Betty

Optimal Selection™ powered by WISDOM PANEL™

Breed: Bengal
Birth date: 2026-03-07
Test date: 2026-07-31
ID kit: FKMNWDMQRL

Betty's Profile

Pet information

Registered name
Betty

Sex
F

Owner reported breed
Bengal

Date of birth
2026-03-07

Genetic Diversity

Betty's Percentage of Heterozygosity
34%

Health summary

At Risk 0 conditions

Carrier 1 condition
- Factor XII Deficiency (Variant 2)

Clear 48 conditions

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Genetic Diversity

Heterozygosity

Betty's Percentage of Heterozygosity

34%

Betty's genome analysis shows an average level of genetic heterozygosity when compared with other Bengals.

Typical Range for Bengals

31% - 36%

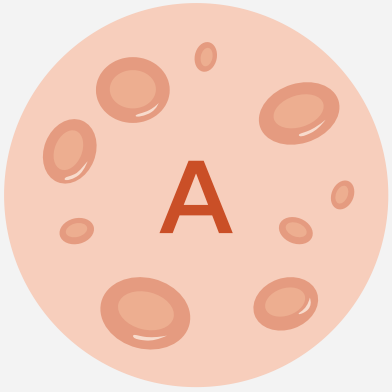
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Blood Type



Blood type
Type A (Most common)

Genotype*
A/A

Transfusion risk
⚠ Moderate

Betty has the most common blood type. She can be transfused with Type A blood.

Breeding risk
Low

If breeding, Betty 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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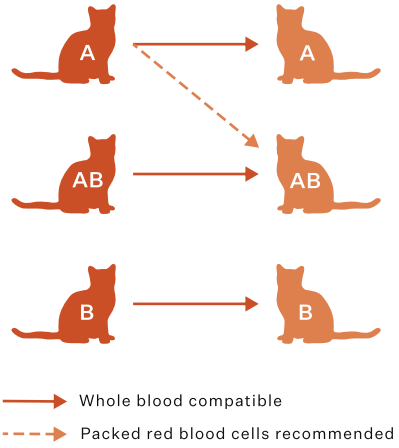
Interpreting feline blood types

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.

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 typing[1]. 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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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.

Progressive Retinal Atrophy (Discovered in the Bengal)	Gene	Risk Variant	Copies	Inheritance	Result
	KIF3B	G>A	0	AR	**Clear**

Information about the genetic condition

Bengal Progressive Retinal Atrophy is characterized by an early-onset degeneration of the retinal photoreceptors with a rapid progression to blindness. The rod photoreceptors degenerate first with reduced rod function seen at about seven weeks of age. The cone photoreceptors degenerate next with reduced cone function seen at about nine weeks of age. Signs of disease include dilated pupils, a hyper-reflective tapetum and attenuated blood vessels. Visual deficits are behaviorally evident in cats by one year of age with night vision affected first. Early indications of visual compromise may include disorientation and lack of awareness of changes to the surroundings. 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 Bengal Progressive Atrophy mutation can be safely bred with a clear cat with no copies of the Bengal Progressive Atrophy mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Bengal Progressive Atrophy 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 Bengal Progressive Atrophy mutation could develop due to a different genetic or clinical cause.

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Health conditions known in the breed

	Gene	Risk Variant	Copies	Inheritance	Result
Pyruvate Kinase Deficiency	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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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	TYRP	b^{l}	0	No effect

Coat Type

	Gene	Variant	Copies	Result
Long Hair (Discovered in many breeds)	FGF5	M4	0	No effect
Long Hair (Discovered in the Norwegian Forest Cat)	FGF5	M2	0	No effect
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^{TX}	0	No effect

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Coat Type

	Gene	Variant	Copies	Result
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 Two copies of the Glitter variant are needed for the glitter coat to be seen.	Pending	gl	1	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

Factor XII Deficiency (Variant 2)	Gene	Risk Variant	Copies	Inheritance	Result
	F12	Deletion	1	ARa	Carrier

Information about the genetic condition

Blood coagulation is a complex process involving many pathways. Factor XII, a plasma protein, classically initiates the intrinsic pathway of blood coagulation; although, there are alternative, slower ways to initiate this pathway. Factor XII Deficiency, also known as Hageman Factor Deficiency or Hageman trait, is a commonly inherited blood clotting disorder in cats. Unlike other bleeding disorders, cats deficient in Factor XII are asymptomatic and do not tend to show spontaneous bleeding or abnormal bleeding after surgery or trauma. However, affected individuals can have prolonged clotting time on the activated partial thromboplastin time (aPTT) screening test. Cats who inherit 2 copies of both Factor XII Deficiency (Variant 1) and Factor XII Deficiency (Variant 2) may show even higher aPTT values. Please note that 1 copy of Factor XII Deficiency (Variant 1) and 1 copy of Factor XII Deficiency (Variant 2) will not cause Factor XII Deficiency.

Breeder recommendation

This condition is autosomal recessive, asymptomatic, meaning that cats with two copies of the variant will show the variant-associated condition but will not suffer disease due to this genetic cause. Current understanding is that a cat with one or two copies of the Factor XII Deficiency variant can be safely bred with a cat with zero, one or two copies of the variant. Please note: It is possible that clinical signs similar to the ones caused by the Factor XII Deficiency mutation could develop due to a different genetic or clinical cause.

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Acute Intermittent Porphyria (Variant 1)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyria (Variant 2)	HMBS	G>A	0	AD	Clear
Acute Intermittent Porphyria (Variant 3)	HMBS	Insertion	0	AD	Clear
Acute Intermittent Porphyria (Variant 4)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyria (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 Porphyria	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
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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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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

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Glossary of genetic terms

Test result definitions

At Risk: Based on the disorder's mode of inheritance, the cat inherited a number of genetic variant(s) which increases the cat's risk of being diagnosed with the associated disorder.

Carrier: The cat inherited one copy of a genetic variant when two copies are usually necessary to increase the cat's risk of being diagnosed with the associated disorder. While carriers are usually not at risk of clinical expression of the disorder, carriers of some complex variants may be associated with a low risk of developing the disorder.

Notable: Inheriting two copies of the genetic variant is noteworthy for specific aspects of health and breeding of the cat, but the cat should otherwise not suffer disease due to this genetic cause when in absence of other genetic variants.

Clear: The cat did not inherit the genetic variant(s) associated with the disorder and will not be at elevated risk of being diagnosed with the disorder due to this genotype. However, similar clinical signs could develop from different genetic or clinical causes.

Inconclusive: An inconclusive result indicates a confident call could not be made based on the data for that genetic variant. Health testing is performed in replicates, and on occasion the outcomes do not agree. This may occur due to an unusual sequence of DNA in the region tested, multiple cell genotypes present due to chimerism or acquired mutations, or due to quality of the DNA sample.

Inheritance mode definitions

Autosomal Recessive (AR): For autosomal recessive disorders, cats with two copies of the genetic variant are at risk of developing the associated disorder. Cats with one copy of the variant are considered carriers and are usually not at risk of developing the disorder. However, carriers of some complex variants grouped in this category may be associated with a low risk of developing the disorder. Cats with one or two copies may pass the disorder-associated variant to their kittens if bred.

Autosomal Recessive, asymptomatic (ARa): For autosomal recessive, asymptomatic disorders, cats with two copies of the variant can exhibit certain aspects of the variant-associated disorder but otherwise, they should not suffer clinical disease as typically expected with autosomal recessive disorders. Cats with one copy of the variant are called carriers and should not exhibit any aspect of the disorder. However, cats with one or two copies may pass the disorder-associated variant to their kittens if bred.

Autosomal Dominant (AD): For autosomal dominant disorders, cats with one or two copies of the genetic variant are at risk of developing the associated disorder. Inheriting two copies of the variant may increase the risk of development of the disorder or cause the condition to be more severe. These cats may pass the disorder-associated variant to their kittens if bred.

X-linked Recessive (XR): For X-linked recessive disorders, the genetic variant is found on the X chromosome. Female cats must inherit two copies of the variant to be at risk of developing the condition, whereas male cats only need one copy to be at risk. Males and females with any copies of the variant may pass the disorder-associated variant to their kittens if bred.

Modifier (MO): Genetic modifiers do not cause disease on their own but can cause disease or change the onset or severity of a disorder when combined with another disorder-associated variant. For some modifier variants only one copy is required to cause an effect, for others two copies are required. Please refer to the associated variant's breeder recommendations regarding safe breeding practices for each modifier variant.