Breed Predispositions to Disease in Dogs and Cats
Alex Gough:
To loved ones, friends and colleagues, for their enormous support over the last three years. Above all, to my daughter Abigail for lighting up my life.

Alison Thomas:
To Richard, Tom and Harry for their patience and for giving me the time to tackle this project.
Breed Predispositions to Disease in Dogs and Cats

SECOND EDITION

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A colour plate section falls between pages 158 and 159
Foreword

This new edition of *Breed Predispositions to Disease in Dogs and Cats* provides an update on the previous edition, with the aim of improving accuracy and usefulness to animal health professionals. Through presentation of lists, this book provides an easily accessed source of diagnostic possibilities that is particularly helpful when dealing with an unusual collection of clinical signs or with unfamiliar breeds. Diagnostic errors are far more likely to arise from a differential list that is too short than too long! The authors’ experiences in busy first opinion and referral practice have no doubt provided the incentive to organize the book appropriately for those who will have most wish to consult it.

This book serves an important purpose in spotlighting the risks of selective breeding. There is considerable evidence from studies in cheetahs (O’Brien et al. 1985) and wolves (Lindberg et al. 1995) that high levels of inbreeding increase the risk of disease. The creation of purebred pets imposes similar genetic restrictions to those that occur in wild species that have been reduced to tiny breeding numbers after natural and man-made disasters. Generally, registration with the Kennel Club of Great Britain requires that a dog be purebred for at least six generations. This degree of inbreeding is further exacerbated by the frequency of overuse of specific sires (Calboli et al. 2008), meaning that even breeds with thousands of individuals often have very restricted effective population size (often as low as ∼40). Such a population structure implies that a randomly arising deleterious genetic mutation in a breeding animal will spread rapidly through subsequent generations. Although not yet formally proven for a large proportion of the diseases of purebred animals, commonsense would suggest that the high prevalence of many of the diseases listed in this book is the inevitable consequence of inbreeding.

There are considerable practical difficulties in assembling a text of this nature given the current methods of recording diagnosis in veterinary medicine and the problem that data are not inevitably collected and recorded from ALL diseased animals. To minimize omissions, the authors have understandably accepted standards of evidence for an ‘increased risk’ that might not be appropriate in a systematic journal review article. Therefore, a wide range of references are cited, including non peer-reviewed sources. The authors have faced the problem that it is not straightforward to determine whether a breed is genuinely more ‘at risk’ of developing specific conditions, or simply that a specific condition ‘happens’ to have been reported in a specific breed. Many reports do not specify the reference population against which comparisons are made and, where they are specified, they vary considerably between reports; for instance versus the population of animals referred to a specific hospital or versus insurance or breed society data. All comparative populations carry implicit sources of bias and, furthermore, the most useful comparison might be against the incidence in mixed-breed animals, for which there is very little data at all. While this type of intrinsic bias is of little relevance to a practitioner wishing to determine if, say, polyneuropathy has been reported in Birman cats, it should be borne in mind when planning academic studies or to inform selection of a family pet.

The diseases to which specific breeds are predisposed can be divided into two main groups: those that have arisen accidentally or incidentally through the process of inbreeding to derive defined breeds and those that have deliberately been introduced through selection for extreme phenotypes as ‘desirable’ traits. The starting point to reduce the incidence and severity of all conditions is their recognition – for which this book is an invaluable aid – but subsequent interventions to promote their elimination would then differ considerably. For instance, brachycephalic obstructive airway syndrome or entropion arises through a deliberate strategy of breeding individuals that are unfit for a healthy life but this is not equivalent to, for instance, the increased risk that has arisen for Fanconi’s syndrome in Basenjis or soft tissue sarcomas in Flat Coat Retrievers.

It is to be hoped that this book, by highlighting breed-associated disorders, can provide an impetus towards more vociferous veterinary leadership in this important area of companion animal welfare.
There are two simple first steps that would aid progress in this respect: firstly, collaboration to collect hard evidence on the prevalence of disorders associated with inbreeding and, secondly, promotion of the considerable merits of mixed-breed animals as healthy pets.

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RCVS Recognised Specialist in Veterinary Neurology
Professor of Veterinary Clinical Studies, University of Cambridge, UK
Introduction

It is well known that most breeds of dogs and cats have diseases and disorders to which they are particularly prone. Breed predispositions in dogs have been put under the media spotlight in the UK recently, prompting the Dogs Trust and Kennel Club to set up a review of the registration, breeding and showing of dogs in the UK. The aim of the review, chaired by Professor Bateson of Cambridge University, is to provide practical recommendations that will benefit pedigree and non-pedigree dogs. At the time of writing, the review panel is collecting data from interested parties and experts on dog breeding and showing.

Breed predispositions are often listed under specific disease conditions in the published literature and textbooks. However, it is hard to find a source of information that lists these conditions by breed. The first edition of this book aimed to correct this deficiency, and this second edition builds on, as well as updates, the information provided in the first edition. It is intended to be of use to prospective or current pet owners and breeders who wish to be fully informed of the diseases to which their chosen or favourite breeds may be prone. Its main utility, though, will be to veterinary surgeons, and it is at this group of people that the book is aimed. Much of the language used is of necessity technical, and non-veterinary readers wishing to use this book are urged to contact their vets if they have any queries regarding the information. Breeders wishing to institute programmes to select against inherited diseases should also speak to their vet or a suitably qualified professional.

For veterinary surgeons, this book should be useful in several ways. Most importantly, it should assist in producing and prioritizing a list of differential diagnoses after a history has been taken and physical examination made, and allow the selection of the most appropriate tests to achieve a diagnosis. It will also be important in advising clients wishing to buy new pets, and owners of existing pets can be warned of preventative measures to reduce the risk of diseases, or to monitor closely for the onset of clinical signs to allow early treatment to be instituted.

For the purposes of this book, a breed predisposition has been defined as an increased risk for a condition in a breed. This does not mean that breed predispositions are necessarily inherited diseases (although many are). For example, the uses to which a breed is put can influence the conditions to which it is prone. A Foxhound may be predisposed to fox bites, but this does not mean that it inherits them!

For this revised second edition, we have been much stricter as to what conditions should be included as breed predispositions. We have attempted to include information, where available, on modes of inheritance, risk of developing the disease compared to the general population (relative risk or odds ratio), how common a condition is in the population (although this is often a subjective opinion, and is subject to geographical variation) and any sex and age predispositions, as well as show where only a small number of cases have been reported. Importantly in response to feedback on the first edition, we have referenced every condition in every breed, and excluded conditions we have not been able to reference. Most references are in the form of papers in peer-reviewed journals, but some references have been obtained from conference proceedings, and occasionally from well-regarded textbooks.

The data in this book have been categorized by body systems affected as far as possible, although some diseases span more than one category. The category ‘physiological conditions’ is intended to describe those abnormalities specific to a breed which are non-pathological, or are accepted as part of a breed standard.

Caution should be exercised when using the information in this book. Many diseases which do not have breed predispositions are not in the text, and will also need to be included in differential diagnosis lists. Also, some predispositions are weak, with only a slightly increased risk in the breed. The existence of a breed predisposition should not lead the clinician to exclude other disease possibilities, nor should the absence of a predisposition in a breed be taken to mean that the breed cannot contract the disease.
It may be noted that some breeds are absent from this book – this is because the authors have not found any data on any predispositions in these breeds.

A final limitation of the data presented in this book is that of geographical variation. Some populations may exhibit predispositions to diseases that others do not. Where possible this has been indicated in the text.

We hope you find this updated and fully referenced version of this book a useful addition to your library.
Basic Genetics

All mammalian life is based on the genetic code stored within the nucleus of a cell. This genetic code is stored in a long molecule called deoxyribonucleic acid (DNA). DNA is composed of four units, called bases, and these bases attract each other—guanine to cytosine and adenine to thymine. When attached together, they form the famous double helix. The order in which these bases (or base pairs, since they always match together) occur along the molecule provides the code for the synthesis of proteins. Proteins are then responsible for most of the functions of the body, from the structure of tissues, to the biological catalysts called enzymes, to the hormones which regulate the body’s metabolic processes.

A length of DNA which codes for a particular protein is called a gene. Long strings of genes, interspersed with areas of DNA which do not code for proteins, make up chromosomes. Each nucleus of a mammalian cell contains a set number of chromosomes, except the sex cells (gametes) – sperm and ova. For dogs this number is 78 and for cats it is 38.

When a somatic (body) cell divides, the chromosomes shorten and thicken within the nucleus, so they become visible under a microscope. They then replicate, and one copy of each chromosome separates into a new nucleus before the cell splits. This process is called mitosis. However, in the production of the gametes (the process of meiosis), the chromosomes line themselves up in the middle of the cell with a companion. This companion is always the same, and two chromosomes that associate together are called homologous pairs. These homologous pairs separate, so the gametes have half the number of chromosomes as normal cells. This means that when a sperm and ova combine at fertilization, the newly formed cell (the zygote) has the correct number of chromosomes.

Homologous pairs code for related genes, but are not identical. The two genes, one on each chromosome, interact in different ways. Sometimes one gene is dominant to the other, the less dominant gene being termed recessive, and the expression of the gene, that is, the protein that is produced, will be determined by the dominant gene. In other cases both genes will play a role in the production of the protein, a situation called co-dominance.

The exception to the homologous pairs are two chromosomes called the sex chromosomes (all the other chromosomes are called the autosomes). These chromosomes determine the sex of an animal. A female’s cells are composed of two X chromosomes, a male’s of an X and a Y. At meiosis, the ova inherit a single X chromosome from the mother, whereas the sperm inherit either an X or a Y from the father. This has significance for the inheritance of conditions carried on the X chromosome, and means that some inherited diseases can be more prevalent in one sex than another.

Although any one animal will carry only up to two versions of a gene, many more can exist within a population because of mutation and natural selection. These different versions of the gene are called alleles.

In conditions and characteristics that are inherited in a simple way, that is, the conditions are autosomal dominant or recessive, then a system of genetics devised by the monk Mendel (hence Mendelian genetics) can be used to predict the likely offspring of two parents, if the parents’ genetic make-up is known. For example, the gene that codes for Labrador coat colours is dominant for black and recessive for brown. If a Labrador has two alleles for black colour (call the allele B) it is described as being BB and hence the coat will be black. If it has one allele for black and one for brown (call the allele b), it will be described as Bb but the coat colour will still be black since this colour is dominant. However, if the dog possesses two alleles for brown (bb), it will be brown. The genetic make-up is called the genotype, whereas the physical expression of the genes is called the phenotype.

The situation is slightly more complex when looking at matings, and a matrix can be used to aid prediction of offspring types. Take the example of a BB black male crossed with a bb brown female. The BB male will produce sperm each carrying a single B gene, and the female will produce ova each carrying a single b gene. These are then recombined at random to produce offspring. The matrix would therefore look like this:
Male
B  B

Female  b  Bb  Bb
   b  Bb  Bb

This means that all the offspring would be Bb. They all carry the b gene for brown coat, but because this gene is recessive, the coat colour is black. An animal with two identical alleles (e.g. BB) is called a **homozygote** while an animal with two different alleles (e.g. Bb) is called a **heterozygote**. If a black Bb female was then crossed with a black Bb male a different pattern would emerge:

Male
B  b

Female  B  BB  Bb
   b  Bb  bb

On average three of the offspring would be black, one a homozygote BB and two heterozygotes Bb. One would be a homozygote for brown coat colour, and, since this gene is not now being suppressed by the dominant gene, the brown coat colour phenotype is expressed.

In fact, since the fertilization process is random, a litter of four pups may not be born in the exact 1:2:1 ratio, but if this was repeated enough times the proportions of pups of the various colours would be close to this.

Generally, the alleles separate randomly from each other, so just because one condition is expressed in an offspring, it does not mean that another shown by its parent will be. However, some alleles that are closely positioned on a chromosome have a tendency to be passed on together. Thus, two traits controlled by different genes may often be found together in the same individual, and the presence of one of these traits may act as a marker for the other. This process is known as **linkage**.

When one allele is not dominant over another co-dominance exists. For example, certain flowers that have alleles for red flowers (R) and white flowers (W) will be coloured red if homozygous for red (RR), white if homozygous for white (WW) but pink if heterozygous (RW). Some genes, even if dominant, do not always produce a physical effect in the host. For example, the condition polycystic kidney disease in cats is inherited as an autosomal dominant trait, but not all cats with the genes have cysts in the kidneys. This situation is called **incomplete penetrance**. Penetrance is the proportion of individuals with a particular genotype that demonstrate the characteristics normally expected with that genotype.

Some characteristics are carried on the X chromosome, and this can lead to the phenomenon of sex linkage. For example, there is a condition called X-linked muscular dystrophy, to which Golden Retrievers are predisposed. The allele for muscular dystrophy (call it M) is carried on the X chromosome, as is the allele for a normal dog not suffering from the condition (call it N). M is recessive to N. Therefore a female carrying a single affected X chromosome (genetic make-up X^M^X^N^) would not show the effects of the disease. If this female was mated with a normal male, (X^N^Y) then the matrix for their offspring would be as follows:

Male

X^N  Y

Female  X^M  X^M^X^N  X^M^Y
 X^N  X^N^X^N  X^N^Y

All of the females born to this cross will be clinically unaffected by the disease but 50% of the females will be carriers of the disease. These will not show the disease since they have a normal gene on the other X chromosome which suppresses the abnormal, recessive gene. However, the males only possess a single X chromosome, so the 50% of males born X^M^Y will show the disease (since they do not possess
another X chromosome with a normal gene). The 50% of males born X<sup>N</sup>Y will not show the disease and will not carry it.

Because of this process, sex-linked diseases usually affect only males, and males cannot normally be asymptomatic carriers. Females are often carriers but the only way they can express the disease is if their mother was a carrier and their father was affected. This situation is rare in nature, especially for uncommon genes, but can occur in domestic animals due to inbreeding.

Some disease inheritances are more complex still, because more than one gene may determine the expression of a disease, or the interaction of genes and environment can determine the outcome in an individual. An example of this is hip dysplasia in dogs. More than one gene is considered to be responsible, but the dog’s nutrition, exercise and other factors can also influence the severity of the disease.

Finally, some diseases are not inherited through the DNA of the nucleus at all, but through the DNA present within the mitochondria (which are intracellular organelles responsible for energy production). Mitochondria are entirely inherited from the mother, hence characteristics and diseases caused by mitochondrial DNA can only be passed down from the mother. Although conditions caused by mitochondrial DNA are rare, some canine myopathies are thought to be inherited this way.

In summary, an autosomal dominant trait is transmitted from generation to generation without skipping. Each affected offspring has at least one affected parent, unless the disorder has arisen because of mutation. If the disorder is lethal, then it will be very rare. An autosomal recessive disorder may skip generations. If the two parents are affected then all the offspring are affected. With an X-linked dominant condition, affected males mated to normal females transmit the gene to their daughters, who are all affected, but not their sons. Affected females then pass the condition on to approximately half of their sons and half of their daughters. In the population as a whole, the incidence in females tends to be twice that of males. With an X-linked recessive disorder, the condition may skip generations. The incidence is more common in males. Affected males do not transmit the disease when mated to a normal female, but all female offspring will be carriers. Females showing the disease who are mated with normal males will pass the condition on to all their sons, and all their daughters will be carriers.

**Clinical genetics**

Genetic diseases are probably more frequently encountered in domestic animals than in most wild populations. The process of domestication involves selecting animals for their most desirable traits from a human point of view. Initially, these traits would have been practical: speed in a horse, fertility and milk production in a cow, herding instincts in a sheepdog and so on. Over time, when animals came to be kept for their companionship and aesthetic appeal, the pressures of selection altered to produce breeds that were poorly adapted to survive in the wild, but fitted in well to the human environment, for example, achondroplasia in many dog breeds. As breeding practices were refined and the science of genetics was discovered, inbreeding was used to create breeds that bred true with respect to certain desired characteristics.

Unfortunately, inbreeding reduces the genetic variation within a breed, and tends to accentuate the presence of recessive genes which are often deleterious. Population bottlenecks occur due to overuse of a desirable individual such as a show champion (particularly males, which are capable of producing many more offspring than a female). Most of the recognized genetic diseases of the dog are inherited as an autosomal recessive. This may be because of inbreeding, but is also due to the difficulty in identifying and eliminating recessive traits in breeding programmes. The often repeated saying that crossbreed dogs are healthier than pedigrees may have some basis in truth because outbreeding tends to mask the effect of many recessive genes. However, crossbreed dogs can still be predisposed to the diseases of their parent breeds.

It should be noted that inbreeding of itself does not cause genetic disease, and some degree of inbreeding is necessary for the concentration of desirable genes. In fact, some inbred strains of mice and rats are entirely homozygous and yet are healthy. Inbreeding promotes homozygosity, and thus deleterious recessive genes are exposed. However, by exposing these genes it is possible to eliminate them by further selective breeding.
Data are currently sparse regarding the prevalence of disease caused by new mutations, but studies of some diseases suggest this is very rare. In those limited cases studied, the mutation seems to be uniform within a breed. This suggests that a ‘founder effect’ applies, that is, a single initial mutation was propagated throughout the breed. In some cases, closely related breeds may have the same mutation causing a disease, for example, phosphofructokinase deficiency in English Springers and American Cocker Spaniels, suggesting that a common ancestor was responsible for the original mutation. Some diseases, however, have more than one mutation in the same gene (*allelic heterogeneity*).

When determining whether or not a disease is hereditary, certain typical characteristics increase suspicion of a genetic predisposition. Often the first thing to suggest that a disease is inherited is that the disorder occurs with a higher frequency in a group of related animals than in the general population. This can help distinguish an inherited disease from a breed predisposition. For example, Saint Bernards are predisposed to osteosarcomas, but it was possible this was merely a reflection of their large size: the faster growth rate leading to more mistakes being made in DNA replication, leading to cancer. However, analysis of pedigrees shows that there is a familial clustering pattern to cases of the disease, which suggests a specific gene or group of genes being responsible. Second, a hereditary defect often involves the same anatomic site in a group of related animals. This is often seen in congenital heart disease in dogs. Third, the disease is often seen to increase in frequency with inbreeding. Fourth, hereditary diseases often have an early onset, and those that do not often have a consistent age of onset.

Genetic diseases usually affect a few individuals within a litter, as opposed to intoxications and infectious diseases which frequently affect them all. Some genetic diseases will cause abortion or resorption, and these are often never diagnosed. Similarly, some genetic disorders will cause a failure to thrive, the ‘fading kitten (or puppy) syndrome’, and again it can be hard to determine the cause in these cases.

There is an extremely wide range of hereditary diseases, from the relatively benign to the invariably fatal. Diagnosis of a hereditary disease is usually based on history, clinical signs, history of disease in related individuals, test matings and specific tests for diseases.

Test matings are often suggested in order to identify autosomal recessives but this does have problems. With late onset defects, the results of the mating will be known too late to be useful in selecting which individuals to use for breeding. Test matings can be more useful for early onset diseases, but the ethics of keeping a known affected animal purely for test purposes, and what to do with affected offspring, can be problematic. Furthermore, the results of test matings may be unreliable. For example, a mating of a suspected carrier (N?) to a known carrier (Nn) which produced six normal puppies would only give an 82.2% certainty that the N? was not a carrier (NN). However, a single abnormal pup would confirm carrier status.

The results of random matings, if performed often enough and with respect to a sufficiently prevalent gene, can provide useful information without the need to maintain a carrier or affected animal, and with less likelihood of breeding unwanted affected individuals. It would require central banks of information and more openness from breeders and breed organizations for this approach to work, however. Recent activity in the Kennel Club means that moves towards this situation may be imminent.

Specific tests for diseases include ultrasonography and histopathology for polycystic kidney disease, or von Willebrand factor assay for von Willebrand’s disease. Some laboratories will test samples using enzyme and immunological assays to detect disorders, and the results may indicate whether an individual is a homozygote or heterozygote. An example of this is testing for haemophilia B. A defect in an affected protein’s size, function or amount allows the identification of carriers of a disease in some cases, although there may be an overlap with normal values. Also, compensatory rises in other proteins, such as a related isoenzyme to pyruvate kinase in pyruvate kinase deficiency, may reduce the accuracy of this sort of test.

In some of the inherited diseases, the molecular defects that cause them have been identified. Those identified on the X chromosome include haemophilia B, severe combined X-linked immunodeficiency and hereditary nephropathy. Those autosomal recessive traits for which the mutation has been identified include copper toxicosis in Bedlington Terriers, progressive retinal atrophy in Irish Setters, von Willebrand’s disease in Scottish Terriers and pyruvate kinase deficiency in Basenjis.
Specific DNA tests currently available to identify genetic diseases include linkage-based tests that look for a marker gene, which is physically in close proximity to the gene of interest. An example of a currently available test using this method is for copper toxicosis in Bedlington Terriers. Mutation-based tests look for a specific mutation causing a disease, but these tests must be used with caution, since there may be more than one type of mutation responsible for a disease, particularly between breeds.

DNA testing shows great promise for the identification and elimination of genetic diseases in dogs and cats. The inherited disorders can be identified before an animal is bred, and affected animals can either be removed from the breeding pool, or, in the case of recessive traits, bred only to normal individuals, in order to preserve desirable characteristics. This allows the genetic diversity of breeds to be retained while inherited disorders are eliminated.

The limitations of DNA testing, such as limited availability of tests, and its utility largely restricted to single gene disorders, mean that there is still a vital role for screening programmes to eliminate inherited disorders. Screening programmes currently in operation in the UK include the British Veterinary Association/Kennel Club programmes for hip and elbow dysplasia and eye diseases, and the Feline Advisory Bureau scheme for polycystic kidney disease in cats.
PART I

DOGS
**ABERDEEN TERRIER**

**Reproductive conditions**
Dystocia
- Breed predisposition to dystocia due to primary uterine inertia
  (Johnson et al. 2001)

**AFFENPINSCHER**

**Dermatological conditions**
Canine follicular dysplasia/seasonal flank alopecia
- Presumed genetic basis
- Alopecia starts at 2–4 years of age and is restricted to the flank in this breed
- May occur in autumn or spring
  (Scott, Miller & Griffin 2001d)

**AFGHAN HOUND**

**Dermatological conditions**
Generalized demodicosis (see figure 1)
- Afghans are in the ten breeds at highest statistical risk of this disease in the Cornell, USA, population
- Common condition

**Endocrine diseases**
Hypothyroidism
- Breed at increased risk
- May occur younger in breeds at risk (2–3 years)
- Neutered males and females at increased risk
  (Panciera 1994, Greco 2002)

Nasal depigmentation
- Also known as Dudley nose
- Cause unknown
  (Scott, Miller & Griffin 2001a)

Testosterone-responsive dermatosis of male animals
- Rare
- Unknown cause
- Seen in castrated males
  (Hubert & Olivry 1990)
Musculoskeletal conditions
Panosteitis
- Also known as enostosis, eosinophilic panosteitis
- Common
- Young males predisposed
- Odds ratio 1.9 (compared to mixed breeds)
  (LaFond, Breur & Austin 2002)

Neoplastic conditions
Perianal (hepatoid) gland adenomas
- Breed at risk in case series
- Average age was 10.5 years
- Entire males were predisposed
  (Goldschmidt & Mcmanus 2000)

Neurological conditions
Afghan myelopathy
- Autosomal recessive inheritance suggested
- Age of clinical onset: 6–9 months
- Rare condition
  (Averill & Bronson 1977)

Ocular conditions
Cataract
- Autosomal recessive inheritance suspected
- Age of onset: 4–24 months
- Localization: initially equatorial, later anterior and posterior cortical. Rapid progression possible, leading to visual impairment at 2–4 years
  (Rubin 1989, Gelatt & Mackay 2005, ACVO Genetics Committee 2007)

Corneal dystrophy
- Inheritance suspected
  (ACVO Genetics Committee 2007)

Corneal oedema (due to infection or vaccination with canine adenovirus type 1)
- Increased susceptibility (less commonly seen with the development of canine adenovirus type 2 vaccines)
  (Curtis & Barnett 1983)

Medial canthal pocket syndrome
- Breed predisposition due to head shape
  (Rubin 1989)

Respiratory conditions
Chylothorax
- Usually idiopathic
- Uncommon condition
- Afghan hounds comprised 37.5% of dogs with idiopathic chylothorax in one study
- No sex predisposition noted
  (Fossum, Birchard & Jacobs 1986)
Laryngeal paralysis
- Idiopathic
- Common condition
- May be inherited by an autosomal dominant mode
  (Burbidge 1995)

Lung-lobe torsion
- Rare
- May be associated with chylothorax in this breed
  (Johnson & Feeney 1984)

**AIREDALE TERRIER**

**Cardiovascular conditions**
Dilated cardiomyopathy
- Increased prevalence with age
- Approximately twice as common in males as in females
- Thought to be familial or genetic
  (Tidholm & Jonsson 1997)

**Dermatological conditions**
Canine follicular dysplasia/seasonal flank alopecia
- A marked predilection in this breed implies a genetic basis for this group of diseases
- Hair loss begins at 2–4 years of age and occurs mainly on the flank
  (Miller & Dunstan 1993)

Generalized demodicosis
- Airedales are in the ten breeds at highest statistical risk of this disease in the Cornell, USA, population
- Common condition
- Younger dogs predisposed
- No sex predilection
- Susceptibility to generalized demodicosis may be inherited
- Incidence within breeds may vary depending on geographical location
  (Scott Miller & Griffin 2001b)

Grass awn migration
- Common in the summer months
- Predisposed due to behaviour
  (Brennan & Ihrke 1983)

Skin tumours
- See under Neoplastic conditions

**Vascular naevas**
- More common in older dogs
- Occur most commonly on the scrotum
  (Scott Miller & Griffin 2001c)

**Endocrine conditions**
Hypothyroidism
- Breed at increased risk
- May occur younger in breeds at risk (2–3 years)
- Neutered males and females at increased risk
  (Milne & Hayes 1981)

Haematological/immunological conditions
Haemophilia B
- Severe Factor IX deficiency in this breed
- Familial in this breed
- Genetic test available (HealthGene) – see Appendix
  (Brooks 1999)

**Musculoskeletal conditions**
Hip dysplasia
- Common condition
- Neutered male dogs predisposed
- Odds ratio in the breed 1.82 in one study, 3.9 (compared to mixed breeds) in another study
  (LaFond, Breur & Austin 2002, Witsberger et al. 2008)

Spondylosis deformans
- Usually clinically insignificant
- Common
- May be associated with type II intervertebral disc disease
  (Levine et al. 2006)

**Neoplastic conditions**
Cutaneous haemangioma
- Breed at risk in case series
- Mean age: 8.7 years
  (Goldschmidt & Shofer 1992, Goldschmidt & Mcmanus 2000)

Cutaneous plasmacytoma
- Breed at risk
- Mean age: 9.2 years
Common sites: ear, lip and digit  
(Goldschmidt & Shofer 1992)

Melanoma – cutaneous
- Breed at increased risk in case series
- Mean age: 8.9 years  
(Goldschmidt & Shofer 1992, Morris & Dobson 2001)

Nasal cavity tumours
- Breed at increased risk
- Median age: 9 years
- Males over-represented in most studies  
(McEntree 2001)

Pilomatrixoma
- Breed at risk in case series
- Mean age: 6.6 years
- Uncommon (1% of all cutaneous neoplasms in the dog)  

Ocular conditions
Corneal dystrophy
- Sex-linked recessive inheritance has been suggested
- Lipid stromal dystrophy
- Age of onset: 9–11 months
- Progressive with vision impairment at 3–4 years  

Entropion
- Polygenic inheritance suspected  
(Rubin 1989)

Reproductive conditions
Cystic endometrial hyperplasia–pyometra complex (pyometra)
- Breed at risk in case series
- Common disease of older entire bitches (mean age: 7.25 years)
- Most cases present within 12 weeks of oestrus  
(Chastain, Panciera & Waters 1999, Smith 2006)

AKITA
(See Japanese Akita)

ALASKAN KLEE KAI

Haematological/immunological conditions
Factor VII deficiency
- 6/18 client-owned dogs had this deficiency in one study
- Inherited condition
- Genetic test available (PennGen; VetGen) – see Appendix  
(Kaae, Callan & Brooks 2007)

ALASKAN MALAMUTE

Dermatological conditions
Alopecia X
- Poorly understood disorder
- Occurs at 2–7 years of age  
(Leone et al. 2005)

Follicular dysplasia
- May affect multiple dogs in a litter
- Clipped areas tend not to regrow
- Signs may not be seen until 3–4 years of age in this breed  
(Scott, Miller & Griffin 2001d)

Generalized demodicosis
- Malamutes are in the ten breeds at highest statistical risk of this disease in the Cornell, USA, population
- Common condition
- Younger dogs predisposed
- No sex predilection
- Susceptibility to generalized demodicosis may be inherited
- Incidence within breeds may vary depending on geographical location  
(Scott, Miller & Griffin 2001b)

Skin tumours
- See under Neoplastic conditions

Zinc-responsive dermatosis
- Syndrome I affects malamutes
- Skin lesions develop despite adequate levels of zinc in the diet
- Malamutes have a genetic defect causing zinc malabsorption
  
  \[(Cole \ 2002)\]

**Haematological/immunological conditions**

Factor VII deficiency
- Inherited as an autosomal dominant trait
  
  \[(Littlewood \ 2000)\]

Haemophilia B
Factor IX deficiency
- Also known as Christmas disease
- Inherited as a sex-linked trait
- Less common than haemophilia A
- Can be severe in this breed
  
  \[(Dodds \ 2005)\]

Stomatocytosis
- Uncommon condition
- Seen in a few cases of malamutes with chondrodysplasia
  
  \[(Giger \ 2003)\]

**Musculoskeletal conditions**

Alaskan malamute chondrodysplasia
- Causes disproportionate dwarfism
- Haemolytic anaemia usually present
- May be zinc responsive
- Autosomal recessive inheritance with complete penetrance and variable expression
  
  \[(Bingel, Sande & Wight \ 1985)\]

Hip dysplasia
- Common condition
- Neutered male dogs predisposed
- Odds ratio in the breed 2.33
  
  \[(Witsberger et al. \ 2008)\]

**Neoplastic conditions**

Anal sac adenocarcinoma
- Breed at increased risk in case series
- Mean age: 10.2–10.8 years
- Some surveys suggest a predisposition for females
  
  \[(Goldschmidt & Shofer \ 1992, Bennett et al. \ 2002)\]

Perianal (hepatoid) gland carcinomas
- Breed at risk in case series
- Entire males were predisposed
  
  \[(Goldschmidt & Mcmanus \ 2000)\]

Sebaceous gland tumours
- Breed at risk of sebaceous adenoma and epithelioma in case series
- Mean age: 10.9 years (adenoma) and 10.7 years (epithelioma)
- Common site: head and eyelids
  
  \[(Scott & Anderson \ 1990, Goldschmidt & Shofer \ 1992, Goldschmidt & Mcmanus \ 2000)\]

Sweat gland tumour
- Breed at risk of apocrine adenoma in case series
- Mean age: 9.5 years
  
  \[(Goldschmidt & Mcmanus \ 2000)\]

**Neurological conditions**

Idiopathic epilepsy
- Age of onset: 6 months to 6 years
- Common condition
  
  \[(Bagley \ 2005)\]

Idiopathic polyneuropathy in Alaskan malamutes
- Uncommon
- Affects mature young adults
  
  \[(Braund et al. \ 1997)\]

**Ocular conditions**

Cataract
- Inheritance suspected
- Age of onset: 1 year
- Localization: posterior subcapsular, slowly progressive; complete blindness is rare
- Schedule A of the BVA/KC/ISDS Eye Scheme
  
  \[(Rubin \ 1989, ACVO Genetics Committee \ 2007)\]

Cone degeneration (hemeralopia or day blindness)
- Autosomal recessive inheritance
- Clinical onset (day blindness) at 4–8 weeks
- Uncommon condition
  
  \[(Rubin \ 1989, Sidjanin et al. \ 2002, ACVO Genetics Committee \ 2007, Gelatt \ 2007)\]

Corneal dystrophy
- Inheritance suspected
- Lipid dystrophy
- Age of onset: 2 years
  
  \[(Rubin \ 1989, ACVO Genetics Committee \ 2007)\]
Glaucoma (primary)
- Inheritance suspected
- Age of onset: >6 years
  (Rubin 1989, ACVO Genetics Committee 2007, Gelatt 2007)

Ocular conditions
Neuronal ceroid lipofuscinosis
- Autosomal recessive inheritance suspected
- Rare condition
- Genetic test available (VetGen; Orthopedic Foundation for Animals) – see Appendix
  (Awano et al. 2006)

Neurological conditions
Neuronal ceroid lipofuscinosis
- See under Ocular conditions

Gastrointestinal conditions
Parvovirus enteritis
- See under Infectious conditions

Infectious conditions
Babesiosis (Babesia gibsoni)
- High incidence reported in this breed in USA, Australia and Japan (unknown if this is due to a true breed susceptibility or increased risk of exposure via ticks or possibly bites)
- Often subclinical
- Tickborne disease but possibly also transmitted by fighting

Parvovirus enteritis
- Breed at increased risk in cases series
- Age 6 weeks to 6 months at higher risk
  (Houston, Ribble & Head 1996, McCaw & Hoskins 2006)

Ocular conditions
Generalized progressive retinal atrophy (GPRA)
- Inheritance suspected
- Cone-rod dystrophy
- Ophthalmoscopic signs at 3–6 months
  (ACVO Genetics Committee 2007, Gelatt 2007)

Renal and urinary conditions
Urolithiasis – cystine
- Cystinuria results from an inherited defect in renal tubular transport of cystine and predisposes to cystine urolithiasis
- Breed at risk in case series
- Young dogs affected (2–5 years)
- Almost all cases are male

Dermatological conditions
Atopy
- Common disease, affecting around 10% of canine population
- Birth in autumn or summer is a predisposing factor
- Dogs between 1 and 2 years of age have the highest probability of an insurance claim for atopy
- Some studies show no sex predilection, others show females predisposed
- This breed had a risk factor of 7.6 cases per 1000 dog years at risk (if 1000 dogs were followed for 1 year, 7.6 would have an insurance claim for atopy)
  (Nodvedt et al. 2006)

Skin tumours
- See under Neoplastic conditions

Truncal solar dermatitis
- A combination of factors are required to cause this condition
- Flank and abdomen most severely affected
  (Scott, Miller & Griffin 2001e)

Musculoskeletal conditions
Cranial cruciate ligament rupture
- Common cause of hindlimb lameness
- Neutered individuals are predisposed
- Older animals are predisposed
American Water Spaniel

- Odds ratio in this breed 1.62
  (Witsberger et al. 2008)

Panosteitis
- Also known as enostosis, eosinophilic panosteitis
- Common
- Young males predisposed
- Odds ratio 2.0 (compared to mixed breeds)
  (LaFond, Breur & Austin 2002)

Neoplastic conditions
Canine cutaneous histiocytoma
- Breed at increased risk in case series
- Mean age: 3.6 years (50% < 2 years)
- Mainly head, pinnae and limbs

Cutaneous haemangiosarcoma
- Breed at increased risk in case series
- Mean age: 9.6 years

Mast cell tumours
- Breed at increased risk
- May be seen at any age (from 4 months onwards), but usually seen in older animals
- Predilection sites include hindlimb, perineum and scrotum
  (Goldschmidt & Shofer 1992, Goldschmidt & Mcmanus 2000)

Sweat gland tumour
- Breed at risk of apocrine ductal adenoma in case series
- Mean age: 9.1 years
  (Goldschmidt & Shofer 1992, Goldschmidt & Mcmanus 2000)

Ocular conditions
Cataract
- Autosomal recessive inheritance suspected
- Age of onset: 1 year
- Localization: suture lines progressing to nuclear and cortical cataract. Complete blindness at 3 years
  (ACVO Genetics Committee 2007)

Entropion
- Polygenic inheritance suspected
  (ACVO Genetics Committee 2007)

Generalized progressive retinal atrophy (GPRA)
- Autosomal recessive inheritance suspected
- Clinically evident at 1.5 years of age
  (Rubin 1989, ACVO Genetics Committee 2007)

Persistent hyperplastic primary vitreous (PHPV)
- Congenital, inheritance suspected
  (ACVO Genetics Committee 2007)

Neurological conditions
Cerebellar cortical degeneration
- Recessive inheritance suspected
- Uncommon condition
- Late onset
- Genetic test available (Optigen) – see Appendix
  (Henke et al. 2008)

Congenital deafness
- Prevalence in this breed not known
- Suspected to be inherited
  (Strain 2004)

AMERICAN WATER SPANIEL

Dermatological conditions
Adult-onset growth hormone-responsive dermatosis
- See under Endocrine conditions

Pattern baldness
- Hair loss occurs at about 6 months of age
- Ventral neck, thighs and tail are affected
- Frequency has been reduced by recognition of the problem by breed clubs
  (Scott, Miller and Griffin 2001d)

Endocrine conditions
Adult-onset growth hormone-responsive dermatosis
- Males may be predisposed
- Clinical signs seen at any age but often 1–2 years
  (Lothrup 1988)
**Ocular conditions**

Cataract
- Inheritance suspected
- Age of onset: <1 year
- Localization: anterior sutures
  - (Rubin 1989, ACVO Genetics Committee 2007)

Distichiasis
- Common condition, breed at increased risk
  - (ACVO Genetics Committee 2007)

Entropion
- Polygenic inheritance suspected
  - (ACVO Genetics Committee 2007)

**Musculoskeletal conditions**

Ankyloglossia
- Rarely reported condition
- Involves ‘tongue-tying’
- Young dogs affected
  - (Temizsoylu & Avki 2003)

Carpal laxity syndrome
- Reported in puppies
- Usually self-limiting
  - (Cetinkava, Yardimki & Saglam 2007)

**Neurological conditions**

Congenital deafness
- Prevalence in this breed not known
- Suspected to be inherited
  - (Strain 2004)

**ANATOLIAN SHEPHERD DOG**

**Gastrointestinal conditions**

Congenital portosystemic shunt
- Breed at risk in case series
- Clinical signs usually seen in young dogs <1 year
- Large intrahepatic shunts often involving the right liver lobe

**Ocular conditions**

Cataract
- Inheritance suspected
- Age of onset: <1 year
- Localization: anterior sutures
  - (Rubin 1989, ACVO Genetics Committee 2007)

Distichiasis
- Common condition, breed at increased risk
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**Musculoskeletal conditions**

Ankyloglossia
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Carpal laxity syndrome
- Reported in puppies
- Usually self-limiting
  - (Cetinkava, Yardimki & Saglam 2007)

**Neurological conditions**

Congenital deafness
- Prevalence in this breed not known
- Suspected to be inherited
  - (Strain 2004)

**AUSTRALIAN CATTLE DOG**

**Musculoskeletal conditions**

Patellar luxation
- Common condition
- Mainly medial luxation observed
  - (Alam et al. 2007)

**Neoplastic conditions**

Mast cell tumours
- Breed at increased risk
- May be seen at any age (from 4 months onwards), but usually seen in older animals
- Predilection sites include hindlimb, perineum and scrotum
  - (Baker-Gabb, Hunt & France 2003)

**Neurological conditions**

Hereditary polioencephalomyelopathy of the Australian Cattle dog
- Uncommon
- Affects young dogs
- Thought to be due to an inherited biochemical defect
  - (Brenner et al. 1997)

Lysoosomal storage disease – ceroid lipofuscinosis
- Suspected to be inherited
- Rare
  - (Bagley 2005)

**Ocular conditions**

Cataract
- Inheritance suspected
  - (Gelatt & Mackay 2005, ACVO Genetics Committee 2007)

Generalized progressive retinal atrophy (GPRA)
- Autosomal recessive inheritance suspected
- Progressive rod-cone degeneration
- Ophthalmoscopic signs at 3–5 years, blindness at 6 years. A second earlier form may exist
- Mostly reported in the USA and Australia
  - (Gelatt & Mackay 2005, ACVO Genetics Committee 2007)