Late onset ataxia (LOA) in the Parson Russell terrier (PRT) and Jack Russell terrier (JRT) is a disease of incoordination of gait and lack of balance. The onset age for the disease is usually between 6 months and 1 year of age, when owners may start to notice that their dog is showing changes in gait pattern (often weaving of the hind limbs) and some difficulty balancing. The disease is progressive and affected dogs become increasingly uncoordinated with difficulty balancing, which makes moving around and everyday tasks such as going up and down stairs difficult. There is no treatment or cure for LOA and affected dogs are often euthanized, typically around two years after onset, on humane grounds as their quality of life diminishes. The Animal Health Trust canine genetics research group has identified the mutation causing LOA.

The AHT also offer a test for Spinocerebellar Ataxia with or without Myokymia and Seizures for the Parson Russell terrier (PRT) and Jack Russell terrier (JRT) known as the SCA test, please see below under Other forms of ataxia for details.

**DNA testing**

CLEAR: These dogs have two normal copies of DNA and are likely to be clear of LOA. The results of our research suggest that there may be other causes of ataxia in the breed so we cannot exclude the formal possibility that clear dogs could develop a genetically different form of ataxia due to other mutations that are not detected by this test.

CARRIER: These dogs have one copy of the LOA associated mutation and one normal copy of DNA. These dogs will not develop LOA themselves as a result of the LOA mutation but they will pass the mutation on to approximately 50% of their offspring. The results of our research suggest that there may be other causes of ataxia in the breed so we cannot exclude the formal possibility that carriers could develop a genetically different form of ataxia due to other mutations that are not detected by this test.

AFFECTED: These dogs have two copies of the LOA associated mutation and have a very high chance of developing LOA.

The test will effectively reduce the number of LOA cases in the PRT. We encourage owners to keep us updated on the health of dogs they have tested. We are particularly interested to hear from owners of dogs that:

i) are clinically affected with ataxia but that do not have two copies of the mutation, or

ii) are over 4 years old and remain free from ataxia despite having two copies of the mutation.

This information will help us to monitor the effectiveness of the test, to possibly refine the test in the future if necessary and to start to investigate additional cause(s) of ataxia in PRTs and JRTs.

Samples submitted should be cheek swabs (a non-invasive sampling method). Sampling kits are obtainable from the Animal Health Trust webshop [www.ahtdnatesting.co.uk/canine_tests](http://www.ahtdnatesting.co.uk/canine_tests). Further information can be obtained by emailing [dnatesting@aht.org.uk](mailto:dnatesting@aht.org.uk)
**Other forms of ataxia**

**Spinocerebellar Ataxia (SCA) with or without Myokymia and Seizures**
The mutation for this condition was identified by the University of Missouri and a DNA test is available both through the Animal Health Trust and University of Missouri. Affected dogs also show signs of cerebellar ataxia as early as 2-6 months of age. At post-mortem examinations, degeneration can be found in the areas of the spinal cord that carry information to the cerebellum, hence the term spino-cerebellar ataxia. The coordination difficulties also progress, but in addition other signs can develop. The majority of cases also develop myokymia, an involuntary twitching of the muscles. The myokymia also becomes progressively worse with age and can result in episodes of generalized muscle spasms and over-heating. In addition, a small percentage of dogs with SCA have true epileptic seizures, some as young as 10 weeks of age. Most dogs with SCA are also euthanized young due to poor quality of life.

**Neonatal Granuloprival Ataxia (NGA)**
Another form of ataxia called Neonatal Granuloprival Ataxia (NGA) is also hereditary but the mutation has not yet been identified. As the name implies, coordination difficulties in dogs with NGA are apparent from the time they begin to walk, which clearly distinguishes it from the other forms. In these dogs, an area of the cerebellum called the granular layer degenerates. There is currently no test for this condition.

**Other cases**
Both the University of Missouri and the Animal Health Trust have found a small number of dogs reported as suffering from cerebellar ataxia which began after weaning age that do not have either of the known mutation. This may represent another form of hereditary ataxia or they may represent an acquired cause of the disease.

**Conclusion**
The Animal Health Trust and the University of Missouri have discovered different mutations which cause similar, but distinct forms of the disease in Jack/Parson Russell Terriers. We are working to investigate those cases of ataxia which do not have either of the known mutations.

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