorthairs, American Shorthairs, Selkirk Rex, and Scottish Folds, also need to be screened for PKD [30–32]. As PKD testing begins to become less common, as breeders remove positive cats, other genetic tests are becoming more popular, such as coat color and other disease traits (Fig. 1).

To date, most cat genetic tests have been for traits that have nearly complete penetrance, having little variability in expression, and early onset. However, some recognized mutations in cats might be considered risk factors, predisposing an individual to health problem. Excellent examples of mutations that confer a risk in cats are the DNA variants associated with cardiac disease in cats. Hypertrophic cardiomyopathy (HCM) is a recognized genetic condition [33]. In 2005, Drs. Meurs, Kittleson and colleagues published that a DNA alteration, A31P, in the gene cardiac *myosin-binding protein C 3* (*MYBPC3*) was strongly associated with HCM in a long-term research colony of Maine Coon cats at UC Davis [34]. The data clearly showed not all cats with the mutation had HCM and some cats with HCM did not have the DNA mutation. Age of onset, variable expression, and disease heterogeneity were alluded to in this report. These aspects suggested that the identified DNA variant should be considered more of a “risk factor” than a directly causative mutation. Recent studies have shown that not all Maine Coon cats with the A31P mutation get HCM [35,36] and one of those papers has mistakenly interpreted this lack of penetrance as being evidence that the A31P mutation is not causal [36]. This interpretation is misleading, causing debate as to the validity of the Maine Coon HCM test. As true in humans with cardiac disease, the finding that not all cats with the A31P mutation in *MYBPC3* get HCM is actually usual in the field of HCM genetic testing.

Like cat HCM mutations, other disease mutations have shown variation in penetrance and expression. The *CEP290* PRA mutation in Abyssinians has a late age of onset and some cats with subclinical disease have been identified [37]. Some cats with the pyruvate kinase deficiency can have very mild and subclinical presentations [38]. Thus, disease or trait causing mutations may not be 100% penetrant, thus, they do not always cause clinically detectable disease.

Cats are one of the few species to have their blood type genetic mutation determined [39]. Blood type incompatibilities can lead to transfusion reactions and neonatal isoerythrolysis for the cat, but inherently this characteristic is not necessarily a disease. A point mutation and an 18 base pair deletion have both been implicated in the gene *CMAH* as indicating the presence of the B blood type, or a B blood type carrier. Because both mutations are on the same allele, a clear indication of the true causative mutation could not be determined. Thus, both mutations should be examined in cats to genetically determine blood type at the current time.

Genome-wide association studies are now feasible for domestic cats and their breeds due to the recent development and release of an Illumina 63K Inflnium feline iSelect DNA array. With this resource, the localization of disease and phenotypic traits can proceed via case – control or cross-breed designs, supporting rapid localization of causative loci, hopefully implicating regional candidate genes. Improvements in the cat genome sequence will also greatly facilitate mutation detection. Therefore, genetic tests for simple disease traits should develop more rapidly for the cat in the future. The genome arrays should also assist with genetic studies on traits that may already have known mutations. As found in many species, specific presentations, such as PKD or HCM, can be caused by different genes and

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mutations in different and even the same populations, known as disease heterogeneity. The current Maine Coon HCM mutation does not account for all Maine Coons with HCM and many other breeds have HCM and not the Maine Coon or Ragdoll mutations. Likewise, many forms of PKD in humans exist. Thus in cats, there would be no reason why a second mutation in a different gene could cause PKD in another breed or as a different form in Persians. The DNA arrays should help clarify disease heterogeneity problems.

2.3. Phenotypic trait tests

The coat color mutations are common to all cats and are effective for genetic typing in all breeds and populations. Six different genes with nine different mutations affect the coat colors of cats, controlled by the common mammalian loci *Agouti (A)*, *Brown (B)*, *Color (C)*, *Dilute (D)*, and *Extension (E)* (Table 1). Most of the mutations have been identified by candidate gene approaches, however, the *Dilute* locus [40], required support from mapping approaches. Several other cat coat color or pattern loci have been localized, such as *Tabby (T)* [41,42], *Ticked (Ti)* [41,42], *Inhibitor (I)* [43,44], *Spotting (S)* [45], and *Orange (O)* [46,47], therefore the causative mutations should be forthcoming. The *White (W)* and *Spotting (S)* loci are likely to be complex with various alleles, as in other species and as demonstrated in initial studies of white feet, *Gloves*, in cats [48].

The common locus for long hair in mammals, *Long (L)*, which is controlled by *fibroblast growth factor 5*, *FGF5*, is also the major factor for cat hair length [49,50]. However, even though long fur is common in breeds and random bred cats, long fur genetic testing is an exception because four different mutations in *FGF5* can cause a cat to have long fur. One mutation, c.475A > C, is common to most all breeds and populations, suggesting this mutation to be the most ancient mutation, but the others are more specific to particular breeds [51]. A second mutation, c.365insT, is highly prevalent in the Ragdoll breed and a third, c.406C > T, is rare among pedigreed breeds but is highly prevalent in Norwegian Forest cats. The additional mutation, c.474delT, was noted in various breeds. Thus, to determine accurately if a cat carries a mutation for long fur, all four mutations must be genotyped.

Additional hair type mutations have also been identified in cats, including nakedness or *Hairless (Hr)* of the Sphynx [52]. The *keratin 71 (KRT71)* gene has a complex mutation that causes the rexoid – curly coat of the Devon Rex, which is one of the oldest curly coated breeds [52]. Although listed originally listed as different loci then later considered allelic, hairless and Devon curly are alleles within *KRT71*, the hairless mutation is recessive but dominant to curly [53]. Several other rexoid-curly coated cats are documented, another historical breed, the Cornish Rex has proven by breeding to be non-allelic to Devon Rex. The first genome-wide association study of the cat has led to the identification of the causative gene and mutation for this rexoid mutation (LA Lyons, personal communication).

2.4. Genetic testing concerns in hybrid cat breeds

Several cat breeds were formed by crossing with different species of cats. The Bengal breed is acknowledged worldwide and has become a highly popular breed. To create Bengals, Asian leopard cats (*Felis bengalensis*) were and are bred with domestic cat breds like Egyptian Mau, Abyssinian and other cats to form a very unique breed in both color and temperament [54]. The breed termed Chaussie is developing from crosses of domestic cats with Jungle cats (*Felis chaus*) and the breed termed Savannahs are from crosses with domestic cats and Servals (*Felis serval*). Bobcat (*Lynx rufus*) hybrids with domestic cats have not been genetically proven, to date. An Asian leopard cat had a common ancestor with the domestic cat about 6 million years ago, the bobcat about 8 million years ago, the Serval about 9.5 million years ago [55]. The Jungle cat is more closely related to a domestic cat

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than the leopard cat to the domestic cat. In addition, for some of these wild felid species, different sub-species were incorporated into the breed. The DNA sequence between a domestic cat and one of these wild felid species will have many genetic differences, less for the Jungle cat, more for Serval as compared to a domestic cat. The genetic differences are most likely silent mutations, but, the variation will interplay with genetic assays and may cause more allelic drop-out than what would be normally anticipated. No genetic tests are validated in the hybrid cats breeds, although the tests are typically used very frequently in Bengal cats. Thus, the accuracy for any genetic test is not known for hybrid cat breeds.

2.5. Species identification

Many markers in the genome can delineate species differences due to high mutation rates. However, historically, the mtDNA genome has been a more simple locus to explore and characterize between species, especially since the mutation rates of the control region and the coding genes tend to be higher than the nuclear genes on chromosomes [56]. Restriction fragment length polymorphism analysis initially described species [57], followed by control region length and coding gene variation [58,59]. Recent studies have furthered the development and analysis of universal primers to distinguish common mammalian species [60–62]. Specifically in cats, the 12S rRNA and cytochrome B genes [63–65], as well as the 16S rRNA genes can be amplified with universal PCR primers to distinguished Felidae [66].

The mtDNA control region has also been analyzed in detail to distinguish individual cats. A study considering 1394 cats, including cats from 25 distinct worldwide populations and 26 breeds determined twelve major mitotypes represented 83% of cats [67,68]. An additional 8.0% of cats are clearly derived from the major mitotypes. Unique mitotypes were found in 7.5% of the cats. The overall genetic diversity for this data set was 0.8813 ± 0.0046 with a random match probability of 11.8%. This region of the cat mtDNA has discriminatory power suitable for forensic application worldwide.

Now that full genome sequences are developing for many species, direct comparisons of genes, including their presence or absence, can be analyzed across species. Recent studies have shown cats to be lacking two common genes, a sweet receptor [69,70] and *UDP-glucuronosyltransferase (UGT) 1A6*, an enzyme important for drug metabolism [71]. Closer cross-species comparisons of genomes will likely unveil many more species-specific differences that can lead to class, order, family, species and sub-species diagnostics.

2.6. Parentage and individual identification

DNA-based parentage and individual identification typing methods have evolved over the years from restriction fragment length polymorphisms (RFLPs) and multi-locus probes, such as variable number tandem repeat (VNTRs), to single locus assays and short tandem repeat (STR) typing. DNA-based genetic testing is used for most domesticated animals to confirm identity and parentage, particularly to validate their registries. The domestic cat is one of the leading household pets, but parentage and identification testing lags for this species since no cat registry requires parentage validation.

Standardized genetic tests are important for sharing information, combining datasets and assisting with population management. Peer-review, research collaborations, and forums and comparison tests hosted by the International Society of Animal Genetics (ISAG) allow both formal and informal oversight of parentage test development in domesticated species. Under the auspices of ISAG, a parentage comparison test was performed among 17 worldwide commercial and research laboratories to identify and to validate a microsatellite-based profiling panel for parentage and identification in the domestic cat [72]. Nine of the 19 markers, plus two additional gender markers [107], were found to be sufficient for

parentage, gender determination, and identification testing of random bred, purebred and several wild felid species (Table 3). However, as more breeds are evaluated and as the tests become more popular, additional panels of markers are being developed for the cat under the auspice of ISAG.

A second panel of STR markers was developed for forensics applications, although a large database has not been developed [105,106]. The system simultaneously amplifies 11 polymorphic tetranucleotide STR loci and one gender identifying sequence tagged site on the Y chromosome sex-determining region Y gene (SRY gene). This panel was tested following the standard 8.1.2.2 of the quality assurance standards for DNA analysis recommended by the DNA Advisory Board (DAB) [73] and recommendations made for animal DNA forensic and identity testing [74]. The overall ability for detection of mutation rate was 0.3%, heterozygosity values ranged from 0.60 to 0.82, while the average cat breed heterozygosity was 0.71, ranging from 0.57 to 0.83. Mutations were observed in seven loci (FCA733, FCA742, FCA749, F124, F53, F85, and FCA441). Null alleles were observed in three loci (FCA736, F53, and F85). Once the null alleles are addressed with potentially new primer designs, this tetra-STR-based panel could have powerful applications in forensics.

2.7. Race and breed identification

A newly developing test for the domestic cat is a race and breed identification panel. Based on the studies by Lipinski et al. (2008) [75], and Kurushima et al. (2012, submitted), STRs have been tested in a variety of random bred cats from around the world and a majority of the major cat breeds of the USA and other regions. The genetic studies have been able to differentiate eight worldwide populations of cats – races – and can distinguish the major breeds. Analyses of the present day random bred cat populations suggest that the regional populations are highly genetically distinct, hence analogous to humans, different races of cats. The regional genetic differentiation is capture and displayed within the breeds that developed later from those populations. The foundation population (race) of the Asian breeds, such as Burmese and Siamese, are the street cats of Southeast Asia, whereas the foundation population (race) of the Maine Coon and Norwegian Forest cat are Western European cats. Phenotypic markers help to delineate breeds within specific breed families, such as the Persian, Burmese, and Siamese families. The cat race and breed identification tests are similar to tests that have been developed for the dog, such as the Mars, Inc. Wisdom Panel (http://www.wisdompanel.com/). Although similar, domestic cats are random bred cats and not a concoction of pedigreed breed cats. Cat breeds developed from the random bred populations that have existed in different regions of the world for thousands of years. Therefore, the claims of the cat race and breed identification tests are different than the dog tests, not claiming that most household cats are recent offspring of pedigreed cats.

3. Conclusion

Genetic testing is an important diagnostic tool for the veterinarian, breeder, and owner. Genetic tests are not 100% foolproof and the accuracy of the test procedure and the reputation and customer service of the genetic testing laboratory needs to be considered. Some traits are highly desired and genetic testing can help breeders to more accurately determine appropriate breedings, potentially becoming more efficient breeders, thus lowering costs and excess cat production. Other traits or diseases are undesired, thus genetic testing can be used to prevent disease and potentially eradicating the concern from the population. Genetic tests for simple genetic traits are more consistent with predicting the trait or disease presentation, but, as genomics progress for the cat, more tests that confer risk will become more common. Veterinarians will have to weigh the relative risk of having a mutation versus having disease as part of their differentials and breeders will have to

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consider risk factors along with the other important attributes of a cat for their breeding decisions.

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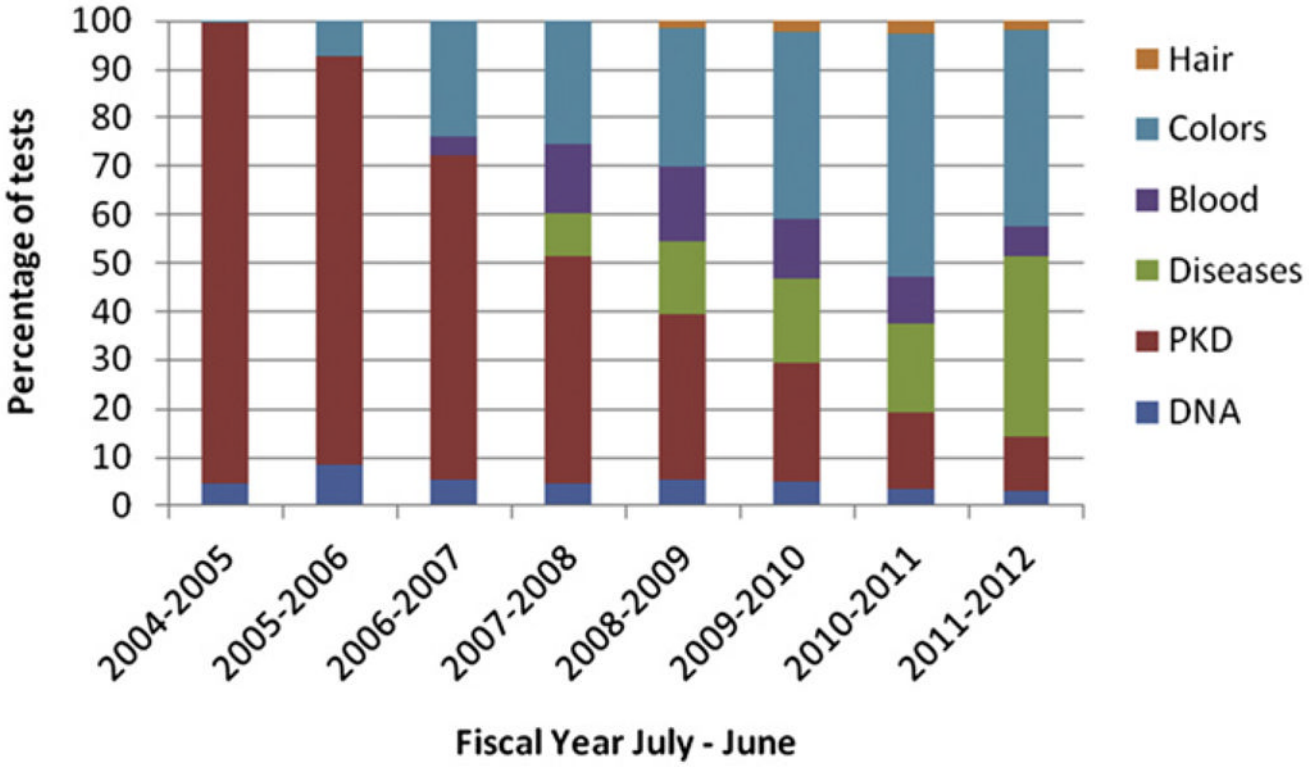

Fig. 1.

Trends of genetic testing in the domestic cat. DNA-based genetic tests are presented for the cat. Parentage and individual identification (DNA) has not increased as cats do not require testing for registration. One of the most popular tests, PKD, is presented separately to show that the testing requests are decreasing as breeders are eliminating positive cats from breeding programs. Other disease tests and color tests are becoming more popular tests in the cat market. Data from UC Davis Veterinary Genetics Laboratory.

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Table 1

Common commercialized DNA tests for domestic cats.

Disease/trait (alleles)	MOI[b]	Phenotype	Breeds	Gene	Mutation[d]
Agouti (*A, a*) [76]	AR	Banded fur to solid	All breeds	*ASIP*	c.122_123delCA
Amber (*E, e*) [77]	AR	Brown color variant	Norwegian forest	*MC1R*	c.250G > A
Brown (*B, b, b*^{l}) [78,79]	AR	Brown, light brown color variants	All breeds	*TYRP1*	*b* = c.8C > G, *b*^{l} = c.298C > T
Color (*C, c*^{b}, *c*^{s}, *c*) [79–81]	AR	Burmese, Siamese color pattern, full albino	All breeds	*TYR*	*C*^{b} = c.715G > T, *C*^{s} = c.940G > A, *c* = c.975delC
Dilution (*D, d*) [40]	AR	Black to grey/blue, Orange to cream	All breeds	*MLPH*	c.83delT
Gloves (*G, g*) [48]	AR	White feet	Birman	*KIT*	(Submitted)
Hairless (*Hr, hr*) [52]	AR	Atrichia	Sphynx	*KRT71*	c.816 + 1G > A
Long fur (*L, l*) [49,50]	AR	Long fur	All breeds[c]	*FGF5*	c.356_367insT, c.406C > T, c.474delT, c.475A > C
Rexing (*R, r*)	AR	Curly hair coat	Cornish Rex	*PYP2R5*	(Submitted)
Rexing (*Re, re*) [52]	AR	Curly hair coat	Devon Rex	*KRT71*	c.1108-4_1184del, c.1184_1185ins AGTTGGAG, c.1196insT
AB blood type (*A, b*) [39]	AR	Determines type B	All breeds	*CMAH*	c.1del-53_70, c.139G > A
Gangliosidosis 1 [82]	AR	Lipid storage disorder	Korat, Siamese	*GBL1*	c.1457G > C
Gangliosidosis 2 [83]	AR	Lipid storage disorder	Burmese	*HEXB*	c.1356del-1_8, c.1356_1362delGTTCTCA
Gangliosidosis 2 [23]	AR	Lipid storage disorder	Korat	*HEXB*	c.39delC
Glycogen storage dis. IV [92]	AR	Glycogen storage disorder	Norwegian forest	*GBE1*	IVS11 + 1552_IVS12-1339 del6.2 kb ins334 bp
Hypertrophic cardiomyopathy [34]	AD	Cardiac disease	Maine Coon	*MYBPC*	c.93G > C
Hypertrophic cardiomyopathy [85]	AD	Cardiac disease	Ragdoll	*MYBPC*	c.2460C > T
Hypokalemia	AR	Potassium deficiency	Burmese	*WNK4*	(Submitted)
Progressive retinal atropy [86]	AR	Late onset blindness	Abyssinian	*CEP290*	IVS50 + 9T > G
Progressive retinal atropy [87]	AD	Early onset blindness	Abyssinian	*CRX*	c.546delC
Polycystic Kidney disease [32]	AD	Kidney cysts	Persian	*PKD1*	c.10063C > A
Pyruvate kinase def.[a]	AR	Hemopathy	Several	*PKLR*	c.693 + 304G > A
Spinal muscular atrophy [88]	AR	Muscular atrophy	Maine Coon	*LIX1-LNPEP*	Gene deletion

[a] Unpublished test, presented only as abstract, paper submitted.

[b] Mode of inheritance of the non-wildtype variant.

[c] Long fur variants are more or less common depending on the breed.

[d] Not all transcripts for a given gene may have been discovered or well documented in the cat, mutations presented as interpreted from original publication.

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Table 2

Other mutations for Inherited domestic cat diseases.[a]

Disease	Gene	Mutation[b]	Disease	Gene	Mutation[b]
Gangliosidosis 2 [89]	*HEXB*	c.1467_1491inv	Mucopolysaccharidosis VI [26]	*ARSB*	c.1427T > C
Gangliosidosis 2 [90]	*HEXB*	c.667C > T	Mucopolysaccharidosis VI [25,91]	*ARSB*	c.1558G > A
Gangliosidosis 2 [84]	*GM2A*	c.390_393GGTC	Mucopolysaccharidosis VII [92]	*GUSB*	c.1052A > G
Hemophilia B [93]	*F9*	c.247G > A	Niemann-Pick C [94]	*NPC*	c.2864G > C
Hemophilia B [93]	*F9*	c.1014C > T	Polydactyla [95]	*SHH*	c.479A > G
Hyperoxaluria [96]	*GRHPR*	G > A I4 acceptor site	Polydactyla [95]	*SHH*	c.257G > C, c.481A > T
Lipoprotein lipase def. [97]	*LPL*	c.1234G > A	Porphyria (congenital erythropoietic) [98]	*UROS*	c.140C > T, c.331G > A
Mannosidosis, alpha [99]	*LAMAN*	c.1748_1751delCCAG	Porphyria (acute intermittent) [100]	*HMBS*	c.842_844delGAG, c.189dupT, c.250G > A, c.445C > T
Mucolipidosis II [101]	*GNPTA*	c.2655C > T	Vitamin D resistant rickets [102]	*CYP27B1*	c.223G > A, c.731delG
Mucopolysaccharidosis I [103]	*IDUA*	c. 1107_1109delCGA or c. 1108_1110GAC	Vitamin D resistant rickets [104]	*CYP27B1*	c.637G > T

[a] The presented conditions are not prevalent in breeds or populations but may have been established into research colonies.

[b] Not all transcripts for a given gene may have been discovered or well documented in the cat, mutations presented as interpreted from original publication.

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Table 3

Genetic markers selected as a “core” panel for ISAG cat parentage and identification testing.

Marker	Chr.	Repeat	Forward primer 5′–3′ Reverse primer 5′–3′	Label	uM	PE (min–max) (breeds)	PE (min–max) (random)
FCA069	B4	AC	AATCACTCATGCACGAATGC AATTTAACGTTAGGCTTTTTGCC	VIC	0.20	0.1324–0.5336	0.3958–0.5948
FCA075	E2	TG	ATGCTAATCAGTGGCATTTGG GAACAAAAATTCCAGACGTGC	NED	0.10	0.1442–0.5771	0.4240–0.5992
FCA105	A2	TG	TTGACCCTCATACCTTCTTTGG TGGGAGAATAAATTTGCAAAGC	PET	0.20	0.2221–0.5585	0.6110–0.7101
FCA149[a]	B1	TG	CCTATCAAAGTTCTCACCAAATCA GTCTCACCATGTGTGGGATG	PET	0.18	0.1783–0.5995	0.3586–0.5767
FCA220	F2	CA	CGATGGAAATTGTATCCATGG GAATGAAGGCAGTCACAAACTG	FAM	0.30	0.0000–0.3383	0.1851–0.4221
FCA229	A1	GT	CAAACTGACAAGCTTAGAGGGC GCAGAAGTCCAATCTCAAAGTC	NED	0.25	0.0452–0.5131	0.3927–0.5813
FCA310[a]	C2	$(CA)_5TA(CA)_7TA(CA)_8$	TTAATTGTATCCCAAGTGGTCA TAATGCTGCAATGTAGGGCA	FAM	0.30	0.1196–0.5256	0.3417–0.5611
FCA441[b]	D3	TAGA	ATCGGTAGGTAGGTAGATATAG GCTTGCTTCAAAATTTTCAC	VIC	0.15	0.2061–0.5774	0.3388–0.5505
FCA678[d]	A1	AC	TCCCTCAGCAATCTCCAGAA GAGGGAGCTAGCTGAAATTGTT	NED	0.25	0.0415–0.4908	0.3016–0.5715
AMEL[c]	XY	–	CGAGGTAATTTTTCTGTTTACT GAAACTGAGTCAGAGAGGC			N/A	N/A
ZFXY[c]	XY	–	AAGTTTACACAACCACCTGG CACAGAATTTACACTTGTGCA	PET	0.20	N/A	N/A
				Total PE		**0.9008–0.9979**	**0.9947–0.9987**

[a]Markers that are of the first ten published feline microsatellites [106].

[b]A marker that is currently included in the feline forensic panel [107].

[c]The two markers on the X and Y chromosomes were added to the panel after the comparison test [108].

[d] Newly designed primers presented herein for *FCA678* generate a product 30 bp less than originally published primers.

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