Copper Toxicosis Genetics

What is Copper Toxicosis

Copper is an essential mineral involved in nerve function, skin health, and respiration. The liver regulates copper metabolism, and when copper accumulates excessively, it causes liver inflammation and chronic hepatitis. If chronic hepatitis progresses to cirrhosis, it can be fatal.

Genetic Markers

Currently, research in other breeds has identified 2 different genes that can cause copper toxicosis, COMMD1 and ATP7B.

COMMD1
Bedlington Terriers

Research in Bedlington Terriers identified the COMMD1 gene, which affects intracellular copper handling and biliary copper excretion. Mutations cause copper buildup in liver cells.

COMMD1 is an autosomal recessive inherited disorder, meaning dogs need two mutated copies of the gene (one from each parent) to develop the disease.

This disease is not the same as Copper Storage Disease (Wilson's Disease in humans)

A DNA test exists for Bedlingtons, and selective breeding has greatly reduced—though not eliminated—cases. Remaining cases may involve other genetic or environmental factors.

ATP7B (ATP7A)
Labrador Retriever (and Doberman)

Labradors have two known mutations linked to copper toxicosis:

ATP7B: Impairs copper excretion into bile, causing liver accumulation. Also associated with Wilson's Disease in humans.

- In dogs with two copies, 80% showed high levels of copper in the liver. And they showed the highest of the copper levels.
- Dogs with one copy also show elevated copper compared to clear dogs.
- ATP7B acts as an autosomal incomplete dominant mutation with incomplete penetrance—one copy increases risk, two copies increase it further.

ATP7A: Reduces intestinal copper absorption (which can cause copper deficiency).

- Acts as a modifier gene, partially protecting dogs that also carry ATP7B.

Written by Ruth Thompson

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- In the podcast by Dr. Hille Fieten, she referred to as the “A for Awesome” gene for its protective effect.

Some Labradors without ATP7B mutations still show high copper, suggesting other genetic factors or environmental/dietary copper exposure.

What about Keeshond?

Keeshonden do experience copper toxicosis, but no current scientific evidence supports COMMD1, ATP7B, or ATP7A as reliable marker genes in this breed. Ongoing studies are investigating whether ATP7B or other genes contribute to copper toxicosis in additional breeds.

This handout is for educational purposes only and does not replace professional veterinary advice.

Source Material

The Functional Dog Collaborative Podcast, March 25, 2025, Episode 68 with Dr. Hille Fieten, Copper Toxicity. The Functional Breeding Podcast.

Listen to the podcast here: https://podcasts.apple.com/ca/podcast/dr-hille-fieten-copper-toxicity/id1513602865?i=1000700773087

The Veterinary Journal 311 (2025) 106335
Serum miR-30b is increased in Labrador Retrievers with elevated copper levels
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