



20th November 2017

Hereditary Cerebellar Ataxia in Norwegian Buhunds

Geneticists at the Kennel Club Genetics Centre at the Animal Health Trust (AHT), in collaboration with the AHT's Neurology unit, have identified a mutation that causes hereditary cerebellar ataxia in Norwegian Buhunds, and are very pleased to announce the launch of a DNA test for this mutation.

Dogs affected by this disease show clinical signs as early as 12 weeks of age, and suffer from ataxia (uncoordinated movement) and head tremors. The disease is progressive, worsening over time. There is currently no effective treatment and Norwegian Buhunds diagnosed with hereditary cerebellar ataxia are often euthanised on welfare grounds.

Hereditary cerebellar ataxia is recessive in Norwegian Buhunds, which means that two copies of the mutation need to be inherited by a dog (one from each parent) for it to be clinically affected. If two carriers are mated there is a chance that affected puppies will be produced, and DNA testing is the best way to prevent this from happening.

A DNA test for hereditary cerebellar ataxia in Norwegian Buhunds will become available from the AHT DNA Testing Service on Monday 4th December 2017 at www.ahtdnatesting.co.uk.

The AHT's research tested 146 Norwegian Buhunds for which no signs of cerebellar ataxia had been reported. This consisted of three sets of dogs: 70 UK dogs for which samples were collected between 2008 and 2015 (born 1994 – 2013), 36 UK dogs collected specifically for this research in 2017 (born 2008 – 2016), and 40 dogs from Finland (born 1998 – 2012). Whilst the UK set of dogs for which samples were collected between 2008 and 2015 included significantly more carriers than the other two sets, carriers of the mutation were identified in the contemporary UK set and the set from Finland. This shows that this mutation is still present within the Norwegian Buhund population. All Norwegian Buhunds affected by cerebellar ataxia that have been tested for this mutation as part of this research have two copies of the mutation. None of the 146 unaffected dogs included in the study had two copies of the mutation.

More information can be found on our website:

http://www.aht.org.uk/cms-display/genetics_success.html

<http://www.aht.org.uk/genetics>