# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18
**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Zari's Profile

### Pet information

**Registered name**
Zari

**Sex**
F

**Owner reported breed**
Bengal

**Date of birth**
2022-08-18

### Genetic Diversity

**Zari's Percentage of Heterozygosity**
34%

### Health summary

At Risk 0 conditions

Carrier 1 condition
- Factor XII Deficiency (Variant 2)

Clear 49 conditions

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## Genetic Diversity

### Heterozygosity

#### Zari's Percentage of Heterozygosity

34%

Zari'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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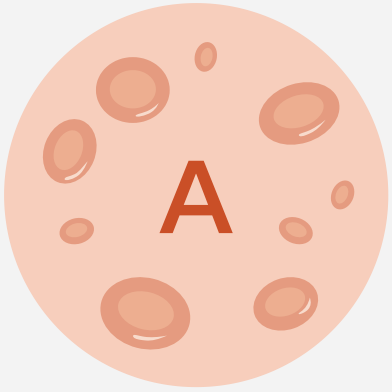
## Blood Type




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

**Genotype***
A/A

**Transfusion risk**
⚠ Moderate

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

**Breeding risk**
Low

If breeding, Zari 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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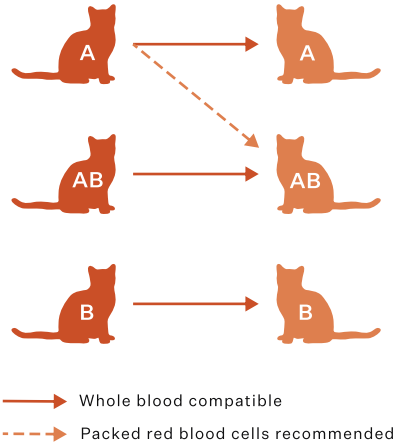
# Interpreting feline blood types

### 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.

# Zari

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## Health conditions known in the breed

### 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.

### Acute Intermittent Porphyria (Variant 1)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| HMBS | Deletion | 0 | AD | Clear |

#### Information about the genetic condition

Acute Intermittent Porphyria (AIP) is a hereditary disorder caused by the decreased activity of the hydroxymethylbilane synthase enzyme needed in the formation and excretion of porphyrins. Porphyrins, in combination with iron, form heme which then combines with other substances to make material that is essential for the normal function of cells. This decreased enzymatic activity leads to accumulation of its substrates in various tissues. Clinical signs are characterized by the brownish discoloration of the teeth and brownish urine. While these discolorations may be the only clinical signs for some, other affected cats develop more severe symptoms, including lethargy, anorexia, anemia, decreased hemoglobin, decreased iron, renal disease, and enlargement of the spleen and liver. Fluorescence of the bones and teeth is a specific diagnostic feature seen in affected cats. Various causative mutations for the disease have been found in cats, with this particular form of porphyria inherited in an autosomal dominant manner.

#### Breeder recommendation

This disease is autosomal dominant meaning that one copy of the mutation is needed for disease signs to occur. Use of cats with one or two copies of the disease mutation is not recommended, as there is a risk that the resulting litter will contain affected kittens. For example if a cat with one copy of the AIP mutation is bred with a clear cat with no copies of the AIP mutation, about half of the kittens will have one copy and half will have no copies of the AIP mutation. Please note: It is possible that disease signs similar to the ones caused by the AIP mutation could develop due to a different genetic or clinical cause.

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# Health conditions known in the breed

## Acute Intermittent Porphyria (Variant 2)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| HMBS | G>A | 0 | AD | **Clear** |

### Information about the genetic condition

Acute Intermittent Porphyria (AIP) is a hereditary disorder caused by the decreased activity of the hydroxymethylbilane synthase enzyme needed in the formation and excretion of porphyrins. Porphyrins, in combination with iron, form heme which then combines with other substances to make material that is essential for the normal function of cells. This decreased enzymatic activity leads to accumulation of its substrates in various tissues. Clinical signs are characterized by the brownish discoloration of the teeth and brownish urine. While these discolorations may be the only clinical signs for some, other affected cats develop more severe symptoms, including lethargy, anorexia, anemia, decreased hemoglobin, decreased iron, renal disease, and enlargement of the spleen and liver. Fluorescence of the bones and teeth is a specific diagnostic feature seen in affected cats. Various causative mutations for the disease have been found in cats, with this particular form of porphyria inherited in an autosomal dominant manner.

### Breeder recommendation

This disease is autosomal dominant meaning that one copy of the mutation is needed for disease signs to occur. Use of cats with one or two copies of the disease mutation is not recommended, as there is a risk that the resulting litter will contain affected kittens. For example if a cat with one copy of the AIP mutation is bred with a clear cat with no copies of the AIP mutation, about half of the kittens will have one copy and half will have no copies of the AIP mutation. Please note: It is possible that disease signs similar to the ones caused by the AIP mutation could develop due to a different genetic or clinical cause.

## Acute Intermittent Porphyria (Variant 5)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| HMBS | G>A | 0 | AR | **Clear** |

### Information about the genetic condition

Acute Intermittent Porphyria (AIP) is a hereditary disorder caused by the decreased activity of the hydroxymethylbilane synthase enzyme needed in the formation and excretion of porphyrins. Porphyrins, in combination with iron, form heme which then combines with other substances to make material that is essential for the normal function of cells. This decreased enzymatic activity leads to accumulation of its substrates in various tissues. Clinical signs are characterized by the brownish discoloration of the teeth and brownish urine. While these discolorations may be the only clinical signs for some, other affected cats develop more severe symptoms, including lethargy, anorexia, anemia, decreased hemoglobin, decreased iron, renal disease, and enlargement of the spleen and liver. Fluorescence of the bones and teeth is a specific diagnostic feature seen in affected cats. Various causative mutations for the disease have been found in cats, with this particular form of porphyria inherited in an autosomal recessive manner.

### 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 AIP mutation can be safely bred with a clear cat with no copies of the AIP mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the AIP 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 AIP mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

### Congenital Adrenal Hyperplasia

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| CYP11B1 | G>A | 0 | AR | **Clear** |

#### Information about the genetic condition

Congenital Adrenal Hyperplasia is a disorder that occurs when a cytochrome P450 adrenal enzyme (11b-hydroxylase) is rendered ineffective by the mutation. This results in inadequate glucocorticoid production by the adrenal glands. The clinical signs are caused partially by the inadequate cortisol levels in the body as well as from the impact of complex effects on hormonal feedback mechanisms. Kittens affected by the disease have high blood pressure and urinate and drink excessively. Other clinical signs include genital abnormalities, early male puberty, presence of secondary sex characteristics post-neutering, small body size, greasy coat, and thickened skin. Affected cats may show behavioral abnormalities (such as intercat aggression).

#### 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 CAH mutation can be safely bred with a clear cat with no copies of the CAH mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the CAH 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 CAH mutation could develop due to a different genetic or clinical cause.

### Congenital Erythropoietic Porphyria

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| UROS | G>A | 0 | AR | **Clear** |

#### Information about the genetic condition

Congenital Erythropoietic Porphyria (CEP) is a hereditary disorder caused by the decreased activity of a specific enzyme in the biosynthetic pathway of heme. Affected cats have decreased activity of the uroporphyrinogen-III-synthase enzyme which leads to accumulation of its substrates in various tissues, such as bone and teeth. Similar to Acute Intermittent Porphyria (AIP), the disorder is characterized by the brownish discoloration of the teeth and brownish urine. Bloodwork may show polychromasia. Fluorescence of teeth is a specific diagnostic feature seen in affected cats.

#### 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 CEP mutation can be safely bred with a clear cat with no copies of the CEP mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the CEP 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 CEP mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

| Cystinuria Type 1A | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | SCL3A1 | C>T | 0 | AR | **Clear** |

### Information about the genetic condition

Cystinuria is a metabolic disorder characterized by the formation of cystine calculi and stones in the urinary tract. The disease is caused by defective renotubular reabsorption of amino acids (arginine, lysine, cystine, and ornithine) resulting in the formation of urinary cystine crystals, urolithiasis, and urinary tract obstruction in some cases. Clinical signs may develop as early as two months of age; however, some individuals may not show signs until middle-age. Clinical signs include nonspecific signs of feline lower urinary tract disease, such as stranguria, pollakiuria, and hematuria. Urolithiasis has potential to cause urinary obstruction. Urinary obstructions require rapid intervention to prevent subsequent development of renal failure. Urinary stones usually require surgical removal. Not all cystinuric cats will form cystine crystals or uroliths, and cats with a later onset of clinical signs may have a milder degree of cystinuria than cats developing signs at an early age. The recurrence of uroliths is very high in affected cats. In addition to the clinical signs of lower urinary tract disease, cystinuric cats may show neurologic signs such as hypersalivation, lethargy, and even seizures. Various causative mutations have been found in cats with equal frequency in males and females.

### 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 Feline Cystinuria mutation can be safely bred with a clear cat with no copies of the Feline Cystinuria mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Feline Cystinuria 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 Feline Cystinuria mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

| **Cystinuria Type B (Variant 1)** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | SCL7A9 | C>T | 0 | AR | **Clear** |

### Information about the genetic condition

Cystinuria is a metabolic disorder characterized by the formation of cystine calculi and stones in the urinary tract. The disease is caused by defective renotubular reabsorption of amino acids (arginine, lysine, cystine, and ornithine) resulting in the formation of urinary cystine crystals, urolithiasis, and urinary tract obstruction in some cases. Clinical signs may develop as early as two months of age; however, some individuals may not show signs until middle-age. Clinical signs include nonspecific signs of feline lower urinary tract disease, such as stranguria, pollakiuria, and hematuria. Urolithiasis has potential to cause urinary obstruction. Urinary obstructions require rapid intervention to prevent subsequent development of renal failure. Urinary stones usually require surgical removal. Not all cystinuric cats will form cystine crystals or uroliths, and cats with a later onset of clinical signs may have a milder degree of cystinuria than cats developing signs at an early age. The recurrence of uroliths is very high in affected cats. In addition to the clinical signs of lower urinary tract disease, cystinuric cats may show neurologic signs such as hypersalivation, lethargy, and even seizures. Various causative mutations have been found in cats with equal frequency in males and females.

### 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 Feline Cystinuria mutation can be safely bred with a clear cat with no copies of the Feline Cystinuria mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Feline Cystinuria 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 Feline Cystinuria mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

| **Cystinuria Type B (Variant 2)** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | SCL7A9 | G>A | 0 | AR | **Clear** |

### Information about the genetic condition

Cystinuria is a metabolic disorder characterized by the formation of cystine calculi and stones in the urinary tract. The disease is caused by defective renotubular reabsorption of amino acids (arginine, lysine, cystine, and ornithine) resulting in the formation of urinary cystine crystals, urolithiasis, and urinary tract obstruction in some cases. Clinical signs may develop as early as two months of age; however, some individuals may not show signs until middle-age. Clinical signs include nonspecific signs of feline lower urinary tract disease, such as stranguria, pollakiuria, and hematuria. Urolithiasis has potential to cause urinary obstruction. Urinary obstructions require rapid intervention to prevent subsequent development of renal failure. Urinary stones usually require surgical removal. Not all cystinuric cats will form cystine crystals or uroliths, and cats with a later onset of clinical signs may have a milder degree of cystinuria than cats developing signs at an early age. The recurrence of uroliths is very high in affected cats. In addition to the clinical signs of lower urinary tract disease, cystinuric cats may show neurologic signs such as hypersalivation, lethargy, and even seizures. Various causative mutations have been found in cats with equal frequency in males and females.

### 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 Feline Cystinuria mutation can be safely bred with a clear cat with no copies of the Feline Cystinuria mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Feline Cystinuria 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 Feline Cystinuria mutation could develop due to a different genetic or clinical cause.

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# Health conditions known in the breed

## Cystinuria Type B (Variant 3)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| SCL7A9 | T>A | 0 | AR | **Clear** |

### Information about the genetic condition

Cystinuria is a metabolic disorder characterized by the formation of cystine calculi and stones in the urinary tract. The disease is caused by defective renotubular reabsorption of amino acids (arginine, lysine, cystine, and ornithine) resulting in the formation of urinary cystine crystals, urolithiasis, and urinary tract obstruction in some cases. Clinical signs may develop as early as two months of age; however, some individuals may not show signs until middle-age. Clinical signs include nonspecific signs of feline lower urinary tract disease, such as stranguria, pollakiuria, and hematuria. Urolithiasis has potential to cause urinary obstruction. Urinary obstructions require rapid intervention to prevent subsequent development of renal failure. Urinary stones usually require surgical removal. Not all cystinuric cats will form cystine crystals or uroliths, and cats with a later onset of clinical signs may have a milder degree of cystinuria than cats developing signs at an early age. The recurrence of uroliths is very high in affected cats. In addition to the clinical signs of lower urinary tract disease, cystinuric cats may show neurologic signs such as hypersalivation, lethargy, and even seizures. Various causative mutations have been found in cats with equal frequency in males and females.

### 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 Feline Cystinuria mutation can be safely bred with a clear cat with no copies of the Feline Cystinuria mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Feline Cystinuria 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 Feline Cystinuria mutation could develop due to a different genetic or clinical cause.

## Dihydropyrimidinase Deficiency

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| DPYS | G>A | 0 | AR | **Clear** |

### Information about the genetic condition

Dihydropyrimidinase (DHP) Deficiency is a rare metabolic syndrome characterized by dihydropyrimidinuria. The DHP enzyme is utilized in various metabolic pathways, including the breakdown of pyrimidine bases (uracil and thymine) as well as certain drugs. The disease causes the substrates of this enzyme to accumulate in the body. These substrates are also seen in the urine of affected cats. Clinical signs of the disease include lethargy, weakness, vomiting, and hyperammonemia. These clinical signs worsen when the cat is fed a high-protein diet. Affected cats may also show signs of malabsorption and malnutrition.

### 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 DHP mutation can be safely bred with a clear cat with no copies of the DHP mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the DHP 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 DHP mutation could develop due to a different genetic or clinical cause.

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# Health conditions known in the breed

| **Earfold and Osteochondrodysplasia (Discovered in the Scottish Fold)** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | TRPV4 | G>T | 0 | AD | **Clear** |

### Information about the genetic condition

Scottish Fold cats are bred for their uniquely folded ears, a feature which starts to occur in kittens around four weeks of age. Unfortunately, the same variant that causes this ear phenotype also causes a skeletal condition called osteochondrodysplasia. Osteochondrodysplasia is a condition that causes abnormal development of joints and cartilage and results in skeletal malformations. Previously, the inheritance pattern was thought to be recessive but today it is known to be codominant. Cats with two copies of the genetic mutation (homozygotes) develop severe, progressive osteoarthritis early in life with radiographic evidence of lesions present as early as seven weeks of age. Cats with one copy of the mutation (heterozygotes) develop more variable signs of the disease, ranging from mild osteoarthritis to a more moderate to severe form. The mild form of the disease usually develops in late middle age or the geriatric years while the moderate to severe form of the disease develops in early adulthood with radiographic evidence of lesions present as early as six months of age. Typical findings include shortened and malformed metatarsal and metacarpal bones, narrowed joint spacing in the distal limbs, and secondary development of degenerative joint disease. The tail is also often shortened and inflexible. These changes can cause short, misshapen limbs, abnormal posture and gait, lameness, and pain. The disease is progressive and incurable and many severely affected cats are euthanized early in life due to welfare concerns.

### Breeder recommendation

This condition is autosomal dominant meaning that one copy of the mutation is needed for signs to occur. If the desire is to produce cats with the breed defining trait of folded ears, it is advised to only breed carrier cats with one copy of the Earfold mutation to clear cats with no copies of the Earfold mutation. This will decrease the likelihood of kittens in the litter developing the severe form of the disease. About half of the kittens will have one copy of the Earfold mutation and half will have no copies of the Earfold mutation. Carrier to carrier matings are not advised as the resulting litter will likely contain kittens with two copies of the Earfold mutation and these kittens are more likely to develop the severe form of the disease. Suspected carriers should be tested prior to breeding. Please note: It is possible that disease signs similar to the ones caused by the Earfold mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

### Factor XII Deficiency (Variant 1)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| F12 | Deletion | 0 | ARa | **Clear** |

#### 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.

### Glutaric Aciduria Type II

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| ETFDH | T>G | 0 | AR | **Clear** |

#### Information about the genetic condition

Glutaric Aciduria Type II, also called Multiple Acyl-CoA Dehydrogenation Deficiency, is a hereditary mitochondrial fatty-acid oxidation disorder. Clinical signs are typically present before 6 months of age. The disease causes vomiting, hypoglycemia, hyperammonemia, diagnostic organic aciduria, and accumulation of medium- and long-chain fatty acids in plasma. Other clinical signs may include a lack of appetite and seizures.

#### 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 Glutaric Aciduria Type II mutation can be safely bred with a clear cat with no copies of the Glutaric Aciduria Type II mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Glutaric Aciduria Type II 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 Glutaric Aciduria Type II mutation could develop due to a different genetic or clinical cause.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### GM1 Gangliosidosis

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| GLB1 | G>C | 0 | AR | **Clear** |

#### Information about the genetic condition

GM1 Gangliosidosis is a neurodegenerative disorder caused by dysfunction in lysosomal storage. Deficiency of the ß-galactosidase enzyme leads to accumulation of GM2 ganglioside within the lysosomes of neurons. This accumulation then leads to cellular dysfunction, degeneration, and eventual neuronal death. The onset of clinical signs occurs at approximately three to five months of age. The first clinical signs are mild intention tremors. The disease rapidly progresses to severe ambulatory difficulties, seizures, and blindness. Affected kittens are usually euthanized on welfare grounds by ten months of age.

#### 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 GM1 Gangliosidosis mutation can be safely bred with a clear cat with no copies of the GM1 Gangliosidosis mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the GM1 Gangliosidosis 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 GM1 Gangliosidosis mutation could develop due to a different genetic or clinical cause.

### GM2 Gangliosidosis

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| GM2A | Deletion | 0 | AR | **Clear** |

#### Information about the genetic condition

GM2 Gangliosidoses are a group of neurodegenerative disorders caused by dysfunction in lysosomal storage. There are different types and causes of the disease. This form of the disease is caused by deficiency of the GM2 activator protein. The disease leads to accumulation of GM2 ganglioside within the lysosomes of neurons. Accumulation leads to cellular dysfunction, degeneration, and eventually to neuronal death. Clinical signs include progressive neurologic symptoms. The onset of clinical signs occurs at approximately twelve to fourteen months of age. The first clinical signs include ataxia, intention tremors, and an exaggerated startle reflex. The disease progresses to severe ambulatory difficulties with loss of coordination, difficulty eating, and blindness.

#### 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 GM2A mutation can be safely bred with a clear cat with no copies of the GM2A mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the GM2A 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 GM2A mutation could develop due to a different genetic or clinical cause.

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

# Health conditions known in the breed

| GM2 Gangliosidosis Type II (Discovered in Domestic Shorthair cats) | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | HEXB | Insertion | 0 | AR | **Clear** |

### Information about the genetic condition

GM2 Gangliosidoses are a group of neurodegenerative disorders caused by dysfunction in lysosomal storage. There are different types and causes of the disease. This form of the disease is caused by reduced activity of the beta-hexosaminidase enzyme. Deficiency of this enzyme leads to accumulation of GM2 ganglioside within the lysosomes of neurons. This accumulation then leads to cellular dysfunction, degeneration, and eventual neuronal death. Clinical signs include progressive neurologic symptoms and visual defects. The onset of clinical signs occurs at approximately six to eight weeks of age. The first clinical signs are mild intention tremors. The disease rapidly progresses to severe ambulatory difficulties due to whole body tremors, severe ataxia, loss of balance, quadriparesis, difficulty eating, and blindness. Affected cats are usually smaller than normal in body size and weight. Affected kittens are usually euthanized on welfare grounds by six months of age. Five different mutations in the HEXB gene have been found as causative mutations for the disease, all in different 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 GM2 Gangliosidosis mutation can be safely bred with a clear cat with no copies of the GM2 Gangliosidosis mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the GM2 Gangliosidosis 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 GM2 Gangliosidosis mutation could develop due to a different genetic or clinical cause.

| Hemophilia B (Variant 1) | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | F9 | C>T | 0 | XR | **Clear** |

### Information about the genetic condition

Blood coagulation is a complex process. Factor IX is one of the proteins necessary for blood coagulation and its deficiency causes Hemophilia B in an affected cat. Hemophilia B is clinically indistinguishable from Hemophilia A, and in some individuals may cause life-threatening bleeding. The disease follows an X-linked mode of inheritance. Given males only have one X chromosome, the condition is seen more commonly in males as a single copy of the mutation will cause the condition; while females require two copies of the mutation in order to exhibit the condition. Clinical signs of the disease are often noticeable before one year of age and include intermittent lethargy, anorexia, and fever. Affected cats can show prolonged bleeding after trauma or surgical intervention. Lameness and signs of pain may occur due to bleeding inside joints and other tissues. Swelling and bruising under the skin may be observed. Depending on severity, anemia may also occur due to the loss of blood, contributing to weakness, shortness of breath, and irregular heartbeats.

### Breeder recommendation

This disorder is X-linked recessive, meaning the genetic variant is found on the X chromosome. Given males only have one X chromosome, a single affected copy will increase the risk of being diagnosed with the disorder. Females typically require two copies to be at an elevated risk. Use of cats with one or two copies of the variant is not recommended for breeding as there is a risk that the resulting litter will contain affected kittens. Please note: It is possible that clinical signs similar to the ones caused by this variant could develop due to a different genetic or clinical cause.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### Hemophilia B (Variant 2)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| F9 | G>A | 0 | XR | **Clear** |

#### Information about the genetic condition

Blood coagulation is a complex process. Factor IX is one of the proteins necessary for blood coagulation and its deficiency causes Hemophilia B in an affected cat. Hemophilia B is clinically indistinguishable from Hemophilia A, and in some individuals may cause life-threatening bleeding. The disease follows an X-linked mode of inheritance. Given males only have one X chromosome, the condition is seen more commonly in males as a single copy of the mutation will cause the condition; while females require two copies of the mutation in order to exhibit the condition. Clinical signs of the disease are often noticeable before one year of age and include intermittent lethargy, anorexia, and fever. Affected cats can show prolonged bleeding after trauma or surgical intervention. Lameness and signs of pain may occur due to bleeding inside joints and other tissues. Swelling and bruising under the skin may be observed. Depending on severity, anemia may also occur due to the loss of blood, contributing to weakness, shortness of breath, and irregular heartbeats.

#### Breeder recommendation

This disorder is X-linked recessive, meaning the genetic variant is found on the X chromosome. Given males only have one X chromosome, a single affected copy will increase the risk of being diagnosed with the disorder. Females typically require two copies to be at an elevated risk. Use of cats with one or two copies of the variant is not recommended for breeding as there is a risk that the resulting litter will contain affected kittens. Please note: It is possible that clinical signs similar to the ones caused by this variant could develop due to a different genetic or clinical cause.

### Hyperoxaluria Type II

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| GRHPR | G>A | 0 | AR | **Clear** |

#### Information about the genetic condition

Hyperoxaluria is a rare hereditary renal disorder. Clinical signs of the disorder are due to nephrocalcinosis and renoliths caused by excessive oxalate accumulation in the kidneys. This causes a reduction in renal function. Affected kittens may show muscular atrophy, dehydration, anorexia, and weakness which may present prior to or after the onset of renal dysfunction. Affected kittens develop acute end-stage renal disease typically between five and nine months of age.

#### 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 Hyperoxaluria type II mutation can be safely bred with a clear cat with no copies of the Hyperoxaluria type II mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Hyperoxaluria type II 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 Hyperoxaluria type II mutation could develop due to a different genetic or clinical cause.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

| **Lipoprotein Lipase Deficiency** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | LPL | G>A | 0 | AR | **Clear** |

### Information about the genetic condition

Lipoprotein Lipase Deficiency (also called chylomicronemia) results from a deficiency of the lipoprotein lipase enzyme. Lipoprotein lipase is crucial in lipoprotein and lipid metabolism. Deficiency of this enzyme leads to impaired uptake and use of fatty acids for energy metabolism. Clinical signs of the disease can be observed in some cats at birth and are, otherwise, often noticeable before one year of age. Affected kittens may show reduced birth weight and slow growth rates, while adults have reduced body mass and body fat. Affected queens may also have an increased number of stillborn kittens. Additional clinical signs include lethargy, anorexia, and xanthomata (lipid granulomas in the skin and internal organs). More severely affected individuals may show various neuropathies (such as facial paralysis, Horner's syndrome, radial and/or tibial nerve paralysis) and lipemia retinalis. Less commonly seen clinical signs include anemia and splenomegaly. Most affected individuals will have a fasting hyperchylomicronemia. Lipoprotein analysis of affected cats will show elevated triglyceride levels, even when fed a low fat diet; a finding that can be shared with some carriers of this mutation. Total cholesterol and HDL-cholesterol ratio is also elevated in affected cats.

### 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 LLD mutation can be safely bred with a clear cat with no copies of the LLD mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the LLD 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 LLD mutation could develop due to a different genetic or clinical cause.

# Zari

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**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### MDR1 Medication Sensitivity

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| ABCB1 | Deletion | 0 | AR | **Clear** |

#### Information about the genetic condition

Cats with this variant are asymptomatic until exposed to a medication that uses the drug transport pump rendered defective by the mutation in the MDR1 (also called ABCB1) gene. Drugs known to use this P-glycoprotein pump are macrocyclic lactones including eprinomectin-containing products labeled for use in cats (antiparasitic drugs), loperamide (antidiarrheal), erythromycin (antibiotic), acepromazine (tranquilizer), butorphanol (opioid), certain drugs used in cancer treatment (vincristine, vinblastine, doxorubicin), and possibly others still to be determined. When these medications are administered, they accumulate in the brain which results in adverse reactions. Typical symptoms involve generalized neurologic dysfunction which may include mydriasis, dyspnea, tremors, hyperreactivity, ataxia or paresis. In more severe cases cats may experience seizures, coma and death. However, with appropriate supportive care by a veterinarian, most affected cats may be able to fully recover.

#### Breeder recommendation

Further research is needed to determine if cats with one copy of the variant may have altered drug responses. At this time, breeding cats with one or two copies of the MDR1 Medication Sensitivity variant should be approached with caution. If a cat with one copy of the MDR1 Medication Sensitivity variant is bred with a clear cat with no copies of the MDR1 Medication Sensitivity variant, on average half of the kittens will have one copy and half will have no copies of the MDR1 Medication Sensitivity variant. If a cat with two copies of the MDR1 Medication Sensitivity variant is bred with a clear cat with no copies of the MDR1 Medication Sensitivity variant, the resulting kittens will all have one copy of the MDR1 Medication Sensitivity variant. If litters are expected to contain kittens with the MDR1 Medication Sensitivity variant, the kittens should be DNA tested as they may show signs of sensitivity to some common medications. Carrier to carrier matings are not advised as the resulting litter may contain kittens with two copies of the MDR1 Medication Sensitivity variant, which is known to cause medication sensitivity. Please note: It is possible that clinical signs similar to the ones caused by the MDR1 Medication Sensitivity variant could develop due to a different genetic or clinical cause.

### Mucopolysaccharidosis Type I

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| IDUA | Deletion | 0 | AR | **Clear** |

#### Information about the genetic condition

Mucopolysaccharidosis Type 1 (MPSI) is a lysosomal storage disease caused by deficient activity of the alpha-L-iduronidase enzyme, which is used to break down dermatan and heparan sulfates. This results in the accumulation of glycosaminoglycans (GAGs) in various types of cells which eventually progresses to cellular damage. The disorder varies from mild to severe, with severe cases carrying a grave prognosis at an early age. Clinical signs typically develop in kittens from eight weeks to six months of age. These signs may include failure to thrive, growth retardation, skeletal deformities, lameness, facial dysmorphism, ataxia, and tremors. Affected cats can also have visually smaller ears than normal cats and thick skin over the dorsal neck. Additionally, some affected cats may show corneal cloudiness and/or have a cardiac murmur due to mitral insufficiency. MPSI follows an autosomal recessive mode of inheritance.

#### 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 MPS1 mutation can be safely bred with a clear cat with no copies of the MPS1 mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the MPS1 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 MPS1 mutation could develop due to a different genetic or clinical cause.

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

# Health conditions known in the breed

## Mucopolysaccharidosis Type VI

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| ARSB | T>C | 0 | AR | **Clear** |

### Information about the genetic condition

Mucopolysaccharidosis Type VI is a rare lysosomal storage disease caused by deficient activity of the N-acetylgalactosamine-4-sulfatase enzyme, which is used to break down dermatan and chondroitin sulfates. This results in the accumulation of glycosaminoglycans (GAGs) in various types of cells which eventually progresses to cellular damage. The typical form of the disease causes dwarfism, reduced flexibility, facial dysmorphia, corneal clouding, degenerative joint disease, and abnormal leukocyte inclusions (with prominent cytoplasmic granules). Affected cats may also have heart valve thickening. Clinical signs typically first appear in kittens at six to eight weeks of age and affected individuals tend to remain cognitively normal. MPS VI follows an autosomal recessive mode of inheritance. Cats with two copies of this variant (also known as T1427C mutation) exhibit the typical form of the disease as described above. Cats with one copy of the Mucopolysaccharidosis Type VI variant (T1427C mutation) and one copy of the Mucopolysaccharidosis Type VI Modifier variant (often referred to as the G1558A mutation) have an increased risk of presenting with a mild form of the disease, manifesting as degenerative joint disease only.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the variant are needed for disease signs to be shown. A cat with one copy of the Mucopolysaccharidosis Type VI variant and zero copies of the Mucopolysaccharidosis Type VI Modifier variant can be safely bred with a clear cat with no copies of these variants. About half of the kittens will have one copy of the Mucopolysaccharidosis Type VI variant and will be considered carriers. 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 MPSVI variant could develop due to a different genetic or clinical cause.

## Mucopolysaccharidosis Type VI Modifier

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| ARSB | G>A | 0 | MO | **Clear** |

### Information about the genetic condition

Mucopolysaccharidosis Type VI is a lysosomal storage disease caused by a deficiency of an enzyme which is essential in breaking down dermatan and chondroitin sulfates. Cats with one copy of the Mucopolysaccharidosis Type VI Modifier variant (also referred to as the G1558A mutation) and one copy of the Mucopolysaccharidosis Type VI variant (known as the T1427C mutation) display a mild form of the disease expressed as a higher incidence of degenerative joint disease than that experienced by non-affected cats. Clinical signs of disease have not been associated with cats who have one or two copies of the modifier variant (G1558A mutation) and zero copies of the Mucopolysaccharidosis Type VI variant (T1427C mutation). These cats have a normal physical appearance and exhibit normal growth. An incidental finding of increased granularity within white blood cells of cats with two copies of the Mucopolysaccharidosis Type VI Modifier variant has been reported with unknown clinical relevance.

### Breeder recommendation

Current understanding is that a cat with one or two copies of the modifier variant can be safely bred with a cat with zero, one or two copies of the modifier variant, as long as both cats are clear for the Mucopolysaccharidosis Type VI variant. Please note: It is possible that disease signs similar to the ones caused by the Mucopolysaccharidosis Type VI variant could develop due to a different genetic or clinical cause.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### Mucopolysaccharidosis Type VII (Variant 1)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| GUSB | G>A | 0 | AR | **Clear** |

#### Information about the genetic condition

Mucopolysaccharidosis Type VII (MPS VII) is a lysosomal storage disease caused by deficient activity of the beta-glucuronidase enzyme. Deficiency of the beta-glucuronidase enzyme leads to accumulation of glycosaminoglycans (GAGs) in various types of cells which eventually progresses to cellular damage. Clinical signs of the disease are typically evident at eight weeks of age. Affected kittens exhibit stunted growth and several skeletal deformities including dysmorphic facial features. Affected cats develop progressive paralysis/paresis of the limbs and clouding of the corneas. The prognosis of the disease is grave due to its progressive nature. In humans, MPS VII is known as Sly syndrome.

#### 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 MPS7 mutation can be safely bred with a clear cat with no copies of the MPS7 mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the MPS7 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 MPS7 mutation could develop due to a different genetic or clinical cause.

### Mucopolysaccharidosis Type VII (Variant 2)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| USB | C>T | 0 | AR | **Clear** |

#### Information about the genetic condition

Mucopolysaccharidosis Type VII (MPS VII) is a lysosomal storage disease caused by deficient activity of the beta-glucuronidase enzyme. Deficiency of the beta-glucuronidase enzyme leads to accumulation of glycosaminoglycans (GAGs) in various types of cells which eventually progresses to cellular damage. Clinical signs of the disease are typically evident at eight weeks of age. Affected kittens exhibit stunted growth and several skeletal deformities including dysmorphic facial features. Affected cats develop progressive paralysis/paresis of the limbs and clouding of the corneas. The prognosis of the disease is grave due to its progressive nature. In humans, MPS VII is known as Sly syndrome.

#### 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 MPS7 mutation can be safely bred with a clear cat with no copies of the MPS7 mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the MPS7 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 MPS7 mutation could develop due to a different genetic or clinical cause.

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

# Health conditions known in the breed

## Myotonia Congenita

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| CLCN1 | G>T | 0 | AR | **Clear** |

### Information about the genetic condition

Myotonia Congenita is a muscular disorder characterized by the inability of muscles to relax after contraction. The disorder is caused by the defective activity of chloride channels in the cell membranes of skeletal muscles. Affected cats have an enlarged tongue, limited range of motion in the jaws, and exhibit drooling. Additional clinical signs include a short-strided, stiff gait and enlarged neck and forelimb musculature. Halitosis (bad breath), various dental abnormalities and poor grooming habits have also been described in affected cats. Physical exam findings may include blepharospasm following testing of the menace response or palpebral reflexes and initially normal proprioceptive positioning that worsens with repetitive testing. Clinical signs of the disease are usually evident in kittens before 1 year of age.

### 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 Myotonia Congenita mutation can be safely bred with a clear cat with no copies of the Myotonia Congenita mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Myotonia Congenita 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 Myotonia Congenita mutation could develop due to a different genetic or clinical cause.

## Polycystic Kidney Disease (PKD)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| PKD1 | C>A | 0 | AD | **Clear** |

### Information about the genetic condition

Polycystic Kidney Disease (PKD), also named autosomal dominant PKD, is characterized by variously sized, fluid-filled cysts in the renal cortex and medulla with hepatic and pancreatic cysts also possible. The cysts develop from birth and enlarge with age. The cysts destroy the renal parenchyma and disturb renal function, eventually causing renal failure. Affected cats present with signs of renal insufficiency such as weight loss, decreased appetite, increased drinking and urination, poor body condition, and vomiting. Biochemical labwork and ultrasonography examination are helpful tools in identifying the severity of disease within an affected individual. An autosomal dominant point mutation in the PKD1 gene has been identified as the most common genetic mutation for the disease. No homozygous cats have been identified, suggesting the mutation is a homozygous lethal mutation in utero. PKD is very common in Persian and Persian-related cats, affecting approximately 38% of Persian cats worldwide. While there is no known sex linkage to the inheritance of the mutation, research has shown male cats have a higher prevalence of the mutation.

### Breeder recommendation

This disease is autosomal dominant meaning that one copy of the mutation is needed for disease signs to occur. Use of cats with one or two copies of the disease mutation is not recommended, as there is a risk that the resulting litter will contain affected kittens. For example if a cat with one copy of the PKD mutation is bred with a clear cat with no copies of the PKD mutation, about half of the kittens will have one copy and half will have no copies of the PKD mutation. Please note: It is possible that disease signs similar to the ones caused by the PKD mutation could develop due to a different genetic or clinical cause.

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

# 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.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### 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.

### Sphingomyelinosis (Variant 1)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| NPC1 | G>C | 0 | AR | **Clear** |

#### Information about the genetic condition

Sphingomyelinosis (Variant 1) disorder is a lysosomal storage disease that is due to the lack of sphingomyelinase enzyme production. This results in an accumulation of sphingomyelin and cholesterol within lysosomes of neurons and other soft tissue cell types, such as liver, spleen, kidneys and lungs. Clinical signs in cats may be evident as young as nine to twelve weeks of age. Affected kittens exhibit intention tremors of the head and body and may demonstrate a progressive hypermetric gait. Other features of the disease include enlargement of the spleen and liver along with changes to the lungs. The progression of the disease is rapid, leading to severe ataxia and motor dysfunction with an eventual inability to move and stand. Some affected individuals display a characteristic chewing motion and may have seizures. The prognosis for the disease is grave due to its progressive nature.

#### 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 Sphingomyelinosis mutation can be safely bred with a clear cat with no copies of the Sphingomyelinosis mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Sphingomyelinosis 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 Sphingomyelinosis mutation could develop due to a different genetic or clinical cause.

# Zari

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Bengal
**Birth date:** 2022-08-18

**Test date:** 2025-01-08
**ID kit:** FCWRJVT

## Health conditions known in the breed

### Sphingomyelinosis (Variant 2)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| NPC2 | G>A | 0 | AR | **Clear** |

#### Information about the genetic condition

Sphingomyelinosis (Variant 2) disorder is a lysosomal storage disease that is due to the lack of sphingomyelinase enzyme production. This results in an accumulation of sphingomyelin and cholesterol within lysosomes of neurons and other soft tissue cell types, such as liver, spleen, kidneys and lungs. Clinical signs in cats are usually evident at three months of age. Affected kittens exhibit intention tremors of the head and body and have a reduced menace response. Additionally, epileptiform seizures may appear. Other features of the disease may include enlargement of the spleen and liver along with changes to the lungs. The progression of the disease is rapid, leading to severe ataxia and motor dysfunction with an inability to move and stand by eight to ten months of age. The prognosis of the disease is grave due to its progressive nature.

#### 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 Sphingomyelinosis mutation can be safely bred with a clear cat with no copies of the Sphingomyelinosis mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Sphingomyelinosis 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 Sphingomyelinosis mutation could develop due to a different genetic or clinical cause.

### Vitamin D-Dependent Rickets

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| CYP27B1 | G>T | 0 | AR | **Clear** |

#### Information about the genetic condition

Vitamin D-Dependent Rickets is a disorder characterized by abnormal conversion of dietary vitamin D into its biologically active form calcitriol (D3). The lack of the biologically active vitamin D3 causes impaired bone mineralization. Clinical signs of the disease are usually evident before five months of age. The disease causes softening of bones which leads to increased susceptibility to fractures and bone deformities. The disease is characterized by growth retardation. Affected cats usually find the condition painful and are unwilling to move. Affected cats may also have delayed or abnormally developed secondary dentition and may have constipation. In severe hypocalcemia cases, epileptiform seizures may occur. Three causative mutations for the disease have been found in Domestic Shorthair cats. The mode of inheritance for all of these mutations is autosomal recessive.

#### 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 VDDR mutation can be safely bred with a clear cat with no copies of the VDDR mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the VDDR 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 VDDR mutation could develop due to a different genetic or clinical cause.

# Zari

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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 | $hr^{Ca}$ | 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**<br>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 |

# Zari

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**ID kit:** FCWRJVT

## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Acute Intermittent Porphyria (Variant 3)** | HMBS | Insertion | 0 | AD | Clear |
| **Acute Intermittent Porphyria (Variant 4)** | HMBS | Deletion | 0 | AD | 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 Myasthenic Syndrome (Discovered in the Devon Rex and Sphynx)** | COLQ | G>A | 0 | AR | Clear |
| **Familial Episodic Hypokalemic Polymyopathy (Discovered in the Burmese)** | WNK4 | C>T | 0 | AR | Clear |
| **Glycogen Storage Disease (Discovered in the Norwegian Forest Cat)** | GBE1 | 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 |
| **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 |
| **Progressive Retinal Atrophy (Discovered in the Persian)** | AIPL1 | C>T | 0 | AR | Clear |
| **Spinal Muscular Atrophy (Discovered in the Maine Coon)** | LIX1 | Deletion | 0 | AR | Clear |

**Breed:** Bengal
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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.