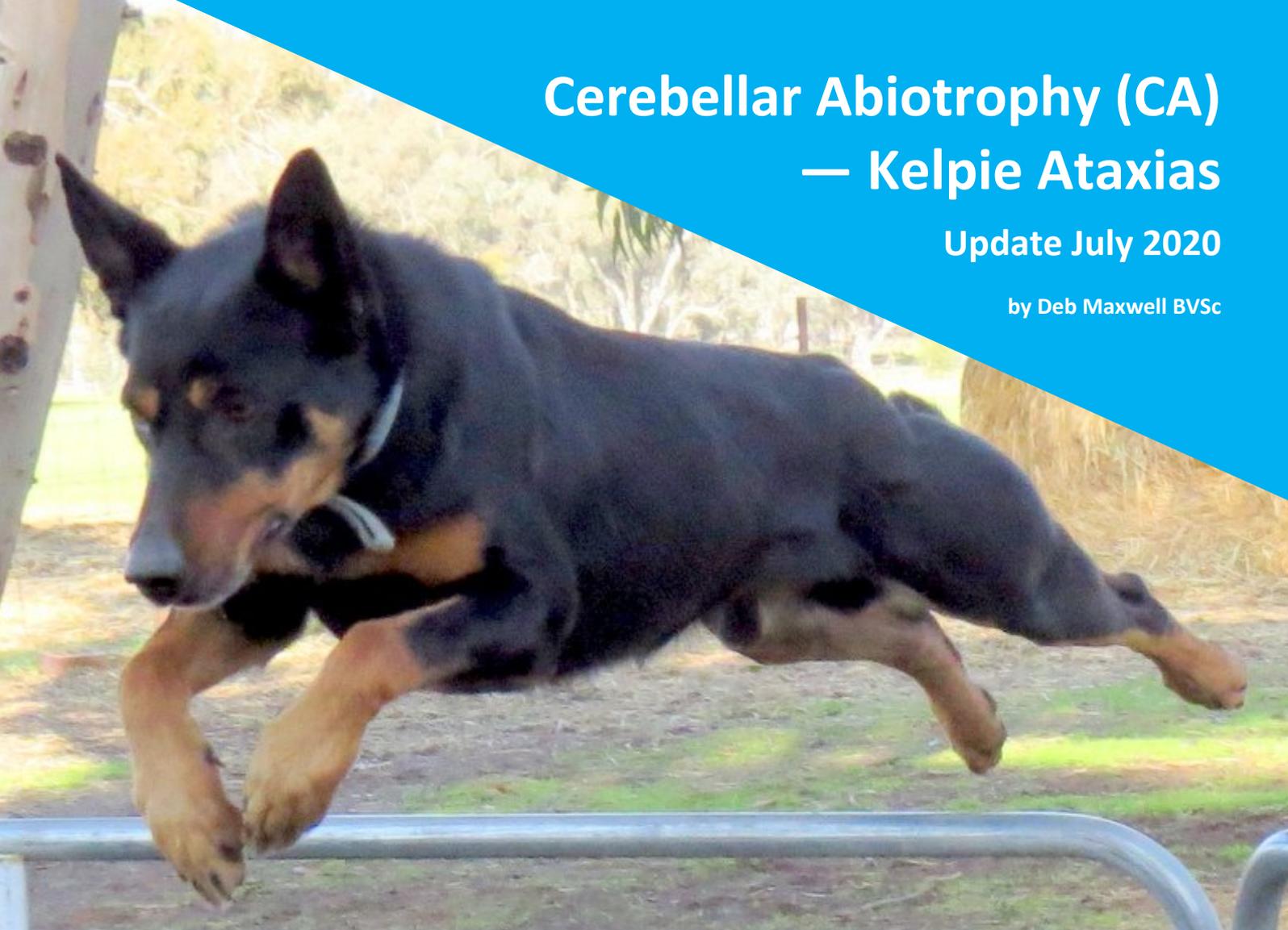


Cerebellar Abiotrophy (CA) — Kelpie Ataxias

Update July 2020

by Deb Maxwell BVSc



With tests now available for CA, the number of CA affected pups being born can be greatly reduced.

In every mating, for each CA type, one parent must be clear.

Cerebellar Abiotrophy (CA) in Kelpies causes ataxia and other difficulties with movement. There are three or more genetic causes, and while DNA tests have been developed, there are still unknowns. This article presents the knowledge to date, but continuing research and the data from wider commercial testing will build on this knowledge and may result in changes to tests and future recommendations.

Nevertheless, the current tests can greatly reduce the number of potentially affected pups.

The same disease variants may also affect ANKC Kelpies, Border Collies and Koolies, as well as crosses of these breeds, due to their common ancestors and occasional cross-breeding. Tests run by the researchers on about 200 Border Collies (mainly ANKC) did not show CA markers. Since the research, the Late- and Early-onset CA has been confirmed through commercial tests in some Border Collies; CA affected Koolies require testing to confirm these.

Cover image: Steven Robinson's Kelpie, Barcoola Kiff, showing the coordination and athleticism of Kelpies, which would not be possible for dogs affected by CA. Image by Jen from Shoot My Style Photography.

There are no images of CA affected dogs included because it is a disease of movement and difficult to capture with a static photograph.



Summary

- Cerebellar Abiotrophies (CA) are incurable, inherited diseases in Kelpies with the majority identified by three known and unrelated genetic markers.
- Signs include ataxia, high stepping [hypermetria], wide stance, incoordination, falling over, difficulty jumping onto objects, fine tremors, a nodding head, difficulty eating or drinking from a bowl, and, occasionally, seizures.
- The disease is not common, with the level of CA markers in the population remaining low, but two carriers may be chosen as mates and produce affected pups, and linebreeding/inbreeding can increase the chance of this occurring.
- Some affected dogs studied were not explained by these three markers; potentially there may be one or more other genes causing CA in Kelpies or there are other genes that affect whether the disease is fully expressed, or other markers may be better indicators, in particular, with the Early-onset variant.
- Dogs with a pair of CA markers are positive or “affected” and most will have signs of ataxia. Carriers only have one copy of any CA marker, and will not be affected.
- The extent of signs in affected dogs varies and is not yet able to be predicted; some dogs can live a relatively normal life; others will need to be euthanised.
- Signs in affected dogs vary in time of onset according to the CA variant: Early-onset from 4–8 weeks, Late-onset from 3–8 months (but sometimes later); the German type CA from 4–8 weeks.
- DNA tests are available to identify whether a dog has any of the markers for the three known CA disease variants, indicating clear, carrier or affected status.
- **The tests vary in their ability to identify affected and carrier dogs:**
 - For Late-onset CA, the test is believed to identify all affected and carrier dogs.
 - **For Early-onset CA, the current test misses some dogs; any dogs that the current test identifies as carriers or affected, are carriers or affected, however some affected dogs with Early-onset CA used in the research were not identified by this marker.** DNA tested with the current marker contributes to CA, but may not be the direct cause. An alternative marker that appears to have higher predictive power is being studied.
 - For the German type CA, very few dogs were in the study. Also, two dogs had tests indicating they were affected, but whether signs of CA appeared in them could not be confirmed.
- The DNA tests are currently only available through one Australian testing company: Dog Breeding Science, but this may change in the future.
- Tests can be used to greatly reduce the number of CA affected pups being born by including a clear result for every test, in every mating, every time.
- Carrier dogs with superior working ability do not need to be removed from the breeding pool, simply ensure that their mate is clear for the CA marker they carry.
- Carrier dogs do not show signs of disease; buyers of working dogs not intending to breed them should have no concern about their health.
- **Dogs with negative or “clear” test results should not be represented as CA-free** because there may yet be an unidentified CA variant, and the Early-onset and German CA tests may not be fully expressed or predict all affected dogs.



Cerebellar Abiotrophy —the diseases

One disease or three?

Kelpies, like other breeds of dogs, other animals, and even humans, can suffer from inherited Cerebellar Abiotrophy (CA), which has commonly been called “ataxia”.

But research has proven this is three (possibly more) separate genetic diseases in Kelpies, which result in a similar set of signs, but should be considered independently.

There is an Early-onset variant and a Late-onset variant of CA and these are present internationally in Kelpies. Both have also been confirmed by commercial testing in Border Collies and may be in Koolies as well, due to common ancestors or through occasional cross-breeding of these working breeds. The third variant is the German type, which was only found in a family of Kelpies in Germany, but further testing may show it to be present elsewhere.

What is CA?

Cerebellar Abiotrophy (CA) and ataxia are **not** interchangeable terms despite them being used as such for many years in the Kelpie community. CA describes a physical process in the brain, whereas ataxia is the result of the CA process and is the loss of muscle control and balance leading to a range of signs. But those signs can also occur from other causes; spinal tumours, certain poisons, tick paralysis and brain injury are just some of the other non-genetic causes of ataxia in dogs and, in fact, humans. As such, dog owners should not automatically assume dogs with ataxia have CA.

Cerebellar Abiotrophy is a disease process in the cerebellum—the small back section of the brain responsible for movement and coordination. It results from genetic mutations occurring in various breeds and species of animals.

What are the signs of CA?

A small change in the DNA of CA affected animals results in damage and death to Purkinje and granule cells within parts of the cerebellum; cells that would normally send the signals for fine movement to the muscles. As a result, a dog can show a number of signs: ataxia, high stepping

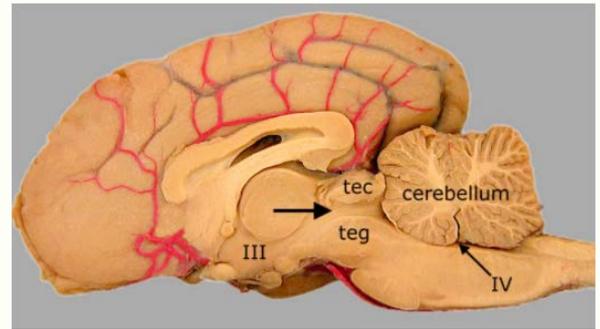


Figure 1. The cerebellum is about 10% of the brain and controls fine movement and coordination. Image provided with permission Minnesota Veterinary Anatomy website (<http://vanat.cvm.umn.edu>).

[hypermetria], wide stance, incoordination, falling over, difficulty jumping onto objects, fine tremors, a nodding head, difficulty eating or drinking from a bowl, and, occasionally, seizures.

Additionally, some of the Late-onset cases had a smaller body size than unaffected litter mates and reduced muscle mass in the legs.

When do the signs appear?

The signs described above take some time to develop; they are not present at birth, and since they are due to separate genetic causes, some have different development times.

The following ages are when signs have typically first appeared in affected dogs studied to date:

- “Early-onset CA”— 4 to 8 weeks old.
- “Late-onset CA”—3 to 8 months old.
- “German CA”— 4 to 8 weeks old.

However, the number of affected dogs provided for research was limited, and more affected dogs have since been identified, in particular, some testing as “Late-onset”, but with signs not showing until between one and two years of age. It is quite possible that the age of onset for each type may broaden as further affected animals are identified.



How severe are the signs?

The severity of signs across the individual dogs also varies. In all cases, there is no cure; the damaged cerebellum does not recover; however, some dogs may learn to adapt and their signs can become less obvious. Also, the signs in some dogs will be quite mild and non-progressive; they advance to a certain extent, but then get no worse, which is typical of the Early-onset condition. In others, the signs are progressive, they gradually worsen until normal functioning is impossible; for instance, inability to stand, intention tremors that make eating and drinking very difficult, and seizures. In severe cases, the dog is best euthanised. Generally, the more severe signs have been noted in Late-onset CA.

Can affected dogs work or lead a normal life?

Because the extent of signs vary considerably, mildly affected dogs may be able to lead a good life as a pet or even a working or sport dog. For instance, fine tremors may be present, but not seriously affect the dog's movement. Others may have signs that require some degree of special care and attention, such as assistance when eating or providing a kennel or bed at ground level if they are unable to jump into a raised box. But there can be severely affected dogs that will have a poor quality of life, even as a pet, and these are best euthanised.

There is currently no way to predict the extent or progression of signs in any individual dog. And unfortunately, the signs may only become apparent, particularly with the Late-onset type, after an owner has bonded with a pup and their training is well underway.

How common are these diseases?

CA is not a common disease. While the CA genes are believed to be present across most lines of Kelpies, the number of these genes is expected to be relatively low, as is the case for any lethal or deleterious gene in other animals and humans. If a gene results in an affected animal not being able to breed or not being chosen for breeding (both of these could apply with CA), the number of the genes diminish in the population to ever-lower levels. Deliberately selecting carrier animals over clear animals will increase the levels, but as there has been no way to differentiate carriers from

clears, they cannot have been intentionally selected on this basis. However, a temporary increase in incidence appeared to occur when one or two carrier sires were widely used, inadvertently, 30 years ago.

Occasionally, two carrier animals will be mated and produce affected offspring—the chance of this increases with inbreeding—and about one quarter of the pups will be affected. However, it would be very unlikely for affected animals to then be used for breeding, and many would not even survive, so this removes some of the genes from the population again.



Figure 2. Potentially, any of these unaffected dogs could be CA carriers. Image Deb Maxwell.

CA genetics and genomics

While many readers, particularly Kelpie breeders, will have a sound understanding of genetics, fewer people are familiar with genomics. Examples of Kelpie coat colour genetics have been included here to help the reader better understand the concepts and terms.

What are the genes and how do they work?

The majority of cases of Cerebellar Abiotrophies in Kelpies result from autosomal recessive genes. The actual causative genes have not yet been found, but they are associated with the VMP1 (Late-onset), LINGO3 (Early-onset), or NUP153 (German type) DNA markers, with the last two types possibly having incomplete penetrance.

Initial research demonstrated that CA in Kelpies was not linked to three genes that cause CA in humans. Later research showed that eleven other CA variants found in fifteen other dogs breeds were also not responsible for CA in Kelpies. This identified that DNA tests available for CA in other dog breeds or humans are of no value to identify CA carrier or CA affected Kelpies.



Researchers subsequently identified three genetic markers associated with CA in Kelpies, which accounted for the majority of cases.

Each of the three known genetic variations causing CA in Kelpies are caused by autosomal genes. Autosomal refers to genes that are not on the X or Y chromosomes, this means they are not “sex-linked” or associated with the dog’s gender.

They are all believed to be recessive alleles (variations) of the genes. Genes work in pairs, but each gene in the pair may not be the same; there may be a number of variations of a gene and these are called alleles.



Figure 3. Two red and tans either side of a black and tan Kelpie. Image Deb Maxwell.

Example: A familiar Kelpie example of an autosomal gene is coat colour. Black and red (at the Brown gene locus) are alleles of the same coat colour gene. Black is the dominant allele and red is the recessive allele. If the pair of genes are both the black allele, the dog will be black. If the pair has one black allele and one red allele, the dog will still be black because the black allele is dominant to (overrides) the recessive red allele, but this dog is a “carrier” for a red coat. It is only when a dog has both of its gene pair as red alleles with no dominant genes to override their expression that it will be a red-coloured dog.

CA genes likely work in a similar way. There is the normal allele for the gene (allowing normal cerebellar function), and a recessive CA allele, (like red coat colour). When there is a pair of normal alleles, the dog will be normal. When it has one normal allele and one CA allele, it will still be normal, but is a “carrier” of that CA variant. However, if both of the gene pair are CA alleles, the dog will be described as “affected” with CA and, generally, will show signs of the disease.

While the VMP1 (Late-onset) variant of CA is fully penetrant, it appears that the variants associated with LINGO3 (Early-Onset), and likely NUP153 (German type) markers may have incomplete penetrance.

This means that every dog with a pair of the VMP1 CA markers will show disease, but some of those with either a pair of LINGO3 CA markers or a pair of NUP153 CA markers may not show disease signs—for some reason the effects of the actual CA alleles are not fully penetrating (they are not expressed in every case). The reason for this is still unknown, but may be due to another separate gene pair overriding the CA expression, or it could mean that the test marker (a known section of different DNA) being used is not quite close enough to the harmful gene (see CA Tests and their use, on the next page).

Incomplete penetrance of a gene can occur due to other modifying genes, epistatic genes (these cover the effect of another gene) or other components across the dog’s genetic material. Kelpie coat colour has two other examples of how genes can modify or override other genes.

Example: Aside from the main coat colour gene, Kelpies also have a Dilution gene that can dilute (lighten) the black or red coat colour. The normal gene (not causing dilution) is dominant and so the presence of two normal genes have no effect on coat colour; the dog will either be black or red. Likewise, when only one of the gene pair is the recessive allele for Dilution, the coat remains unaffected. However, when both of the gene pair are the recessive Dilution alleles then a black coat becomes “blue” (a grey colour) and a red coat becomes “fawn” (light brown).

The dilution gene “modifies” how the black or red coat is expressed. The signs of CA might be similarly modified by other genes, affecting the extent that the CA signs are expressed in some dogs.



Figure 4. A red and tan dam carrying the dilution gene with tan-pointed black, blue, red and fawn pups. Image Deb Maxwell.



Example: Another example shows how one gene can completely mask another. All Kelpies are either black or red as their base colour, as described earlier, but some appear cream-coloured. This is not another allele of coat colour alongside black and red (and it is not related to the dilution gene). Instead, it is due to yet another gene, called the Extension gene, where the recessive allele codes for cream colour in Kelpies and the dominant allele is normal, that is, it does not affect the main coat colour. A dog with no recessive cream alleles will be their normal black or red colour. With one recessive cream allele they will carry cream, but not show it. But if the pair of genes are both the cream alleles, then the black or red colour will be completely overridden and the dog will be cream-coloured.

Likewise, there may be genes that override the expression of CA, completely hiding it in some “affected” dogs.



Figure 5. A red and tan, a cream, and a black and tan Kelpie. Image Deb Maxwell.

The final point to emphasise is that the CA variants identified to date are indeed from three separate and totally unrelated genes shown by the LINGO3 (Early-onset), VMP1 (Late-onset) and NUP153 (German type) markers. These genes have no effect on each other and must be considered separately.

Potentially, one dog could have three different alleles for CA—they could carry the CA allele associated with the LINGO3 marker, one with the VMP1 marker and one with the NUP153 marker. **But these do not add together to make an affected dog.** Such a dog would only be a carrier for the three separate diseases, not showing signs of any of these diseases.

A dog could also be affected by more than one type of CA at the same time if it has inherited both the CA alleles in each of two or even the three variations. While it would be extremely rare, it is possible, as there are dogs already tested that have both LINGO3 and VMP1 CA markers. The NUP153-associated variation of CA has so far only been found in one small family of dogs in Germany, so is less likely to be found in combination with the other CA genes.

CA Tests and their use

What tests are available?

In late 2019, Dog Breeding Science, a commercial DNA-testing laboratory in Sydney, Australia, released three tests for CA in working dogs using the markers identified from the results of the University of Sydney research, specifically, the PhD of Dr Annie Pan. Currently, they are the only commercial provider of CA tests.

As such, this article will refer to the testing done by Dog Breeding Science (DBS), however other laboratories may offer similar tests in the future.

DBS offer DNA marker tests for the LINGO3 (Early-onset), VMP1 (Late-onset) and NUP153 (German type) associated CA variants.

What are marker tests?

Markers are the known variations between dogs at specific positions along the genome (the genome is the complete set of DNA). Finding the actual gene that causes a disease is like finding a needle in a haystack without even knowing what a needle looks like. Instead, scientists initially look at known DNA markers to see if one of their variations is present as a pair (homozygous) in all, or most, clinical cases of the disease. While a marker may not be the causative gene, it can indicate the gene’s presence close by and be used as the test. It can also narrow down the search area to allow discovery of the causative gene.

Canine genome sequencing has already found many thousands of markers. These known variations may be part of a gene or in the DNA between genes. Some marker locations only have two variations, others have many. The test array looks at these markers and which of their variations make up each marker pair.



The CA genes are believed to be recessive, but are still unknown. The scientists ran a marker test array that looked through approximately 170 thousand variations in the genome at the same time, called a genome-wide association study, and they compared the results from affected animals with normal animals to find a marker that consistently showed two copies of the same variation (homozygous) in the affected animals only. If the marker turns out to be the causative gene, it can result in a test that is 100% accurate, providing there are no other complicating factors. Other times the marker turns out to be near the causative gene, and the closeness of the marker to the actual, but as yet, unknown gene will affect the test accuracy.

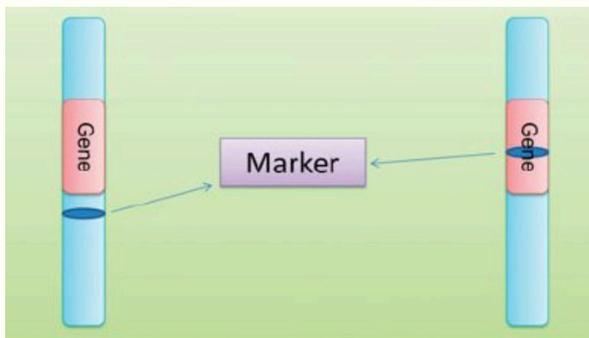


Figure 6. Sometimes the marker is near the gene, other times it is part of the gene.

The closer it is, the better it will predict a carrier or affected animal. Variations along the genome have occurred over a great many years. When ova and sperm cells are created and the DNA duplicates, each pair of chromosomes tangle, break and recombine and some of the matching pieces that break off join back to their opposite matching chromosome. This creates new unique combinations of all those variations along the DNA—that's part of what makes individuals different. The chance of a break occurring between a marker and a gene is very low when they are close together, so a particular variation on the marker, and the variation on a nearby gene, tend to stick together over the years. But as described earlier, there can be other genes that modify or mask the effects of the causative gene.

Do the tests identify all CA carrier or affected dogs?

The University of Sydney research used data from 38 dogs showing signs of CA, as well as 168 dogs that were either related or unlikely to be carrying

CA. Potentially, other dogs with quite different variants of CA exist in the Kelpie population that were not provided to the researchers, and the tests would not identify these animals as carrying or being affected by CA.

Also, different CA variants occur in other dog breeds and these were not found in the Kelpies studied; in future it would be possible for one or more of these different CA genes to be introduced into Kelpies by intentional or accidental cross-breeding, although it is unlikely they would be perpetuated in the Kelpie population if they were from non-working breeds.

Based on the dogs studied, the VMP1 marker appears to identify all carrier and affected dogs for the Late-onset CA variant with no other genes appearing to interact.

The current LINGO3 (Early-onset) marker test has not identified up to half of the affected dogs with Early-onset CA that were in the research project; this would mean some carriers would also not be identified. However, those it does identify as carriers and affected, are carriers and affected. Nevertheless, the test is still considered valuable to reduce the incidence of Early-onset CA.

The researchers have since confirmed that a different nearby marker identifies all of the research cases affected with the Early-onset CA (excluding the German CA variant, which is explained by the NUP153 test). The current LINGO3 (Early-onset) test does not appear to identify the causative gene for CA, but instead it appears to identify another piece of DNA that has a modifying effect on the causative gene. Further work will confirm the value of the other marker as a test for Early-onset CA.

The NUP153 test for the German type CA is more difficult to assess. This variant of CA was found in only one family of affected dogs in Germany and no other dogs used in the research; there were only four affected dogs (all from the one litter). The study identified ten related dogs tested as carriers (one CA marker), but there were also two related dogs that were homozygous (had a pair of the CA markers) for this variant and were initially reported as unaffected, however confirmation that no signs became apparent in these two dogs was **not** able to be established.



As such, some animals identified as affected by the NUP153 marker test may not show signs of CA. At this point, this particular test is likely only of value to specific breeders with dogs related to those where the condition has already been found. The laboratory has indicated they are planning to remove this test from the combined CA panel, but would still have the individual test available if required.

How are samples collected?

All three tests can be carried out at the same time on the one sample from a dog, or they can be tested individually. The CA tests require a sample of DNA from each dog and this can be obtained from cheek (buccal) cells collected onto a cotton bud or swab that is inserted into the dog's mouth and rubbed firmly against the inside of the dog's cheek for a short time. Ensure the laboratory directions are closely followed, particularly in how long the swab/bud should be rubbed against the cheek, as some samples have arrived at the laboratory with insufficient DNA. The swabs or buds should be carefully handled to prevent cross-contamination from other dogs, and are allowed to dry, are sealed into a paper envelope with the dog's and owner's identity on it, and are posted to the laboratory. The payment and the full dog and owner details are completed online.

Once the samples are received, the DNA is extracted, the test is carried out and results are returned within a week or two of the samples arriving.

The current cost for the CA testing provided by Dog Breeding Science (at July 2020) is a \$40 per animal set-up fee plus test costs: \$14 each CA test individually or all three for \$40, totalling \$80 for all three (other tests aside from CA can also be included).

DBS indicated they can also use plucked hair samples. Also, later tests (at the cost of the test only) can be done for dogs already set up in the system. This provides the opportunity to use the current tests now, but to add any further new tests without the set-up fee again. Consult the laboratory for further information on both these matters.

What do the results show?

The results section of each Dog Breeding Science report shows two letters for each test marker, because the dog has these markers in pairs. Table 1, below shows the possible result combinations. Table 2 shows an example of the results table from a Dog Breeding Science CA test report.

Table 1. Possible results from the Dog Breeding Science CA tests.

CA variant and marker	Clear (normal/negative)	Carrier	Affected	Notes
Early-onset CA (LINGO3)	C C	T C	T T	C = Cytosine, the normal marker T = Thymine, the CA marker
German type CA (NUP153)	n n	del n	del del	n = negative/normal marker del = deletion, the CA marker
Late-onset CA (VMP1)	C C	A C	A A	C = Cytosine, the normal marker A = Adenine, the CA marker

Table 2. An example extract from a Dog Breeding Science CA test report showing carrier status for LINGO3 and affected for VMP1.

Test	Genotype	Comments
Cerebellar abiotrophy (LINGO3)	T C	Positive, one copy (CA carrier for CA with signs appearing around 4-8 weeks of age)
Cerebellar abiotrophy (NUP153)	n n	Negative for the CA: NUP153 marker
Cerebellar abiotrophy (VMP1)	A A	Positive, two copies (double CA carrier and probably affected with signs appearing around 3-8 months of age)



How can results be used for breeding?

Carriers of VMP1 (Late-onset) and LINGO3 (Early-onset) CA markers are believed to occur in many Kelpie bloodlines.

The CA-tests will allow breeders to:

- Greatly reduce the number of affected dogs being born for the three known variants.
- Continue to breed outstanding working dogs, even if they are carriers.

Removing outstanding carrier working dogs from the breeding pool is not necessary to prevent CA, and would risk losing superior working traits from the Kelpie gene pool.

In every mating, for each CA type, one parent must be clear.

This greatly reduces the chance that affected dogs will be born, but half of the pups will be carriers if one parent is a carrier. Any pups that turn out exceptional can, in turn, be tested before breeding.

How many pups will be affected, carriers or clear?

The expected proportion of pups of each type, **on average**, from some specific matings, is shown in Table 3, below. However, chance plays a role, just like the occasional litters where all pups are male or all are female, rather than the expected half and half.

Do clear tests guarantee freedom from CA?

The three known tests did not identify every dog affected by CA. Because the possibility that another, yet unidentified, CA variant exists in a small proportion of dogs, and that some of the tests are not 100% accurate as predictors of CA, dogs should not be claimed to be CA-free, even with clear tests. Instead, use the term "Tested CA clear", to indicate that the test markers were negative.

Can a dog be declared clear by parentage (obligate clear)?

If both parents have tested clear, is there any need to test the pups? No, not for your own breeding purposes, but buyers may request the actual pups to be tested. Various other dog breed societies will categorise pups as "clear by parentage" or "obligate clear" when parents have tested clear for the disease in question. Many of these, however, provide an assurance by having strict sample collection and submission protocols, typically by a veterinary surgeon who will also scan and verify microchip details of pups and the parents, and those parents may need to be confirmed through parentage tests. There are no similar protocols in place for Kelpies.

Table 3. Example matings and expected percentage of affected, carrier and clear pups.

Example matings	Proportion of pups (%)				
	Two of the same markers (affected)	One marker (carrier)	Other marker (carrier)	Both markers (carrier)	No markers (clear)
Both parents clear of all CA markers	0	0			100
One parent a carrier for one CA marker, the other parent clear for all CA markers	0	50			50
Both parents are carriers for the same CA marker	25	50			25
One parent a carrier for one CA marker, the other parent a carrier for a different CA marker	0	25	25	25	25
One parent a carrier for two different CA markers, the other parent clear for all CA markers	0	25	25	25	25



Can the CA test results be obtained for dogs to be purchased or used as sires?

Testing is not mandatory. Any request for CA test results for a dog to be purchased or a sire to be used for stud needs to be agreed between the buyer and seller, including the cost of the testing and the consequences if there is a positive test after purchase or use.

Should dogs intended only as workers or pets, not for breeding, be tested?

Generally, Early-onset and German type CA will be evident before a pup is sold at 8 weeks*, or at least, soon after, so testing is not likely warranted for these variants in pups not intended for breeding.

*(*State laws say that pups are not to be sold until 8 weeks old.)*

However, dogs affected by Late-onset CA may not display signs for many months, indeed over a year old, and the purchaser may have already spent considerable time in caring for and training the young dog by then. Having VMP1 test results (which are highly predictive) for the pup or parents would be an advantage for the buyer to determine the likelihood of the pup later being affected by CA.

If only one of the parents tested clear for VMP1 (Late-onset CA) (only one was tested or two were tested, but only one was clear), the pup would be clear or a carrier and testing is not warranted if the pup is not intended for breeding. If both parents were untested or carriers, then a test would accurately indicate whether the pup could be affected in the future.

It is up to the buyers and sellers to agree before a sale on whether testing will be done, by whom, when, who pays and the consequences when the results are returned.

Are carrier dogs healthy?

Pet buyers or livestock owners purchasing a working dog and who don't intend to breed it, should have no concerns buying a dog that is a carrier for CA. Sellers or buyers may choose to desex these dogs (notwithstanding the pros and cons, and costs of desexing) to avoid unplanned matings that may result in affected pups. Many buyers are already open to purchasing desexed dogs, as they are easier to manage and there is no evidence desexing affects working ability.



Figure 7. Barrkel Peter Sinner and Dunyellan Jasper head off a beast. Image: Pilbara Working Dogs.



Figure 8. Pup, GoGetta Try, showing calm strength. Image: Joe Spicer.



Figure 9. Brandy and Zoey keep the mob moving. Image: Eddie Shirley.