Rhodesian Ridgeback Health and Genetics
A Breeders perspective

Vicki Moritz, Ujamaa Ridgebacks

The Rhodesian Ridgeback is a relatively recent breed, first recognised and registered in 1924 by the South African Kennel Club. The recent formation of this breed has provided some genetic vigour, but also diversity in the “type” or “style” of the Ridgeback. In relative terms and in the generalist press the Ridgeback appears to have few genetic issues, and compared with many other pure breed dogs this may be the case. However there is no room for complacency. George Padgett in “Control of Canine Genetic Diseases” listed 26 diseases of genetic origin reported in the Ridgeback1. In a 2002 seminar at the US Ridgeback specialty a further list containing 58 genetic conditions was developed. Padgett’s research showed that any one dog will carry a number of genes that are defective and may cause inherited conditions. His analysis of the Ridgeback determined each Ridgeback carried genes for approximately 6.6 issues.2,3 Experienced breeders know the breed is subject to a considerable number of inherited conditions, many cosmetic, which make the breeding of Ridgebacks suitable for the conformation ring a challenge. This is certainly borne out by Padgett’s analysis and the figures below. The data demonstrates that over 27% of pups born are unsuitable for show or breeding due to ridge issues alone.

Selection of the best breeding stock in Ridgebacks- and pedigree dogs in general- is always a challenge no matter how much research is done on the pedigrees and characteristics of both parents. In addition to the obvious phenotypic factors such as structure, movement etc, relatedness of the dogs (see Coefficient Of Inbreeding sidebar) and inherited diseases must be taken into account. In many cases the inherited conditions are polygenic, that is controlled by a number of genes and expressed variably. This makes elimination of affected animals from the gene pool complex, as they may be carriers who never produce a defect they may be carrying. It is a responsibility of the breeder to weigh up the importance of any genetic defects the animal may be carrying and balance it with the overall quality of the animal. There is no place for kennel blindness in this assessment!

Data on genetic conditions affecting Ridgebacks in Australia was gathered in 1996 and again in 2006. The latter survey was done under the auspices of the National Rhodesian Ridgeback Club with 25 breeders providing information on 1397 dogs. In the USA the Rhodesian Ridgeback Club of the US (RRCUS) conducted Health Surveys in 1996 and then again in 20002. 1763 dogs were represented in the 2000 survey. Selected comparative survey results are as follows, with the US data sourced from the 2002 “Overview of genetic issues” paper by Cynthia Roethel3

<table>
<thead>
<tr>
<th>Disease/Disorder</th>
<th>Percentage affected</th>
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<td>Aust 2006</td>
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<tr>
<td>1.Ridgelessness</td>
<td>6.8%</td>
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<tr>
<td>2.Dermoid Sinus</td>
<td>4.7%</td>
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<tr>
<td>3.Allergic Dermatitis*1</td>
<td>0.6%</td>
</tr>
<tr>
<td>4.Hypothyroidism*1</td>
<td>0.3%</td>
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<tr>
<td>5.Mast Cell Tumours*1</td>
<td>0.2%</td>
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<tr>
<td>6.Cardiac issues/Heart murmur*1</td>
<td>1.1%</td>
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Inherited conditions fall into two main categories—those which require intervention and those that are mainly cosmetic, which generally mean the affected animals are sold as pets and de-sexed.

1. Inherited conditions requiring veterinary attention

Dermoid sinus
This is the inherited condition most commonly thought of in Ridgebacks. It is related to the formation of the ridge and as a developmental issue of the tissue layers it has some similarity to spina bifida in humans. It is generally detected at birth (by most breeders and some veterinary surgeons). In Australia most affected animals are euthanised as a requirement of the breed clubs Code of Ethics. However, in the US by 2000 only 52% were euthanised. The dermoid sinus is felt as a fine thread from the skin surface going down toward the vertebrae—and keep in mind some animals may have multiple sinuses. It is generally felt along the midline between the occiput bone and the beginning of the ridge, or from the end of the ridge and down into the tail. However, sinuses have been reported elsewhere such as tracking through the epithelial surface and surfacing elsewhere as on the cheek area. Left unchecked, epithelial cells may slough off into the sinus and cause the area to abscess.

An early paper on Dermoid sinus (DS) suggested inheritance is dominant with "inconstant penetration." A study has recently been published which determined the genetic sequence responsible for the ridge and stated this is autosomal dominant. This is similar to the brown nose inheritance pattern—that is there are 2 "alleles" and one is dominant ("R"). So dogs with ridges are "RR" and "Rr", those without ridges are "rr". The researchers also demonstrated a link between dermoid sinus and the ridge. They noted that in selectively breeding dogs with ridges together and removing ridgeless dogs from the breeding population, the genetic sequence that codes for both the ridge and dermoid sinus is over represented! Their findings are not surprising as my experience is such that where there are more ridgeless pups born (Rr parents), there is less dermoid sinus. The following proposal by the researchers has not been well received (italics are mine!):
The most straightforward way of reducing the incidence of DS in Rhodesian Ridgeback dogs is to reduce the frequency of homozygotes for the Ridge mutation. This can be accomplished by allowing the use of ridgeless dogs for breeding. While we are aware that dogs with a DS are not usually kept for breeding, matings between homozygous (R/R) ridged dogs (presumably without DS) and ridgeless dogs (r/r) would give progeny all of which would be heterozygous ridged (R/r) and therefore show ridging, and the incidence of DS would be low. 

As this approach would result in a reduction in ridged dogs, breeders are hoping for a test for carriers of the dermoid sinus from this work rather than breeding with ridgeless ridgebacks!

**Hip Dysplasia**

Most Ridgebacks are now X-rayed for hip dysplasia prior to breeding. Selective breeding in the US has reduced the incidence from 11.8% dysplastic dogs born prior to 1980 to 3.4% for dogs born in 2003/2004. There are a number of grading schemes used, some providing more information on the characteristics of the hip. The US Orthopaedic Foundation for Animals (OFA) provides a 7 step grading scheme (Excellent, Good, Fair, Borderline and 3 levels of dysplasia. Two main schemes are used in Australia for grading of hips: the British Veterinary Association (BVA) scheme, and more recently the PennHip scheme, with the BVA scheme being the most common. There are a number of views and articles on the relative value of each scheme. In 2008 the BVA overall breed mean score for Ridgebacks is 119. The breed average in Australia is approx 6.7. This may be lower than the actual average as unlike the UK- there is no requirement for all X-rays taken to be submitted. The Pennhip scheme is also quantitative and use of this evaluation tool appears to be increasing.

Hip dysplasia is an uncommon but debilitating condition. In my experience some Ridgebacks are radiologically but not clinically dysplastic. However, the ideal is to choose breeding stock from those dogs with scores less than or around the breed mean score, while still keeping in mind the need to weigh up the quality of the whole animal when selecting breeding stock.

**Gastric Dilation and Bloat**

This is a life threatening condition, characteristic of large deep chested dogs and is sometimes seen in the Ridgeback. Early signs of this condition include attempts to vomit, restlessness, hunched up appearance and then a swelling abdomen. One of the most comprehensive studies on this condition was undertaken by the Purdue University in the US and they concluded risks increased with:

- Increasing age
- Increased thorax depth/width ratio (eg more depth, less width)
- Having a first degree relative with a history of Bloat
- A faster speed of eating
- Using a raised food bowl

Other studies have also implicated stomach tumours, a narrowed pylorus, eating non digestible foodstuff, stress and fear, eating only one meal a day, nasal mites (which contribute to air swallowing) and anything that delays gastric emptying. It is thought to have a genetic component, but the mode of inheritance is unknown.

**Elbow and Shoulder disease**

The term “Elbow disease” covers a number of conditions such as ununited anconeal process (UAP), fragmented medial coronoid process (FCP) and osteochondrosis (OCD). Breeding stock are increasingly Xrayed for elbow.
dyplasia, although the low incidence of problems in this area have made this a recent phenomenon. Willis\textsuperscript{15} states that while there is breed predisposition for OCD, genetic overtones are uncertain. Recent work suggests elbow disease is inherited (polygenic) and animals with any evidence of elbow dysplasia (score of 1 or above) are breeding risks. This is a change in thinking from earlier days where dogs with scores 2 and above were regarded as breeding risks\textsuperscript{14}. However, it has been also shown that scoring is not the same in all countries eg dogs have been scored 1 in one country but not another. Whether this is a difference in scoring systems or due to the subjective nature of grading is yet to be demonstrated. Scoring may vary between Xrays taken at different times, so there are differences related to positioning and quality of Xrays and the actual scoring. OFA figures from 2007 put elbow dysplasia in US Rhodesian Ridgebacks at 6.5\%\textsuperscript{16}.

Shoulder disease is more rarely reported, but OCD of the shoulder has been reported in the breed.

**Eye problems:**
Entropion (in-growing eyelids) is sometimes seen in the Ridgeback, and if early surgical correction is undertaken, rarely causes an on going problem. However, this is an inherited condition and affected animals should not be bred from. Cataracts, persistent papillary membranes, ectropion, corneal dystrophy and glaucoma have also been reported as inherited issues\textsuperscript{3}.

**Heart “murmurs”:**
Cardiac conditions may be diagnosed by vets following detection of “heart murmurs”, which are caused by a variety of different clinical conditions. Affected animals may be exercise intolerant, and in more severe cases may die suddenly. Specific diagnosis may need expensive diagnostic procedures to determine the cause and severity of the condition (Echocardiogram). The most common heart defects resulting in murmurs are patent ductus arteriosus (PDA-also seen in humans), cardiomyopathy and subaortic stenosis (SAS). These conditions are thought to be of polygenic nature- that is, inheritance is determined by a number of genes.

Other heart defects with an inherited basis seen in the breed include persistent right aortic arch and pulmonary stenosis\textsuperscript{3}. Some conditions can be detected by routine screening of pups prior to sale, but others such as SAS may not be found until later in life. It is not recommended to breed from animals with these conditions. It is also interesting to note that heart conditions are one of the few areas where numbers have increased between 1996 and 2006, and where the Australian numbers are higher than the US.

**Canine autoimmune diseases**
The immune system is responsible for recognizing “self and non-self” and defending the body against invading organisms and other foreign matter. When the immune system becomes weakened or disturbed in some way the body defenses can turn against the host and cause autoimmune disease. The most common manifestation of this in the Ridgeback is hypothyroidism and various skin conditions

- **Hypothyroidism (autoimmune thyroiditis)**
Hypothyroidism is thought to be inherited as a recessive gene in the Ridgeback, and the US figures demonstrate a high prevalence in that country\textsuperscript{2}. Comparative figures from other countries would be of interest, but
few countries undertake this test as a routine screening procedure as yet! It can be hard to diagnose but signs may include lethargy, poor coat quality (“rat tail”), failure to be attentive and weight gain.

Hypothyroidism is generally a result of autoimmune disease which destroys the thyroid gland. Diagnosis requires a blood sample. The disease can be treated, but treatment is life long.

- **Chronic skin conditions**
  Some Ridgebacks are prone to allergic dermatitis and some skin conditions have an inherited basis due to autoimmune disease. Allergic dermatitis can be distressing for dog and owners, and in some lines it may be necessary to select against this condition. Systemic diseases such as hypothyroidism and Cushing’s Syndrome (hyperadrenal corticisim) can also manifest as skin conditions.

In addition to the above a form of mange caused by the *Demodex canis* mite (demodicosis) may have an inherited component related to deficient immunity.

- **Degenerative myelopathy (DM)**
  DM is characterized by rear end weakness, usually in older dogs, and has many similarities to Multiple Sclerosis in humans. In this condition the immune system targets and reduces myelin, which surrounds the nerve fibres. With the recent development of test for the DM gene, the OFA is now recommending Ridgebacks be tested for DM. However, care must be taken with the use of the results as the dogs homozygous for the allele coding for this disease (“AA”) are at risk, and not all such dogs will develop DM. The OFA states: “We recommend that breeders take into consideration the DM test results as they plan their breeding programs; however, they should not over-emphasize the test results. Instead, the test result should be one factor among many in a balanced breeding program.” This appears to be good advice for most genetic disorders in the breed. At the time of writing 223 Ridgebacks had been tested with 9% at risk, 46% carriers (Aa) and 46% clear (aa).

- **Other autoimmune conditions**
  Other autoimmune diseases which have been reported in the breed include Addison’s disease (hypoadrenocorticism), Cushing’s Syndrome (hyperadrenocorticism), autoimmune haemolytic anaemia, immune mediated polyarthritis, immune mediated thrombocytopenia, systemic lupus erythematosus (SLE) and degenerative myelopathy. Autoimmune diseases are thought to have an inherited component, but some may not manifest without environmental triggers. Systemic Lupoid Onychodystrophy- a disease characterised by loss of toe nails has also been reported in the Ridgeback.

**Hernias**
These have been reported- generally as umbilical hernias. Depending on severity they may need surgical correction. It is suggested the condition has a polygenetic inheritance.

**“Wobblers syndrome” (Spondololithesis)**
This is sometimes seen in the rapidly growing young dog, and is seen in the Ridgeback. It is characterized by increasing “clumsiness” and loss of rear leg coordination and may result in paralysis. Recent veterinary progress has offered
new techniques for treatment of this condition. A genetic basis has been reported, and such animals should not be bred from\textsuperscript{19}.

**“Swimmers” (Pectus excavatum)**

This condition is rarely seen and characterised by new born pups which seem “flattened” and incapable of standing. It appears to be inherited and culling is recommended.\textsuperscript{15}

**Cryptorchidism**

In some cases one (or more rarely, both) testicle does not descend in the male pup. Breeders should check male pups carefully prior to sale as potential breeding animals, and ensure the desexing of dogs with one or no testicles. This is important as the retained testicle(s) may become malignant.

**Oncological Diseases (Cancer)**

As with most animals (including humans), Ridgebacks are prone to cancer. I have lost older Ridgebacks with Osteosarcoma and Malignant Melanoma. The US figures suggest Mast Cell Tumours are prevalent in the breed and they have been reported in Australian Ridgebacks. Mast Cell Tumours are thought to have a genetic component, with some suggestion Osteosarcoma also has an inherited basis.\textsuperscript{26,3}. Haemangiosarcoma has also been reported in the breed as having a polygenic mode of inheritance.\textsuperscript{3}

**Megaoesophagus**

This is a condition where the oesophagus is flaccid or weak and cannot push food, water and air into the stomach. In the Rhodesian Ridgeback this is the most prevalent gastrointestinal disorder, with an 81% mortality rate\textsuperscript{27}

**Diabetes**

Diabetes has been reported in the Ridgeback and is usually detected by animals having excessive thirst and urination. Diagnosis and treatment is similar to that in humans- that is measurement of blood glucose to determine required insulin doses.\textsuperscript{28} An inherited basis has been demonstrated in some breeds but mode of inheritance in Ridgebacks is currently unknown\textsuperscript{3}. Diabetes may also be seen in conjunction with other conditions such as Cushing’s disease.

The list of conditions presented above is not a complete list genetic disease that may be seen in the breed. A more complete listing can be found in Cynthia Roethel’s summary of the presentation by Dr George Padgett.\textsuperscript{3} The one thing breeders can be sure of, is the longer they breed, they more they can add to their own list of issues seen!

2. **Cosmetic inherited issues:**

This is where we leave the realm of science and enter a more subjective arena.

**The Ridge**

While this is the “escutcheon” of the breed, breeders have to contend with ridgeless pups, short ridges, animals born with offset (not parallel) crowns, or less or greater than two crowns. As stated previously the genetic sequence coding for the ridge has now been found.\textsuperscript{7} Many otherwise fine animals are sold as pets as either they are born without ridges or their ridges are not suitable for the conformation ring. Breeders, and indeed judges, will vary in what degree of
“offset” they will accept for the crowns, but should agree that animals with more (or less) than two crowns should not be bred from.

**Structure:**
As with any breed many structural faults appear to “run in families”. These may include poor fronts (lacking, or too wide), short muzzles, bad mouths, muzzles lacking in depth, “cheeky” heads, heads lacking in stop, high set ears, poor top lines, poor feet, steep croups or those lacking in slope resulting in high set tails etc etc. Also, as with other breeds, in many cases the worst of the structural defects are those which result in poor movement, with lesser defects more cosmetic in nature. Kinked tails, short and overlong tails are sometimes seen, and little has been done to look at the inheritance of these characteristics- although it is suggested kinked tails are inherited.

In my experience dental problems are associated with head structure eg lines with less depth of muzzle may see more overshot jaws, those with shorter and deeper muzzles are more likely to have issues with undershot jaws.

**Coat Colour:**
The breed standard calls for a light wheaten to red wheaten coat, with breeders sometimes debating what is too light or too dark. The standard of 1922 also allowed for brindles, sables “or mixed with white”, and excess white and black is sometimes still seen in the show ring. Most breeders tend to judge these animals on their merits and an otherwise outstanding specimen with a long white sock or excess black will be bred with by some breeders, but perhaps not by others. Blue/Greys (either a Weimeraner type grey, or a blue/grey overlay on a red coat) and Black and Tan Ridgebacks are rarely seen in litters, and should not make the show ring or be bred from! The genetic recessive “brown nose” with a flesh coloured nose and light eye is quite acceptable in the breed and should be judged on conformation like any other Ridgeback. Given the relatively recent development of the breed and the mixed source material, it is not surprising non standard colours are occasionally seen. Sandra Fikes has sourced and placed some photos of unusual coloured Ridgebacks on the internet.

**Additional whorls in the coat:**
In my experience these are not unusual and may be seen on the hindquarters, each side of the tail or on the neck below the ears. In some litters pups may also be born with whorls or crowns on the occiput area. They are also more rarely seen on the shoulders. Additional whorls on the top of the head or shoulders are usually penalised by breeders, whereas those on the hindquarter or sides of the neck are less likely to be seen as unacceptable.

3. **Breeders responsibilities in the age of genetic testing**

The breeder has a great responsibility to maintain the health of the breed. There is a range of tools to assist with choice of mates for our breeding animals. Selection of desirable phenotypic characteristics has always been a starting point eg is the Ridgeback you are considering sound, and does it complement your dog/bitch? Will the resulting pups be likely to have good breed type? Another tool now available is calculation of the relatedness of the animals eg is the Coefficient of Inbreeding within a range acceptable to the breeder? These questions are more about genetics than health, although there is some evidence to suggest inbreeding leads to some problems such as smaller litters and decreased life span.
The breeder then has a responsibility to breed in a manner that will reduce the possibility of genetic disease appearing in their litters. Given that Dr Padgett’s analysis showed that each animal carries the genes for around 6.6 defects this is a challenge.3 Breeders are currently informing themselves of some issues in their breeding stock by the use of phenotypic screening (eg the results of tests such as those for hip and elbow dysplasia, hypothyroidism etc) These demonstrate whether animals are affected with particular conditions. Recent advances also allow for genotypic testing with some tests currently available and more under development.

Given the increasing development and availability of additional genetic tests there is a danger that animals known to be carriers may all be excluded from the gene pool. Using Padgett’s “guesstimation” of carriers 35% of Ridgebacks will be carriers of hypothyroidism, 34% carriers of dermoid sinus, 31% carriers of mast cell tumours, 25% carriers for hip dysplasia etc. It does not take long to realise that if all carriers were to be excluded from breeding the breed gene pool may become dangerous small with a possible loss of desirable attributes (also known as “throwing the baby away with the bath water“). For example, outcomes of the genetic test for degenerative myelopathy have been discussed by breeders recently. As an alternative to not breeding from “at risk” carriers, breeders can minimise the possibility of this condition by breeding known carriers to animals that have tested clear of the condition. This would give the opportunity for dogs that have other required attributes to selectively contribute to the gene pool.

However, generally breeders do not know the genotype- only whether or not an animal is affected (phenotype). In most cases of inherited conditions requiring veterinary intervention or severely affecting the health of the animal, the response of the breeder to finding an inherited condition is clear cut. In general, do not breed from the affected animal and risk transmitting the condition to future generations. In the current age of litigation breeders should also be aware that knowingly breeding from faulty stock may result in puppy buyers successfully suing the breeder.

In the case of the more cosmetic issues the decision to breed from an animal may not be so clear cut eg should an excellent specimen of the breed with a slight tail kink, too much black or white, or having a crown on the head be removed from the breeding pool? I’m not proposing to answer this question, but only to remind breeders that they are the custodians of their breed, and must breed with care and knowledge of the genetic make up of their animals.

The question of how much screening should be done- and what is to be done with the results of the screening- is yet to be resolved and varies between countries. The following is taken from the Code of Ethics for members of the RR Club of the USA:

“Only dogs screened and certified clear of hip and elbow dysplasia by the Orthopedic Foundation for Animals, Pennhip or comparable foreign registry shall be bred. Other recommended genetic testing includes a complete thyroid panel* from an OFA-approved laboratory or canine endocrinologist, cardiac certification (OFA or cardiologist evaluation) CERF* (Canine Eye Registration Foundation) and BAER (Brainstem Auditory Evoked Response or hearing test). Breeders shall endeavor to stay current in their knowledge of all known inheritable diseases present in the breed and demonstrate reasonable care in eliminating those diseases (*indicates annual re-test requirement)“.

8 11/05/10
In many other countries breeders are required by breed clubs to screen only for hip and elbow dysplasia. If not in a breed club, there may be no breeding requirements. The approach of some breeders in these countries is to perform additional targeted testing should particular conditions be seen, rather than blanket testing all breeding stock. Furthermore in many countries there are few specialists able to perform some of the tests recommended above, and certainly there are not certification schemes.

However, with the current focus in the UK in particular on the health of pure breed dogs, perhaps it is time to consider the level of testing undertaken to maintain the health of our breed, and to better inform our breeding selections. It is certainly time to be honest with each other to enable breeding with all available information for the betterment of our breed.

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**Side Bar**

**Coefficient of Inbreeding**

The Coefficient of Inbreeding (COI) is defined as “The proportion of all variable  

gene pairs that are likely to be homozzygous due to inheritance from ancestors  

common to the sire and dam.” As such it is a measure of the relatedness of two  

dogs and is calculated by examination of pedigrees. Some breeders now use  

commercial pedigree programmes for pedigree analysis and generation. Many of  

these can perform the COI calculation and it is now appearing on some pedigrees.  

It is recommended the calculation be performed on 10 generation pedigrees for  

better accuracy.
To provide an understanding of the numbers that follow, breeding of uncle to niece gives a COI of 12.5%, first cousins 6.25% and parent to offspring or brother/sister is a 25% COI (note these figures assume uncle, niece etc to be unrelated). Dogs with a high COI (higher numbers) are the products of inbreeding or line breeding. The COI of dogs in the US was calculated by Dr Jerome Bell to be 15.2%. The figure for Australia is 12.5% and that of Sweden is 1.9%.

There are very few actual recommendations for desirable COI figures from Breed clubs. The only one found for Ridgebacks is from the Swedish Ridgeback Club, which recommends a COI of 6.25% or less. Also, Willis (Practical Genetics for Dog Breeders, p206) observes the highest inbreeding that would be legally possible in most human societies would be that of first cousin matings (6.25%).

Breeders are encouraged to use this tool in addition to the other information they have available to them when planning a mating.