

WHISPERS OF DAËNERYS DES GOUPI...

SOMALI

Microchip number: 900113003165717
ID kit: FCQVGJK

Test date: 2025-09-12
French Cat Club



Health conditions known in the breed

Progressive Retinal Atrophy (Discovered in the Abyssinian)	Gene	Risk Variant	Copies	Inheritance	Result
	CEP290	T>G	0	AR	Clear

Information about the genetic condition

Progressive Retinal Atrophy (PRA), in the rdAc form, follows the typical pattern where functional loss of rod photoreceptors occurs first, followed by loss of function of cone photoreceptors. Age of onset for this form of PRA is typically late, with the first ophthalmoscopic signs of affected cats seen at one to two years of age. These signs may include a slight grayish discoloration along the central fundus progressing to the entire tapetal fundus, a hyper-reflective tapetum and attenuated blood vessels. The disorder is progressive, causing increasing levels of vision loss and eventual blindness by three to seven years of age. Early indications of visual compromise may include disorientation and lack of awareness of changes to the surroundings, especially in low light conditions. Affected cats may accidentally bump into things and become more vocal.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to be shown. A carrier cat with one copy of the PRA mutation can be safely bred with a clear cat with no copies of the PRA mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the PRA mutation. Kittens in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected kittens. Please note: It is possible that disease signs similar to the ones caused by the PRA mutation could develop due to a different genetic or clinical cause.

Pyruvate Kinase Deficiency	Gene	Risk Variant	Copies	Inheritance	Result
	PKLR	G>A	0	AR	Clear

Information about the genetic condition

Pyruvate Kinase (PK) Deficiency presents as a chronic, intermittent, hemolytic anemia. The disorder has a high variability of age of onset and severity of clinical signs. The age of onset of clinical signs varies from six months to five years of age. Clinical signs of the disorder are highly variable but may include lethargy, weakness, diarrhea, pale mucous membranes, anorexia, poor coat quality, weight loss, icterus (jaundice), splenomegaly, and ascites in severe cases. The severity of clinical signs also varies greatly with some cats maintaining adequate quality of life and others requiring euthanasia. The disorder has been reported in multiple cat breeds.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to be shown. A carrier cat with one copy of the Pyruvate Kinase Deficiency mutation can be safely bred with a clear cat with no copies of the Pyruvate Kinase Deficiency mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Pyruvate Kinase Deficiency mutation. Kittens in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected kittens. Please note: It is possible that disease signs similar to the ones caused by the Pyruvate Kinase Deficiency mutation could develop due to a different genetic or clinical cause.

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Inheritance Mode Key

Autosomal Recessive (AR)

The trait is only expressed when both alleles (inherited from mother and father) contain the detrimental mutation.

Regarding to the presence of mutations dogs are classified into three groups:

- Affected (mut/mut)- both alleles carry mutation, disease could be clinically expressed
- Carrier (mut/normal)- one of two alleles carry mutation (heterozygotes), disease is not clinically expressed
- Clear (normal/normal)- mutation is not detected, normal genotype, healthy animal for the trait

Heterozygotes in this case are the carriers of mutation since they do not express the disease (unwanted trait). It is especially important to test such animals for mutations, since mutated alleles are “silently” (without seeing unwanted phenotype) carried through the population.

Autosomal Dominant (AD)

The trait is expressed when one of the alleles (inherited either from mother or father) is damaged (contains detrimental mutation). Only one single mutated allele already could cause the disease. The importance for genetic testing of such animals is primarily in early diagnostics of the disease and identification of animals before they mate because most of diseases with autosomal dominant mode of inheritance have an onset later in animals life.

X-linked Recessive (SR)

The trait is carried on a sex chromosome and that a trait is expressed only when both alleles (inherited from mother and father) are damaged (contain detrimental mutation). Males carry only a single copy of the gene, inherited from mother, since male sex chromosome Y does not contain full DNA sequence as female X chromosome does. Females on the other hand contain two X chromosomes. Heterozygotes in this case are the carriers of mutation since they do not express the disease (unwanted trait). Males carry only one copy of a gene: they could be normal homozygote or affected homozygote.

X-linked Dominant (SD)

The trait is carried on a sex chromosome and the trait is expressed when one of the alleles (inherited from mother or father) is damaged (contains detrimental mutation). Only one single mutated allele already could cause the disease (unwanted trait). Males carry only a single copy of the gene, inherited from mother, since male sex chromosome Y does not contain full DNA sequence as female X chromosome does. Females on the other hand contain two X chromosomes. Homozygotes in this case may be at higher risk or show a more severe form of the disease than heterozygotes. Males carry only one copy of a gene: they could be normal homozygote or affected homozygote.

Mitochondrial (MT)

Rather than genomic DNA, the trait is associated with mitochondrial DNA (mtDNA) of which there are thousands within each cell of the body. For disease (unwanted trait) to occur, a certain ratio of mtDNA, inherited only from mother, must contain the detrimental mutation compared to normal mtDNA.

Modifier (MO)

Genetic modifiers do not cause disease (unwanted trait) on their own. It is only when inherited in combination with specific detrimental mutations, the trait expression can be further influenced by the presence of a genetic modifier—either increasing likelihood of disease or the severity of a disease. It is dependent on the genetic modifier as to if heterozygotes or homozygotes will influence the trait expression.

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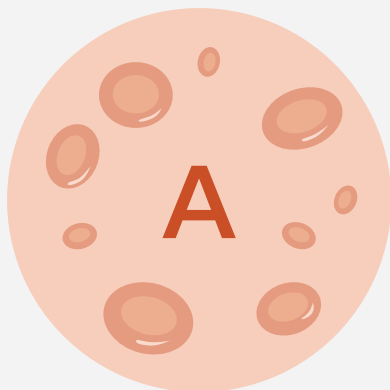
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Blood Type



Blood type

Type A (Most common)

Genotype*

A/A

Transfusion risk

⚠ Moderate

WHISPERS OF DAËNERYS DES GOUPILS DES CINQ CLOCHERS has the most common blood type. She can be transfused with Type A blood.

Breeding risk

✅ Low

If breeding, WHISPERS OF DAËNERYS DES GOUPILS DES CINQ CLOCHERS has a low risk of blood type incompatibility with nursing kittens.

Blood variants tested*

Variant Tested	Description	Copies
b variant 1	(Common b variant)	0
b variant 2	(Discovered in Turkish breeds)	0
b variant 3	(Discovered in Ragdolls)	0
c variant - Causes AB Blood Type	(Discovered in Ragdolls)	0

*This test identifies three known 'b' variants and one known 'c' variant in the CMAH gene when determining a cat's genetic blood type. Blood Type A is inferred in reporting when less than two genetic blood variants are detected.

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Interpreting feline blood types

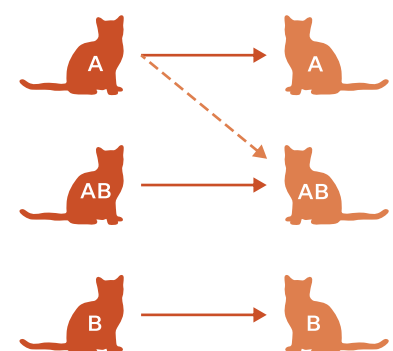
About blood type determination

The three important feline blood types of A, B, and AB are governed primarily by variants in the CMAH gene. A cat's blood type can be determined by its genotype, which consists of two gene variants – one inherited from each parent – that should be interpreted together. When determining blood type based on genotype, the A variant associated with blood type A is most dominant while the b variants associated with blood type B are most recessive. The c variant associated with blood type AB is intermediate between the A and b variants, meaning it is recessive to the A variant but dominant to b variants. Therefore, a genotype with at least one A variant will result in blood type A. For a cat to have blood type B, the genotype must consist of two b variants. Because the c variant is intermediate, a cat with blood type AB can either have a genotype consisting of two c variants or one c variant and one b variant.

About transfusion risk

Similar to humans, the different cat blood types will express different antigens on the surface of their red blood cells. This is significant because both type A and B cats are born with antibodies against other blood cell antigens. Notably, type B cats have high levels of antibodies against type A antigens. Cats with the rare blood type AB are most versatile as they express both red cell antigen types and, thus, can receive both type A and type AB blood transfusions.

Unlike humans, there is no cat blood type that can act as a universal blood donor. If a cat receives a non-compatible blood type during a transfusion, it may cause a severe, life-threatening reaction including fever, kidney failure, and widespread destruction of red blood cells. Prior to all transfusions, cats should be serologically typed and crossmatched to ensure compatibility.



→ Whole blood compatible
- - - - - → Packed red blood cells recommended

About breeding risk

During pregnancy, kittens are shielded from their mother's immune system. However, when kittens begin nursing, they receive some of their mother's antibodies in colostrum. Type B cats have high levels of antibodies against type A blood, so when blood type A or AB kittens are born to a blood type B mother, these antibodies, when absorbed by the newborn kitten, cause neonatal isoerythrolysis, a potentially fatal destruction of the kitten's red blood cells. Kittens of type B mothers with fathers of unknown or type A blood should be bottle fed or foster-nursed, and separated from their mother for the first 24 hours to avoid this reaction, unless blood typing performed immediately following birth shows the kitten to have a compatible blood type to the mother.

Although some blood types are less common and require additional planning when breeding, they represent normal genetic variation and should not be selected against when choosing breeding pairs.

Current limits of this test

This test identifies 4 variants (b variants c.269T>A, c.179G>T, c.1233delT and c variant c.346C>T) in the CMAH gene discovered in the domestic cat population and has been confirmed 99% concordant with serologic blood typing¹. Mik antigens also play a role in blood type compatibility, and are not included in this test. Cats carrying undetermined, new, or undiscovered variants in CMAH or other genes may have a different blood type compatibility than that reported by this test. Accuracy of this test at predicting blood type in wildcats or wildcat hybrid breeds has not been determined.

1. Anderson H, Davison S, Lytle KM, Honkanen L, et al. Genetic epidemiology of blood type, disease and trait variants, and genome-wide genetic diversity in over 11,000 domestic cats (2022) PLOS Genetics.

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Traits

Coat Color

	Gene	Variant	Copies	Result
Charcoal (Discovered in the Bengal)	ASIP	A ^{Pb}	0	No effect
Solid Color	ASIP	a	0	Banded hairs, tabby patterns likely
Gloving (Discovered in the Birman)	KIT	w ^g	0	No effect
Partial and Full White	KIT	W or w ^s	0	No effect
Amber (Discovered in the Norwegian Forest Cat)	MC1R	e	0	No effect
Russet (Discovered in the Burmese)	MC1R	e ^r	0	No effect
Dilution	MLPH	d	0	No effect
Albinism (Discovered in Oriental breeds)	TYR	c ^a	0	No effect
Colorpoint (Discovered in the Burmese)	TYR	c ^b	0	No effect
Colorpoint (Discovered in the Siamese)	TYR	c ^s	0	No effect
Mocha (Discovered in the Burmese)	TYR	c ^m	0	No effect
Chocolate	TYRP	b	0	No effect
Cinnamon Two copies of the Cinnamon variant result in cinnamon coat color.	TYRP	b ^l	2	Cinnamon coat color likely

Coat Type

	Gene	Variant	Copies	Result
Long Hair (Discovered in many breeds) Two copies of any Long Hair variant must be inherited for a cat to have a long coat. This can either be two copies of a particular variant, such as this one, or two of any combination of Long Hair variants.	FGF5	M4	2	Long coat likely
Long Hair (Discovered in the Norwegian Forest Cat)	FGF5	M2	0	No effect

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Coat Type

	Gene	Variant	Copies	Result
Long Hair (Discovered in the Ragdoll and Maine Coon)	FGF5	M3	0	No effect
Long Hair (Discovered in the Ragdoll)	FGF5	M1	0	No effect
Lykoi Coat (Variant 1)	HR	hr ^{Ca}	0	No effect
Lykoi Coat (Variant 2)	HR	hr ^{VA}	0	No effect
Hairlessness (Discovered in the Sphynx)	KRT71	re ^{hr}	0	No effect
Rexing (Discovered in the Devon Rex)	KRT71	re ^{dr}	0	No effect
Rexing (Discovered in the Cornish Rex and German Rex)	LPAR6	r	0	No effect
Glitter	Pending	gl	0	No effect

Tail Length

	Gene	Variant	Copies	Result
Short Tail (Variant 3)	HES7	jb	0	No effect
Short Tail (Variant 1)	T	C1199del	0	No effect
Short Tail (Variant 2)	T	T988del	0	No effect

Extra Toes

	Gene	Variant	Copies	Result
Polydactyly (Variant 1)	LIMBR1	HW	0	No effect
Polydactyly (Variant 2)	LIMBR1	UK1	0	No effect
Polydactyly (Variant 3)	LIMBR1	UK2	0	No effect

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Acute Intermittent Porphyrria (Variant 1)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyrria (Variant 2)	HMBS	G>A	0	AD	Clear
Acute Intermittent Porphyrria (Variant 3)	HMBS	Insertion	0	AD	Clear
Acute Intermittent Porphyrria (Variant 4)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyrria (Variant 5)	HMBS	G>A	0	AR	Clear
Autoimmune Lymphoproliferative Syndrome (Discovered in British Shorthair)	FASL	Insertion	0	AR	Clear
Burmese Head Defect (Discovered in the Burmese)	ALX1	Deletion	0	AD	Clear
Chediak-Higashi Syndrome (Discovered in the Persian)	LYST	Insertion	0	AR	Clear
Congenital Adrenal Hyperplasia	CYP11B1	G>A	0	AR	Clear
Congenital Erythropoietic Porphyrria	UROS	G>A	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Devon Rex and Sphynx)	COLQ	G>A	0	AR	Clear
Cystinuria Type 1A	SCL3A1	C>T	0	AR	Clear
Cystinuria Type B (Variant 1)	SCL7A9	C>T	0	AR	Clear
Cystinuria Type B (Variant 2)	SCL7A9	G>A	0	AR	Clear
Cystinuria Type B (Variant 3)	SCL7A9	T>A	0	AR	Clear
Dihydropyrimidinase Deficiency	DPYS	G>A	0	AR	Clear
Earfold and Osteochondrodysplasia (Discovered in the Scottish Fold)	TRPV4	G>T	0	AD	Clear
Factor XII Deficiency (Variant 1)	F12	Deletion	0	ARa	Clear
Factor XII Deficiency (Variant 2)	F12	Deletion	0	ARa	Clear
Familial Episodic Hypokalemic Polymyopathy (Discovered in the Burmese)	WNK4	C>T	0	AR	Clear
Glutaric Aciduria Type II	ETFDH	T>G	0	AR	Clear

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Glycogen Storage Disease (Discovered in the Norwegian Forest Cat)	GBE1	Insertion	0	AR	Clear
GM1 Gangliosidosis	GLB1	G>C	0	AR	Clear
GM2 Gangliosidosis	GM2A	Deletion	0	AR	Clear
GM2 Gangliosidosis Type II (Discovered in Domestic Shorthair cats)	HEXB	Insertion	0	AR	Clear
GM2 Gangliosidosis Type II (Discovered in Japanese domestic cats)	HEXB	C>T	0	AR	Clear
GM2 Gangliosidosis Type II (Discovered in the Burmese)	HEXB	Deletion	0	AR	Clear
Hemophilia B (Variant 1)	F9	C>T	0	XR	Clear
Hemophilia B (Variant 2)	F9	G>A	0	XR	Clear
Hyperoxaluria Type II	GRHPR	G>A	0	AR	Clear
Hypertrophic Cardiomyopathy (Discovered in the Maine Coon)	MYBPC	G>C	0	AR	Clear
Hypertrophic Cardiomyopathy (Discovered in the Ragdoll)	MYBPC	C>T	0	AD	Clear
Hypotrichosis (Discovered in the Birman)	FOXN1	Deletion	0	AR	Clear
Lipoprotein Lipase Deficiency	LPL	G>A	0	AR	Clear
MDR1 Medication Sensitivity	ABCB1	Deletion	0	AR	Clear
Mucopolysaccharidosis Type I	IDUA	Deletion	0	AR	Clear
Mucopolysaccharidosis Type VI	ARSB	T>C	0	AR	Clear
Mucopolysaccharidosis Type VI Modifier	ARSB	G>A	0	MO	Clear
Mucopolysaccharidosis Type VII (Variant 1)	GUSB	G>A	0	AR	Clear
Mucopolysaccharidosis Type VII (Variant 2)	USB	C>T	0	AR	Clear
Myotonia Congenita	CLCN1	G>T	0	AR	Clear
Polycystic Kidney Disease (PKD)	PKD1	C>A	0	AD	Clear

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Progressive Retinal Atrophy (Discovered in the Bengal)	KIF3B	G>A	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Persian)	AIPL1	C>T	0	AR	Clear
Sphingomyelinosis (Variant 1)	NPC1	G>C	0	AR	Clear
Sphingomyelinosis (Variant 2)	NPC2	G>A	0	AR	Clear
Spinal Muscular Atrophy (Discovered in the Maine Coon)	LIX1	Deletion	0	AR	Clear
Vitamin D-Dependent Rickets	CYP27B1	G>T	0	AR	Clear