Diseases Afflicting Abys, Somalis ... and Others

(This attempts to be a "what are they and what is their current status?" article on major diseases, by a breeder, not a veterinarian)

Feline Leukaemia Virus (FeLV)

FeLV is a retrovirus similar to feline immunodeficiency virus (FIV) and human immunodeficiency virus (HIV) that works by producing an enzyme that then infects healthy cells. It is now relatively uncommon, with a recent US survey showing a 2-3% incidence among the broad US cat population. FeLV is spread through viral infection from all fluid secretions of an infected cat. FeLV is not breed-specific.

The virus was first isolated in 1964, and by the late 1970s, there were FeLV tests available from two laboratories in the US, one in the UK and one in the Netherlands. They all required cell membrane antigens whose importation here was prevented by Australian quarantine regulations; hence our cats in the early 1980s had to be tested with blood samples sent to overseas laboratories. FeLV was then regarded as pretty serious, and the situation was complicated by the test being supersensitive, yielding about 30% of false positive results, so any cat that came up positive, needed to be tested again a few months later, to confirm or deny the original result.

Things brightened up considerably later in the 1980s when the ELISA (enzyme-linked immunoabsorbent assay) test was developed commercially. It quickly became available here, produced immediate results with no false positives, and could readily be performed by veterinarians. A decade later an effective FeLV vaccine became available, and so now we can readily test and/or vaccinate our cats. It is worth mentioning that vaccinated cats do not give false positive results to FeLV tests.

So if you test all your cats, the kittens you buy and the studs you send your queens to, there is no problem. If you don't, you run a serious risk — among the cat population at large, FeLV is still a significant killer. It should be noted that feline leukaemia virus is not breed specific, nor can it be spread across species, e.g. from cats to people or dogs.

Feline Immunodeficiency Virus (FIV)

This disease is similar to FeLV in cats and HIV in people. It is decidedly rarer than FeLV, and exists in the blood, saliva and other fluids of an affected cat. The most likely cause of infection is through bites or scratches from fighting with affected cats, generally in the wild, so confined pedigreed cats are unlikely to contract it, especially since the virus does not survive for long outside the cat's body. While FIV is unlikely to be spread through casual contact, FIV-affected queens can pass it on to kittens they are carrying. FIV, which is not breed-specific, cannot be passed on to humans.

There is a straightforward blood test for FIV often carried out at the same time as a FeLV test. There is also a vaccination which appears to work quite well, but it has the drawback that vaccinated cats will come up FIV-positive under the current test, with is not sufficiently refined to tell the difference between a cat that is infected and a cat that has been vaccinated.

Infected cats may show no outward signs for years. As with HIV in people, FIV gradually affects the cat's autoimmune system, resulting in an increasing likelihood of infections and chronic inflammations, as well as diarrhoea and eye diseases However, FIV affected cats can live for long periods in a reasonable condition, if efforts are taken to protect them from injuries and infections.

Progressive Retinal Atrophy (PRA)

Found mainly in Abyssinians, derived breeds (Somalis and Ocicats) and several breeds of dogs, this is a disease which, like the name suggests, causes its sufferer to go blind. The typical onset is about 18 months, and complete blindness occurs at about 5 years. PRA is inherited as an autosomal recessive, so that the mutation must be present in both parent cats for offspring to be affected. The rules are exactly the same as for being Cinnamon or Somali and the proportion of affected and carrier offspring is also the same.

No-one knows how the mutated gene (CEP290) got into Abyssinians, but in the 1970s a number of Scandinavian breeders gradually became aware that some of their Abys were blind. It wasn't particularly obvious, because the cats had got used to the layout of their houses or runs, but owners eventually noticed when they moved some furniture and some cats started walking into it. These cats were descended from a small group of mainly Dutch imports, and were

heavily inbred. I became aware of PRA in 1981when approached by a Swedish breeder who wanted a kitten. A veterinarian doing research at the local university, in Uppsala, had worked out the mode of inheritance, and recommended the importation of a kitten that was as unrelated as possible, but we still had to have the parent and grandparent cats tested for retinal atrophy by a veterinary ophthalmologist here. There were no DNA tests, and it was no use testing the kitten, because the earliest onset of PRA was about seven months.

The kitten, Nile Shasta, really was PRA free and produced several normal offspring for the Marasha and Brunnbäckens catteries. I was even able to help the researcher by sending her some Aby pedigrees that she didn't have. Kristina Narfström, probably the world's most famous veterinary ophthalmologist in the world, is these days Professor of Veterinary Ophthalmology at the University of Missouri, leading research at the Laboratory for Comparative Ophthalmology.

I am not aware of any Abys or Somalis in Australia suffering from PRA over the past couple of decades, but there certainly have been carriers. It is easy enough to make sure that your cats are normal: send a swab to a laboratory, and they will DNA-test it for both forms of PRA (rdAc and Rdy). You will then be advised if your cat is normal, a carrier or affected for each of them. In the unlikely but by no means impossible even of an adverse result, you can weed out PRA by mating carriers (or even affected cats) to normal cats, testing the offspring and keeping normal ones. It goes without saying that a normal cat stays forever normal. The other good thing is that cats that are blind from PRA do not actually suffer pain as a result, and as long as you don't move the furniture about, they will live out happy lives for many years.

Renal Amyloidosis

This disease affects mostly the kidneys, though amyloids can also be found in the liver, and less frequently, in other organs. Testing is by biopsy. In the course of the disease, amyloid deposits cause protein loss and eventual kidney failure. It is important to appreciate that RA is involved in only a small proportion of kidney failures. The mode of inheritance is not known, though amyloidosis in Abyssinians is called familial, meaning that it is more likely to occur in some "cat families" than others. However, my pedigree database lists many cats that did not appear to have amyloidosis though at least one of their parent cats died of it. A cat that has amyloidosis will die as a result, either from RA or something else that strikes because the cat is weakened by RA. The symptoms are weight loss, excessive drinking and urination, a shaggy coat and bad breath. Cats may die as early as 2 years or as late as 14. RA has been known at least since Dbl Gr Ch Gallantree's Casey Jones (b. 8 May 1970) died of it in 1984.

A lot of veterinary research has gone into finding out the mode of inheritance, and possibly even prevention and cure during nearly the subsequent 30 years, but without any break-throughs. It is now known that phosphorus binders like Amphojel and anti-inflammatories may alleviate the symptoms and prolong life somewhat. The good news, though, is that considerably fewer Abys died of it during the last decade than either of the previous two decades. I know no real explanation for that, so it's just as well that the chances of any one Aby getting RA are not much more than 1 in 1000. We've been through it once with an imported cat, in the early 1990s, and that was enough. Quite fortuitously and perhaps to illustrate the vagaries of amyloidosis, none of the three kittens in her only litter got RA.

Pyruvate Kinase (PK) Deficiency

An autosomal recessive, this is a disease in which the affected cat suffers from a deficiency in the pyruvate kinase enzyme normally found in blood cells. It is most frequent among Abyssinians and Somalis but occasionally found in unconnected breeds. It was isolated by Urs Giger, a Swiss researcher at the University of Pennsylvania in Philadelphia in 1998; Dr Giger also devised the DNA test is still used of detect the disease. The results of the test will be that the cat is normal (does not have PK deficiency), or is a carrier (does not have the disease but carries it as a recessive), or else it is affected, in which case it may suffer from cyclical anaemia from which it may die.

A lot of testing for PK deficiency was done by Australian breeders starting a dozen years ago, and a number of carriers were found, most but by no means all traceable to recently imported cats. A number of cats overseas had died of anaemia caused by PK deficiency, and so many of us took it very seriously, and gradually weeded out carriers. It helped a lot that the test was almost infallible, and identified carrier as well as affected cats. As a result of the measures taken by breeders, there are now very few PK deficiency carriers in Australia, but the picture is somewhat different in the UK, where testing and weeding out started later. A fairly recent survey of UK Somalis discovered 8 affected cats and 47 carriers in a sample of 141 cats tested. Over the years, PK deficiency tests have become more widely available – currently at the universities of Pennsylvania in Philadelphia, California at Davis, and Bristol in the UK, and a laboratory in Germany – better organised and less expensive.

As a postscript, there are two Abys and one Somali in Australia know to have tested as affected, one of them in my house. They are all around 9 years old. They get blood tests yearly, and all three owners can happily report that their cats have not had a day's illness or a bad blood test result among them. It would appear that the effects of PK deficiency are a lot milder than we initially feared. Finally, note that PK deficiency is not to be called "PKD"; that abbreviation is reserved for Polycystic Kidney Disease, which affects Persian and Exotics.

Feline Infectious Peritonitis (FIP)

There are a number of strains of the feline coronavirus (FCoV) in many cats, but fortunately they are not virulent. However, in some cats, and for reasons that are not fully understood, the FCoV they carry turns virulent and those cats get FIP, which invariably fatal. This transformation appears to be caused by a mutation and/or the cat having a weakened immune system, but either way the process is not yet fully understood. It is known that cats infected with FeLV are more susceptible to FIP, and that FIP-infected cats do not shed much virus, so it is not highly contagious. It is thought that cats under stress are more susceptible. Of the cats that have been exposed to FCoV but do not develop FIP, a broad survey shows that over 70% shed the virus for a while but then lose all symptoms, about 13% shed FCoV for life, and 4% seem totally immune and don't ever shed.

It has to be stressed that no more than 10% of cats exposed to FCoV develop FIP, and that can occur months or years after the contact. Early symptoms of FIP are loss of appetite and consequently weight, high temperature and a rough coat. They then develop very quickly – more quickly with so called wet (effusive) than with dry (non-effusive) – with fluid in the abdomen (wet FIP), fever, listlessness, weight loss and often jaundice. Cats that reach this stage are almost invariably euthanised. FIP does not appear to be breed-specific or heritable.

There is no single test for FIP and it can therefore be difficult to diagnose other than by the symptoms. Likewise, there is no way of testing healthy cats for their likelihood of developing FIP. There is a vaccine, but while safe it is not regarded as effective, and so it is not recommended by veterinary authorities. In catteries, the best way of minimising the chances of developing FIP is by keeping cats as healthy as possible, minimising exposure to any kind of infection and keeping litter tray clean.

While FIP is relatively rare, my impression is that over the past few years, more Abys have died of it in Australia than all of the other diseases covered by this article.

Research into FIP is continuing. One aspect is the search for immunosuppressive drugs to slow down progress of FIP in affected cats. As well, researchers are trying to find drugs to slow down or even prevent the virus replicating in a sick animal. The (US) Winn foundation awarded a $25,000 grant in 2012 to a Belgian university for a clinical trial of anti-immune evasive therapy in the treatment of FIP.

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