ALISA



Breed: Russian Blue **Birth date:** 2024-01-10

Registration number: 0501-03051335

Test date: 2024-10-24 ID kit: FWGBDLS

ALISA's Profile

Pet information

Registered name Sex ALISA F

Owner reported breedDate of birthRussian Blue2024-01-10

Genetic Diversity

ALISA's Percentage of Heterozygosity

25%

Health summary

At Risk O conditions

Carrier 0 conditions

Clear 50 conditions

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Genetic Diversity

Heterozygosity

ALISA's Percentage of Heterozygosity

25%

This may make her more susceptible to genetic health complications when compared with other Russian Blues.

Typical Range for Russian Blues

26% - 34%

Optimal Selection

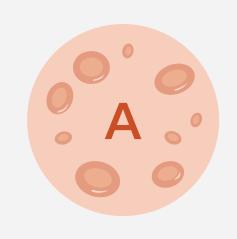
POWERED BY WISDOM PANEL*

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Blood Type



Blood type Genotype*
Type A (Most common) A/A

Transfusion risk

 $\ensuremath{\mathsf{ALISA}}$ has the most common blood type. She can be transfused with Type A blood.

Breeding risk

✓ Low

If breeding, ALISA 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

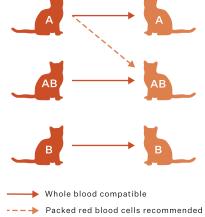
About blood type determination

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 variants.

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 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.



Optimal Selection

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Traits

Coat Color

| | Gene | Variant | Copies | Result |
|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|---------|--------|-----------------------------|
| Charcoal (Discovered in the Bengal) | ASIP | APb | 0 | No effect |
| Solid Color Two copies of the Solid Color variant are needed for a cat to have solid colored hair. However, orange coloration overrides this effect, meaning that cats with partial or full orange coats can show tabby patterning in orange areas. Cats with zero or one copy of this variant are likely to have a tabby pattern due to color banding of the hairs. | ASIP | а | 2 | Solid color hairs likely |
| Gloving (Discovered in the Birman) | KIT | Ma | 0 | No effect |
| Partial and Full White | KIT | W or ws | 0 | No effect |
| Amber (Discovered in the Norwegian Forest Cat) | MC1R | е | 0 | No effect |
| Russet (Discovered in the Burmese) | MC1R | er | 0 | No effect |
| Dilution Two copies of the Dilution variant are required to have a lightening effect on the coat. | MLPH | d | 2 | Lightened coat color likely |
| Albinism (Discovered in Oriental breeds) | TYR | Ca | Ο | No effect |
| Colorpoint (Discovered in the Burmese) | TYR | Cp | 0 | No effect |
| Colorpoint (Discovered in the Siamese) | TYR | Ce | 0 | No effect |
| Mocha (Discovered in the Burmese) | TYR | Cm | 0 | No effect |
| Chocolate | TYRP | b | 0 | No effect |
| Cinnamon | TYRP | рı | Ο | 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 |



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Coat Type

| | Gene | Variant | Copies | Result |
|-------------------------------------------------------|---------|------------------|--------|-----------|
| Long Hair (Discovered in the Ragdoll and Maine Coon) | FGF5 | МЗ | 0 | No effect |
| Long Hair (Discovered in the Ragdoll) | FGF5 | M1 | Ο | No effect |
| Lykoi Coat (Variant 1) | HR | hr ^{Ca} | 0 | No effect |
| Lykoi Coat (Variant 2) | HR | hr ^{VA} | Ο | No effect |
| Hairlessness (Discovered in the Sphynx) | KRT71 | re ^{hr} | Ο | 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) | Т | C1199del | 0 | No effect |
| Short Tail (Variant 2) | Т | T988del | 0 | No effect |

Extra Toes

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



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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 | O | 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 |
| 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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| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|--------------------------------------------------------------------|-------|--------------|--------|-------------|--------|
| 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 | O | XR | Clear |
| Hyperoxaluria Type II | GRHPR | G>A | O | 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 | Ο | MO | Clear |
| Mucopolysaccharidosis Type VII (Variant 1) | GUSB | G>A | O | AR | Clear |
| Mucopolysaccharidosis Type VII (Variant 2) | USB | C>T | O | 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 Abyssinian) | CEP290 | T>G | 0 | AR | Clear |
| 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 |
| Pyruvate Kinase Deficiency | PKLR | G>A | 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 | O | 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 guality 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