Asia

Breed: British Shorthair
Birth date: 2024-07

Test date: 2025-08-13
ID kit: FWGZDCQ

Asia's Profile

Pet information

Registered name Asia

Sex F

Owner reported breed British Shorthair

Date of birth 2024-07

Genetic Diversity

Asia's Percentage of Heterozygosity
39%

Health summary

At Risk	0 conditions
Carrier	0 conditions
Clear	50 conditions

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Health conditions known in the breed

Autoimmune Lymphoproliferative Syndrome (Discovered in British Shorthair)	Gene	Risk Variant	Copies	Inheritance	Result
	FASL	Insertion	0	AR	Clear

Information about the genetic condition

Autoimmune Lymphoproliferative Syndrome (ALPS) is a rare, severe disorder that has been described in British Shorthair cats. The disorder is caused by the inability of the body to destroy unneeded T-lymphocytes, which leads to massive accumulation of T-lymphocytes within lymph nodes. Diffuse enlargement of lymph nodes is typically noticed in kittens around six weeks of age. The progression of clinical signs is rapid and affected kittens develop severe abdominal distension, lethargy, and weight loss. Regenerative anemia is also typically present. No effective treatment has been described and affected kittens are typically euthanized for humane reasons before four 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 Autoimmune Lymphoproliferative Syndrome mutation can be safely bred with a clear cat with no copies of the Autoimmune Lymphoproliferative Syndrome mutation. About half of the kittens will have one copy (carriers) and half will have no copies of the Autoimmune Lymphoproliferative Syndrome 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 Autoimmune Lymphoproliferative Syndrome mutation could develop due to a different genetic or clinical cause.

Familial Episodic Hypokalemic Polymyopathy (Discovered in the Burmese)	Gene	Risk Variant	Copies	Inheritance	Result
	WNK4	C>T	0	AR	Clear

Information about the genetic condition

Familial Episodic Hypokalemic Polymyopathy is caused by excessive potassium loss into the urine which results in symptomatic hypokalemia. Clinical signs usually develop by 2 to 10 months of age. The most characteristic sign of the disease is passive ventroflexion of the head and neck, which is almost pathognomonic but not every cat has this distinct presentation and, thus, diagnosis can be difficult. Further signs include skeletal muscle weakness occurring only in the forelimbs while in others the weakness affects mainly the hindlimbs; and, in others still, appendicular weakness may affect all four limbs. Affected cats may also have muscle tremors, head bobbing, lameness, and alterations in gait. Hypokalemia is linked to muscle pain (myalgia) which can have further variable clinical presentations. If the disease is not diagnosed early enough, the cat may develop incapacitating weakness with an inability to walk, seizures, or cardiopulmonary arrest. The disease has a variable, episodic nature and worsens with stress.

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

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Traits Coat

Color

	Gene	Variant	Copies	Result
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	a	1	Banded hairs, tabby patterns likely
Dilution Two copies of the Dilution variant are required to have a lightening effect on the coat.	MLPH	d	1	No effect
Colorpoint (Discovered in the Siamese) Two copies of this variant result in a colorpoint pattern, although this can be blocked by other variants. Cats with one copy of the Colorpoint (Discovered in the Burmese) variant and one copy of the Colorpoint (Discovered in the Siamese) variant will show a darker base coat color and less contrasting colorpoint pattern than cats with two copies of the Colorpoint (Discovered in the Siamese) variant.	TYR	c^s	2	Siamese colorpoint pattern likely
Chocolate Cats with either two copies of the Chocolate variant or one copy of the Chocolate variant and one copy of the Cinnamon variant are likely to have a chocolate coat color.	TYRP	b	1	No effect

Coat Color

	Gene	Variant	Copies	Result
Cinnamon Two copies of the Cinnamon variant result in cinnamon coat color.	TYRP	b^l	1	No effect

Coat Type

	Gene	Variant	Copies	Result
Long Hair (Discovered in many breeds) Two copies of any Long Hair variant must be inherited for a cat to have a long coat. This can either be two copies of a particular variant, such as this one, or two of any combination of Long Hair variants.	FGF5	M4	1	Long coat possible, short coat likely

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Acute Intermittent Porphyria (Variant 1)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyria (Variant 2)	HMBS	G>A	0	AD	Clear
Acute Intermittent Porphyria (Variant 3)	HMBS	Insertion	0	AD	Clear
Acute Intermittent Porphyria (Variant 4)	HMBS	Deletion	0	AD	Clear
Acute Intermittent Porphyria (Variant 5)	HMBS	G>A	0	AR	Clear
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
Glutaric Aciduria Type II	ETFDH	T>G	0	AR	Clear
Glycogen Storage Disease (Discovered in the Norwegian Forest Cat)	GBE1	Insertion	0	AR	Clear
GM1 Gangliosidosis	GLB1	G>C	0	AR	Clear

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Other health conditions tested

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

Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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	0	AR	Clear

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.

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