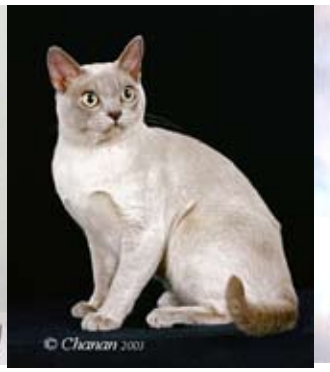
United Burmese Cat Fanciers

UBCF Newsletter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Volume I, Issue 3 -- September 2008



President's Message by Willa Rogers-Hawke

From the desk of the President

It gives me enormous pride to preside over UBCF once again and to be working with people with such dedication and talent. Nancy, our Newsletter Editor, has put together an edition which I believe will live on our coffee tables as a reference tool; with input from the likes of Drs. Pedersen and Lyons and Drs. Bell and Battaglia. Fantastic job, Nancy. (Also, special thanks to Nina Pearlmutter for her efforts in securing the articles from Drs. Bell and Battaglia.)

The UBCF was the defacto standard for the Burmese cat in the early days -- writing the original standards (much of which is still included in most standards today) -- and enjoyed the respect of Burmese breeders from all associations. As President, it's my goal to see that UBCF continues to cross all association lines and includes peripheral Burmese breeds such as the beautiful European Burmese or colors which are not currently accepted by CFA. This is not meant to be defiant in any way -- simply to be inclusive because our organization is nondenominational in concept.

With the able talent I am surrounded by, I believe we can all accomplish that goal.

Respectfully submitted,
Willa K. Hawke, President
United Burmese Cat Fanciers, Inc

Burmese Rescue

Through a variety of circumstances, some of our adult and senior Burmese find themselves without a home or in danger of losing their home. UBCF considers it an important responsibility for Burmese breeders and fanciers to help spread the word about these at risk cats, or to provide direct help in obtaining a new home for them. Following are only a few of the cats that currently need homes. This information comes from the Burmese Rescue Site at the National Alliance of Burmese Breeders (www.burmesecat.org), and we applaud their ongoing work and success in finding homes for Burmese that need rescue. We are using this forum to help in that effort. For additional information about these cats or others that need our help, please contact the rescue coordinator:

burmeserescue@yahoo.com

Denver, CO (8/31/08) – Denver area 2 Lovely girls - Zumi 3 years old and Oki 4 years old need loving home. They are very sweet and gentle girls. They get along with all animals and children. Owner cannot take them with her to new location. For more information contact Cheryl.Peck@UCHSC.edu

Manhatten, KS (8/14/2008) – 3 Burmese arrived at a high kill shelter in Manhatten, Kansas. "Rags," a neutered Male, is 1yr 2mo. old sable with yellow eyes. "Mitzi," female, also 1.2yrs, sable with yellow eyes. "NoName," male, is approx. 7mo. old and has an old absess on his neck, which is healing well. He has green eyes. for more information please contact Amy Hansen, ahanson@vet.k-state.edu.

Watertown, CT (7/14/2008) – McKitty is a 3.5 year old neutered male. His owner describes him as velcro. He needs a new home because his owner took a new job that requires lots of traveling. Please let his owner know details about your household (do you have other pets, etc.) in an email. Please email his owner Ginnto@hotmail.com for more information.

Health is Everything

Advertising slogans are abundant in our society, and frequently remind us that without good health we can't enjoy our lives. This is also true for our beloved Burmese. We bear a great responsibility as breeders and exhibitors of Burmese cats to assure that our cats and the kittens we help bring into this world are healthy and can enjoy their lives.

In an ideal world, Burmese breeders want the following scenario. We breed a beautiful Burmese female to a male who brings desirable and complementary qualities to the genetic mix. The female has an uncomplicated pregnancy and an easy birth. The litter is large and the kittens have good type. The queen has plenty of milk and the kittens grow quickly, weaning easily at the right age. They readily demonstrate Burmese qualities we love -- the intelligence, playfulness, confidence and affectionate natures that are hallmarks of our breed. We find good homes for all that we want to place. Transitions into their new environments are seamless for the kittens, and we receive glowing reports about how well they are doing and how they have enhanced the lives of the families who now love them. And the kittens grow into beautiful Burmese cats who enjoy long, happy and healthy lives. We keep a kitten out of the litter, some of us will show and grand him or her, and he or she becomes a breeding cat that produces a new generation of healthy and beautiful Burmese.

This perfect scenario can happen and has happened -- and when it does, the joy it brings to everyone involved is immeasurable.

But in the real and complex world of cat breeding, we are not always so lucky. Pedigreed cats like Burmese exist because different humans admire and prefer the various aesthetic looks and personalities that are found in different cat breeds. These preferences have caused the development of pedigreed cats that range from the small, shorthaired, energetic Singapura to the large, long haired, mellow Maine Coon.

To achieve and maintain specific qualities in a breed, the gene pools of those breeds must to a certain extent become inbred and genetically isolated. Tastes and standards of the breeds can change over time, and as breeders work to develop and set type to match those standards, they use inbreeding to accomplish this. With inbreeding, genetic diversity dwindles. And while cats inherit multiple copies of "good" genes from related parents which create the type that is wanted, they also can inherit multiple copies of "bad" genes. Those bad genes on top of a lack of genetic diversity can result in greater susceptibility to disease.

We may succeed in breeding a beautiful Burmese with exceptional type -- but if that cat develops chronic health problems or dies at a young age from a heart defect, FIP or another disease, everyone loses. The cat suffers physically, the breeder suffers emotionally and financially, and any family that developed an attachment to that animal suffers as well. Even the reputation of the breed as a whole can suffer.

Invisible Enemies

Most Burmese breeders and fanciers agree that the Burmese cat is at a crossroads. Besides the lethal genetic head defect that divides our community, and the inherent low genetic diversity in any pedigreed cat, we also recently learned through two research studies that the Burmese, along with the Singapura, has in fact the lowest genetic diversity of all cat breeds. That low level of genetic diversity is a serious problem, and an even worse one because the Burmese breed is further divided into contemporary and traditional gene pools. In conjunction with the declining numbers of Burmese being bred, this low level of genetic diversity can not only result in a higher incidence of health problems, but there is even a risk of breed extinction should a lethal disease take hold in multiple catteries.

Susceptibility to disease may have genetic components, but often the sources of disease are in our surroundings. Countless invisible enemies are everywhere. Bacteria, viruses, fungi and other tiny life forms are more or less ubiquitous in the environment. They also can travel with and even on us from other homes we visit, cat shows we attend, veterinary hospitals, and other catteries. Some of these are essential to our survival, such as the bacteria that live in our digestive systems. Others are not so friendly. To

keep harmful bacteria and viruses at bay, strong immune systems are essential both for humans and cats. A clean environment is also key to sustaining good health.

I have been breeding Burmese for almost 10 years. In that decade, even in a small cattery such as mine, I have had my share of problems. I've seen a litter of flat-chested kittens, had several cases of pyometra in queens, eye and upper respiratory infections in kittens and cats, various intestinal parasites, ringworm, and immune system problems that manifested at weaning -- which in several cases resulted in the deaths of affected kittens. Those who know me and have been to my house see how hard I work to keep my home and cattery clean -- I definitely have days when it's very hard to keep up, but I know how important it is. I also make every effort to provide my cats with good nutrition, to give the proper vaccinations at the right times, and to consult with my veterinarian and fellow breeders when problems arise. And I try to learn from the problems I have combated, so I can take preventative measures to avoid facing the same problems in the future.

During this time I have been working with American Burmese lines from different catteries which most breeders would consider outcrosses. Naturally there are other factors involved in what my cats and I have experienced. Nevertheless, to have seen this many health problems in spite of so many outcross pairings and so much work to sustain a healthy environment has not only been frustrating, but makes me recognize on a personal level the precarious situation our breed is in.

I also know that I am not alone in the battles I wage against feline disease. Most Burmese breeders and catteries will at some point see many of the problems I have experienced. To ease the anxiety of these problems when they occur, I sometimes like to joke -- albeit half heartedly -- that just when you think you've seen it all, that's when something new will surprise you. Prophetic words indeed.

The Call You Never Want to Get

No experience with disease could have prepared me for the one I feared the most -- Feline Infectious Peritonitis. It became a reality when I received a call from a tearful client, only a few weeks after I'd placed a kitten with her. "He's critically ill, the vet thinks he has FIP and that I should euthanize him." Hearing this news was like receiving a physical blow -- it was emotionally devastating and ultimately life changing. I was aware of the disease, of course, and almost every breeder I knew had experienced it at some point. Those few who hadn't, like me, felt very lucky. But I also had been told that sooner or later, if I kept breeding, I would see it. And now it had arrived. Even though I'd heard the average loss from FIP is estimated to be only one kitten per litter, the results in my cattery were four out of six littermates dying of dry FIP, and a ten month old half brother of theirs taken by wet FIP, all within a few months.

The ten month old, Buster Brown, as sweet and playful and affectionate a Burmese that you ever will meet, had gone to a loving home and received good care, but he had been bonded to me and apparently the stress of going to a new environment contributed to the onset of the disease. He deteriorated so quickly in such a short time it was truly shocking -- he was almost unrecognizable. His family loved him so much they struggled to make the decision to put him to sleep. I offered to step in and help them. When I saw how much he was suffering I knew there was no choice but to end his agony. That level of suffering is unforgettable.

Another kitten, Madison, had been evaluated by the client's vet several days after arriving at her new home. The vet declared after examing her,"This is the healthiest kitten I've ever seen!" Within a matter of weeks Madison had lost significant weight, was almost blind, and had difficulty walking. The woman who loved her did everything possible to save Madison, but there was nothing she could do to stop the disease so she made the difficult decision to euthanize.

Three of of the kittens that died had been placed in homes. When I realized there was FIP in the litter of six, I kept the two kittens that hadn't been placed and cared for them until it was time to end their pain and their all too short lives. There were also two more in the litter of six that were in homes, and I felt it was important to tell their families what had happened to the other littermates, and to offer to take the kittens back. Needless to say this news was very stressful for the two families, but they decided to keep

Madison
One of the four littermates lost to FIP

the kittens in the hope that they would beat the odds and survive. So far, those two kittens continue to grow and be healthy. Only time will tell if they will have long lives.

The grief that I and the families who loved these kittens experienced through all this was indescribable and remains a raw and open wound. And I can't begin to express the gratitude I feel to family, friends, fellow breeders, my veterinarian, and the researchers at U. C. Davis who supported us through this horrible ordeal. My one consolation is that three of the five kittens that we lost made it up to Davis for FIP research, in the hope they would help in the search for solutions to this terrible disease. I also chose to assure that no more kittens would be produced from these FIP susceptible lines -- I neutered the father and spayed the two mothers of the kittens that died.

Some of you reading this may be shocked at my admitting to having experienced FIP. It is a terrible disease that not only is greatly feared but has long been kept secret, whispered about but hidden from view, breeders worried about being branded as "an FIP cattery" knowing other breeders have applied that label even when unwarranted. And unfortunately this still happens. Indeed, FIP is a complex disease, about which there is much misinformation. It is affected by many factors and is most prevalent in catteries, though it also occurs in the stressful environments of animal shelters. Sooner or later it is likely to hit everyone who breeds cats. We must not be ashamed when it does. Instead, we must take action. Sharing affected bloodline, health and pedigree information can prevent future outbreaks. And supporting each other through diseases like FIP is in everyone's interest. Because, unfortunately, you and some of your kittens may be next.

Knowledge is Power

I am sharing my experience because we can no longer afford this kind of secrecy with FIP or any other problem that the Burmese breed faces, whether it is a disease, a lethal genetic defect such as the cranial facial mutation or cardiomyopathy, or the lack of genetic diversity in our breed. Shared knowledge and working together will help us not only to prevent problems but to find the solutions we need when diseases occur.

And there are many reasons to be hopeful that solutions to some of these problems will be found in the very near future. There are new tools available for genetic research. The feline genome has been mapped and will be completely sequenced within the next two years. Researchers are close to identifying genetic markers for diseases and defects that can help us make informed decisions for the betterment of our breed. And as a breed we also have an opportunity to participate in furthering research not only to help Burmese, but for the benefit of all cats. And that is a very exciting prospect that will be addressed later in this newsletter.

Health issues are complex and no newsletter can address them all. This newsletter looks at the big picture -- exploring genetics and pedigrees, FIP and the Head Defect, as well as UBCF members sharing tips for working with your veterinarian and for good nutrition and care of our beloved Burms. I hope the articles we have included will expand our knowledge, assist us with decisions about our breed, and empower us to work together for the future of the Burmese cat. We will continue with more health articles in future UBCF newsletters.

Most important, I hope every Burmese breeder and fancier will fully participate in providing all the information that is needed to assess the health of our breed, bank our cats' DNA for the future, and assist with the critical health and genetics research that is happening right now. We need EVERYONE to participate for the sake of our breed. Some solutions may be just around the corner, and they will allow us to work towards restoring Burmese as a whole to genetic health. That in turn will help us restore the reputation of the breed and to increase the number of breeders of the healthy litters of kittens we want. And we'll send those kittens on to new homes to bring joy to families for many, many years to come.

We are very fortunate to have some of the top animal researchers in the world contributing to this issue of the UBCF newsletter.

Please join me in expressing gratitude to: Dr. Niels C. Pedersen for his interview and research work on FIP; Dr. Patricia Pesavento for her work on FIP at U.C. Davis, and for providing the images used in Dr. Pedersen's interview; Dr. Leslie A. Lyons for her plan of action for the Burmese breed, and for her research on and progress towards isolating the cranial facial mutation gene; Dr. Jerold S. Bell for allowing us to publish several of his articles presented at the 2007 Tufts Genetics Conference; Dr. Carmen L. Battaglia for his article on Pedigree Analysis; and Nina Pearlmutter who obtained Dr. Bell's and Battaglia's articles for us. Thanks also to Sierra Milton for her thought provoking article on Omerta: The Breeder's Code of Silence. And to Olen Wilford for making us aware of that article.

And gratitude to all the UBCF members who helped with this newsletter.

Nancy L. Reeves
Burma Pearl Cattery

The Burmese Cranialfacial Mutation:

A Brief History and Update

What is the Burmese cranialfacial mutation or "Head Defect?"

The Burmese cranialfacial deformity is a genetic mutation that affects developing kittens in utero and presents at birth. This defect is not compatible with life. Kittens affected by this mutation can be born alive and should be humanely euthanized.

Dr. Leslie A. Lyons, of the Comparative Genetics Laboratory in the School of Veterinary Medicine at U.C. Davis, describes the defect: "The area of the upper jaw is duplicated and two hard palates and two sets of whisker pads can be easily seen. The head region above the upper jaw does not form properly. Eyes and ears are malformed and there is not complete closure of the skull. The brain appears to be protruding from the skull but it is generally covered by skin that may or may not be covered with fur."

How did this mutation originate?

It is not known which cat was the originator of this defect. However, according to Dr. Lyons, "During the 1970's, a alternative style Burmese cat was established. Phenotypically still within the CFA standard, this 'strain' of Burmese expresses a more rounded head with a higher frontal prominence, a shorter, broader muzzle, seeming larger and more prominent eyes, and generally a more demarcated nose break. This shorter, broader muzzle form has been referred to as the 'Eastern', 'new look', 'Contemporary', or 'more extreme'. The longer, narrower muzzle form is referred to as 'Traditional' or 'less extreme'.

"The 'more extreme' Burmese quickly became popular in the show ring and intensive breeding programs ensued. Shortly after the widespread establishment of the 'more extreme' cats, litters involving the 'more extreme' cats as both parents began to produce kittens with a severe congenital craniofacial deformity."

Dr. Lyons indicates "The most extreme facial conformations of the contemporary lines of Burmese cats have the highest likelihood of carrying the defect, but the exact demarcation as to facial conformation and carrier status is difficult to determine. Outcrossing of the contemporary lines of the Burmese cats to other breeds and Burmese populations naive to the defect have proven the mode of inheritance . . .

"The common genealogy of the cats producing the deformity in their litters revealed cats of common ancestry that had been extremely proliferative, including a line of show-winning cats that had been extensively bred."

Is the defect dominant or recessive?

According to Dr. Lyons, "The craniofacial defect that presents is very unique and distinct, not easily mistaken. The defect has an autosomal recessive inheritance pattern, implying two copies of the defect are required . . . When 2 carrier cats are bred, 25% (1 in 4) of the kittens should have the defect. But this is an overall average."

Dr. Lyons' research has also shown that "Most genes that affect structure are influenced by environment and other 'background' genes. When breeders use a 'more extreme' Burmese, the shorter face is more dominant, but variation is seen. Thus, the inheritance of the 'more extreme' facial structure is incompletely dominant. So far, we can not predict which of the 'more extreme' cats carry the head defect. Other mild defects and abnormalities provide some clues, such as the dermoids and the color variation on the midline of the nose, but they are not 100% accurate or 100% predictive. . .

"Until a marker is developed, breeders are cautioned as to using 'extreme' cats from contemporary lines, either within their program or as outcrosses. The defect does manifest in Bombays and breeds that have used contemporary Burmese. Since this defect is autosomal recessive, it will be difficult to eradicate without a genetic test."

How did Dr. Lyons become involved in research on the Head Defect?

In 1995 a Burmese head deformity research project was initiated by Dr. Lyons while at the Laboratory of Genomic Diversity (LGD) of the National Cancer Institute. This project was in response to a request by the National Alliance of Burmese Breeders (NABB) to the Winn Foundation. The project proposed combined prospective and retrospective studies for examination of the genetics of the craniofacial deformity.

After joining the faculty at U.C. Davis, Dr. Lyons continued her research on the craniofacial deformity at the Comparative Genetics Laboratory in the School of Veterinary Medicine. She is currently searching for the gene(s) that causes the head defect. Dr. Lyons has worked closely with the Burmese breeding communities for many years to collect samples for her research on the deformity.

An update on Dr. Lyon's research on the Head Defect

Dr. Lyons and her researchers have made considerable progress towards identification of the gene(s) responsible for the cranicalfacial mutation. They have identified the correct chromosome, which was announced in 2006. Progress since then has narrowed the search to the right area of the chromosome, and as of Spring 2008 researchers have reached the point of testing individual genes. Dr. Lyons is confident that a marker for the head defect will be found in the near future, and that there will be a genetic test for the defect.

What steps can to be taken to eliminate the Head Defect when a genetic test is available?

According to Dr. Lyons, at a presentation to the CFA Burmese Breed Council in June 2008, Burmese breeders will need to commit to genetic testing, so that the carrier status of each cat can be determined. Once both parents test clear, then there will be no need for their offspring to be tested, though parentage testing would be recommended so that the breeder can prove that a particular kitten came from two clear parents. Those cats who have been identified as carriers should not be bred together. However, Dr. Lyons feels not all carriers should be eliminated immediately as this would further lower the genetic diversity of the Burmese cat, which already is the lowest in the cat fancy.

The Burmese breed needs as large a group of breeders as possible, and a large group of cats. Carriers should only be bred to non-carriers, and breeders should work to slowly reduce the frequency of the head defect mutation over time. It is possible there will be an affect on type as a result of the culling of carriers.

Some breeders may wish to eliminate the carrier cats right away, others may wish to do it gradually, and perhaps some not at all. The genetic test will allow each breeder to make an informed choice. Dr. Lyons believes it's likely that there will be more carriers, if not all of them, in the contemporary lines. But all cats from both contemporary and traditional lines will need to be tested.

The above information on the Burmese cranialfacial mutation is based on the proposals, research and findings of Leslie A. Lyons, PhD, Comparative Genetics Laboratory in the School of Veterinary Medicine at the University of California at Davis, and from an update on the project that Dr. Lyons presented to the Burmese Breed Council at the Cat Fanciers' Association Annual meeting in June 2008.

The Burmese Cranial Facial Mutation

A Plan of Action for Burmese

This plan of action has been proposed by Dr. Leslie A. Lyons to improve the health and genetic diversity of the Burmese breed. It was included in Dr. Lyons' presentation to the CFA Burmese Breed Council in June 2008

1. **Do a very good breed census**
    - Identify all active breeders
    - Activate the retired breeders – ask them to help, to breed "just one more litter" or keep just one breeding pair
    - Identify all breeding cats -- ALL cats that are breeding today
    - Do a health survey
        - Art Graafmans is leading this effort in developing the survey
        - Add the information gathered previously by Dr. Susan Little on the kitten mortality survey

2. **Put together a comprehensive pedigree database**
    - Capture all the breeding cats correctly – do parentage testing
        - If pedigree errors are found, do your best to correct mistakes and move forward
    - Register breed ing cats in conjunction with U. C. Davis through a formalized web based system called Cat PHIR, sponsored by NIH, which includes a DNA registry and health history for each cat
        - Send DNA samples to Dr. Lyons at U. C. Davis
        - Proactively access PHIR to add health information for the cat
        - Davis will contact breeders/owners who have previously sent DNA for the HD study to ask if those samples can be submitted to the PHIR database
        - PHIR DNA and data will be available to other feline labs and researchers as well
        - Davis will ask for periodic updates on the cats in PHIR in the future

3. **Analyze genetic diversity in your breeding cats**
    - Find computer programs that can tell you which cats offer the most genetic diversity for your cat, based on your pedigree database
    - Using such a program takes the emotion out of determining who this cat should be bred to
    - For genetic health, the inbreeding coefficient (COI) should be low and the genetic variation high

4. **Try to import at least one cat per year**
    - Import cats from Thailand or other countries. There are health, type and color risks involved in doing this, but many of these can be tested for

5. **Consider outcrossing to another breed**
    - The cats closest to Burmese genetically are Bombays, Singapuras, Tonkinese, European Burmese and Southeast Asian import cats
    - Get away from thinking just about show cats
    - You may lose type in the short term but the long term benefit will be a healthier breed

6. **Actively share bloodlines as much as possible**
    - Use cats from other registries
    - Try to remove any barriers that keep breeders from working with each other.

7. **Participate in the current cranial facial defect, FIP and other feline research studies at Davis**
    - Research on these projects can only progress through the cooperation and assistance of Burmese breeders and fanciers

Omerta: The Breeders' Code of Silence

by Sierra Milton © 2004

Originally published in The Canine Chronicle
April, 2004

Although this article is about dog breeding, it contains important perspective for cat breeders as well.

What do most modern-day breeders and the Mafia have in common? What a strange question, you may say. It is, sadly though, a very real commonality. The answer is simply what Padgett, a well-known geneticist, refers to as the "Code of Silence" for breeders and perhaps more commonly discussed as "omerta" for the Costa Nostra. Both are deadly silences. It's easy to understand the reasons for the conspiracy of silence when it refers to criminals, but what reasons can a breeder possibly have for maintaining "omerta"?

The reason most often given for not sharing genetic information is the fear of being made the object of a "witch hunt." It lies much deeper though. It begins with ownership and the human need to see what one owns as being the best. Remember the "keeping up with the Jones" mentality? Everyone wants the very best and the accolade of owning the best. Admitting that what one owns or has bred may have faults is difficult for most people. Also at fault is the huge financial and emotional investment that breeders have in their dogs. Discovering that there may be defects in the sires and dams that breeders have so much of themselves invested in becomes frightening and causes many to refuse to even contemplate that their dogs may possess defective genes. Egos and fear of being labelled "poor breeders" are ultimately the reasons for breeders maintaining this detrimental code of silence.

Even more dangerous than the Code of Silence though is the refusal to contemplate defective genes may exist within a breeding program and be present for generations, quietly meshing through many bloodlines before manifesting itself. Could it be possible that dogs which appear healthy can actually be spreading dangerous, sometimes lethal genes throughout the breed community until finally two healthy, but gene-defective carriers combine to produce that first tell-tale affected offspring? Of course it is and time and again the geneticists tell us how this is possible.

Simplistically, breeders cannot see defective genes and what they don't see must not exist. Therefore using that logic, all the untested dogs must be as beautifully healthy inside as they are structurally beautiful outside. If only that logic were true! Unfortunately, far more emphasis is placed upon structural and superficial beauty simply because it is something that is easily seen, acknowledged and obtained. It's also something without any "unnecessary" financial investments. One doesn't need to pay for x-rays or blood tests or specialists' knowledge in order to evaluate how a dog conforms to a physical standard.

The real danger, though, comes not from those dogs who are tested, but from those breeders who keep their heads in the sand and refuse to believe that their dogs could be less than 'perfect'. We can begin to fix that which we reveal, but that which remains hidden is a threat to the future. But here omerta, that "Code of Silence" is very evident. Not only do these breeders hold fast to the belief that their dogs are untainted by defective genes, structural defects or temperament problems, but they also believe that no dog that they choose to bring into their breeding program through mating with their dogs could possibly be carriers either. After all, they only "breed to the best," and of course, that best just has to be perfect.

Now the truly criminal act occurs. These breeders are quite often very successful in the show ring; their dogs are thought to be the best -- after all, they have ribbons and placings and titles to prove how worthy their dogs are! Because of their show ring success, they are seen as breed authorities, people that newcomers to the breed trust for knowledge and information. And the information these newcomers get is that there are no genetic problems to be concerned with, no need to do that "expensive testing when the dogs are all healthy." Even more disastrous to the breed's future is that these breeders' attitudes begin to prevail. The newcomers see the success of these breeders' dogs and buy them (even though few, if any, have had even the most rudimentary testing for structural faults, poor health or defective genes). The newcomers then have a financial and emotional investment to protect which begins to spread this attitude, with predictable results. Soon, because these breeders are the "powers" within the breed (quite often judges, people selected to discuss the breed at seminars, breeders who command respective prices for puppies and stud fees, breeders seen winning), they use this "power" to ensure that it becomes unethical to discuss any defects, in either health or temperament, found in any of the pedigrees of their sires, dams or progeny of their sires or dams. All too

often one hears "I don't dare say anything if I want to win" or "there are three lines with epilepsy (or heart or eye or pick a health problem), but you don't need to know about them." Of course we need to know about them, how else are we to make intelligent decisions about which dogs would best benefit the future we plan for our dogs unless we consider not only the structural beauty, but also the hidden genetics that we are attempting to also improve?

What about the breeders who openly discuss the defects found in their own dogs? Unfortunately, they are all too often labelled as "poor breeders" and their dogs said to be "defective". They are shunned and spoken of in whispers and sneers. The very fact that these breeders are striving to share knowledge openly and to scientifically test their dogs make these breeders the subject of witch hunts by the very people who are either too cheap, too unconcerned, too egotistical, too uncaring about the future to even test their dogs, much less have the courage to honestly discuss their dogs. Instead of applauding these breeders who choose to share information, these breeders become shunned and hounded. As a result, and because human nature makes us want to be part of a group instead of outside the group, breeders begin to do what they do best, they maintain silence and lie or refuse to admit what they do know.

As more and more newcomers join a breed and inexperienced breeders and exhibitors all jump on the bandwagon of showing, owning and practicing the art of breeding, they turn to the breeders who are winning, equating winning with superior quality dogs. The breeders are, therefore, more determined to have nothing bad revealed about any of their dogs, further establishing in their minds the perfection of the dogs they breed and further increasing the financial and emotional investment that they have in perpetuating this theory. Winning in the show ring has nothing to do with genetic health. Indeed, a number of the winning dogs are carriers of genetic disorders at the least and, in some instances, are known to have genetic health disorders. While a genetic disorder itself, depending upon type and severity, should never preclude the dog from the genetic pool, it is absolutely mandatory that people be aware of any area of concern in order to breed intelligently. At the very least, the dogs that the dog is bred to must be tested and their backgrounds looked at carefully to limit the possibility of affecting more dogs or making more dogs carriers of the disorder. Yet, because the winners don't want to be labelled as "poor breeders" and lose the accolade of being the best (as well as the possible financial loss in not being able to sell puppies or stud fees at as high a price), the "Code of Silence" becomes even more firmly embraced.

The newcomers, because they want to be accepted, avoid talking about the sires and dams that produce poorly, whether it is structure, health or temperament problems. Also, they too now have a financial and emotional investment in addition to wanting to be accepted into the "winners club." They may even recognize trends in one or more lines in their own pedigrees, but refuse to acknowledge these trends and keep them secret for fear of being labelled.

Often, the breeders, while not openly acknowledging that there are any problems, will attempt to dilute the possibility of the disorder rearing its head by out-breeding to another totally different line. Dr. Jerold Bell, a well-known geneticist, has this to say about this method: "Repeated out-breeding to attempt to dilute detrimental recessive genes is not a desirable method of genetic disease control. Recessive genes cannot be diluted; they are either present or not. Out-breeding carriers multiples and further spreads the defective gene(s) in the gene pool. If a dog is a known carrier or has high carrier risk through pedigree analysis, it can be retired from breeding, and replaced with one or two quality offspring. Those offspring should be bred, and replaced with quality offspring of their own, with the hope of losing the defective gene."

Unfortunately, refusing to acknowledge or test for genetic disorders doesn't make them go away. What we can't see still has a huge impact on the breed and continuing to breed these carriers of defective genes allows the defect to take a firmer hold in the breed. Those breeders who try very hard to breed healthy dogs and take every scientific precaution to ensure genetic health are shunned for the very passion that should be applauded; the efforts they take are trivialized at best and more often ridiculed as "unnecessary" or "fear-mongering." As a result, these breeders work alone and, outside of their own kennel, their efforts make little impact on the breed as a whole.

Omerta can only be broken by people who have the courage, conviction and passion to ensure that the breed as a whole becomes stronger and healthier. Instead of witch hunts for those who have the heartache of dealing with the problems, the goal of applauding those with the courage and determination to speak out openly should be taken up by every breed club in every country. Awards in addition to those given to breeders who have the most winning dogs should be given to those breeders who work tirelessly to improve the breed. Prettiness and beauty doesn't improve a breed; genetic health and the ability to live a pain-free, healthy life far surpass beauty, but are more difficult to obtain.

The cost of genetic testing is not high when one looks at the effects that refusing to test may have on the breed. Ask any knowledgeable breeder whose breed has rampant heart, blood disorder, eye or hip problems whether they blame the lack of foresight and the refusal of past

breeders in making a further financial investment in the breed for the almost insurmountable problems now and the answer is predictable. In the UK, it is possible to do testing by certified specialists for hip, elbow, eye, heart, blood, immune disorders for around a total investment of £295.00 (far less in the United States), less than a cost of a puppy or a stud fee. It's possible to do far less testing, but at what cost? Will the breed suffer from heart problems in the future because a simple £7.50 stethoscope test (done through one of the breed-sponsored heart clinics, in this case the Boxer) was not important at the time? Will the breed be faced with trying to eradicate blindness years from now because a £16.00 eye exam (done through one of the many eye clinics held each month or free if done at Crufts dog show at the clinic they hold each year) was thought unwarranted? Will the descendants be filled with pain from bad hips and/or elbows because the breed moved well in the show ring and didn't look dysplastic to the naked eye? (X-rays necessary for hip and elbow evaluations are the most expensive testing at a cost of approximately £110 for hips and an additional £80 for elbows when done with the hips; unfortunately it takes six different films to evaluate elbows and the cost reflects the number of films necessary.) Testing for things such as von Willebrand's Disease (vWD) and thyroid testing (immune system) can be done inexpensively as blood tests at perhaps £30 and £50 each. Granted, testing for these genetic disorders won't guarantee that a problem won't occur in future breedings, but testing will greatly reduce the chances of problems and that is a good place to start.

If a breeder cannot provide proof in the form of veterinarian-issued certificates or reports that genetic testing has been done, the buyer should be aware that they purchase at their own risk! Caveat emptor! Breeders may claim that their dogs have never limped or that there is no need to do any testing because the breed is healthy. Some may even claim that their veterinarians have said that genetic testing was unnecessary. Those stances are irresponsible. Once again, genes are not visible and carriers of defective genes may themselves appear healthy to the naked eye. It is only with testing that we really know whether our dogs are affected or not and only then with honest evaluation of pedigrees having tested or affected dogs that the potentiality for carriers are realized.

What can we do to break the deadly Code of Silence? The majority, if not all, breed clubs have a code of ethics that require members to breed healthy dogs. One of the places to start is with the clubs. Instead of being social institutions or "good ole boy" clubs, these breed organizations could begin upholding the very real goal of protecting the future of the breed by demanding and requiring that genetic testing be undertaken prior to breeding. Far more serious than breeding a sixteen-month old bitch is the practice of breeding without taking every possible safeguard that genetic health is a priority. Yet, in many clubs "poor breeders" are identified by the age at which they breed or the frequency in which they breed rather than the very real criteria that proof of health be mandatory. Take the emphasis off winning; how many clubs determine "breeder of the year" based on the number of progeny that wins? Are there clubs that actually require that the breeder also must show proof that they are doing all they can do to ensure the future of the breed?

We can break the silence by commending those with the courage and determination to talk about problems, share successes and knowledge instead of ostracizing them. Omerta fails if every puppy buyer and stud dog user demands that proof of genetic testing is shown. The Code of Silence fails when we realize that it is not enough to breed winning dogs or to command the highest price for puppies or to have a stud dog that is used fifty, sixty, a hundred times; we must take back the passion with which we all first embraced our breeds and passionately work with determination toward a future where the numbers of genetic disorders are reduced each year.

If those you know breed without testing, ask yourself why: is it lack of courage in perhaps finding a carrier within their breeding stock? Is it because they fear a financial loss if they test? Is it because they truly believe that their dogs couldn't possibly be less than perfect? Is it because they fear they will lose their "top breeder" standing if they admit that there are problems that need working on? Is it because they fear that it will be harder to breed beautiful and healthy dogs? Or have they lost the passion with which they first loved the breed while they were climbing the road to winning success? Or, more sadly, is it because they really just don't care about that which they cannot actually see?

It's hard work and takes great courage to develop a breeding program using scientific methods and tests, but the hope of a better future should drive us all to that very commitment. The key is being able to work together without fear of whispers or silence. Omerta, the code of silence, can be broken if more of us decide that we are not going to tolerate the quiet any longer.

Sierra Milton has been involved in pure-bred dogs for over 36 years, initially in the US and Canada and presently in the UK. Sierra has published numerous articles on the ethical issues facing breeders, exhibitors, trainers and owners, as well as on the effect of evolving animal law on dog ownership and purebred activities.

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Responsible Breeding and Management of Genetic Disease

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Breeders and Breeding

Breeders of dogs and cats desire to produce the best with their matings. However, breeding has become more complicated today, and more people with intact dogs and cats are becoming "breeders". Understanding breed characteristics, historical selection parameters, and the continuous evolution of health testing has not been as important in the development of today's breeders. It is up to all veterinarians, breeders, and breed associations to educate prospective breeders on these aspects to promote healthy breeding practices for dogs and cats.

Adding to the complexity of breeding is the expansion of planned cross-breedings (designer breeds) to produce offspring. Recently this has become more of a factor in dog breeding than cat breeding, but it does occur in both. Therefore, the discussion is no longer between pure-bred and cross-bred, but between purposely-bred and random-bred dogs and cats.

There is a general misconception that mixed-breed dogs and cats are inherently free of genetic disease. This may be true for rare, breed-related disorders; but the common genetic diseases that are seen across all breeds are seen with the same frequency in mixed-breeds. A mixed-breed dog with hip arthritis has no less a case of hip dysplasia than a pure-bred dog. The only difference is that conscientious breeders test and label their dogs as dysplastic prior to the onset of clinical signs. I do not see a difference between the relative frequencies of old pure-bred dogs versus old mixed-breed dogs with hip arthritis requiring arthritis pain medication.

Testing for inherited hypothyroidism (for thyroglobulin autoantibodies by Michigan State University) shows 10.7% of 55,053 tested mixed-breed dogs to be affected. The average percentage of affected dogs for all pure breeds is 7.5%. This does not tell us that mixed-breed dogs are more prone to autoimmune thyroiditis: more mixed-breed dogs are tested based on clinical signs. However, these results show us that this hereditary disorder is seen frequently in both pure-bred and mixed-breed dogs. To those that feel that this disorder is not genetic, we look at the historical breed predilections for the disorder. Those breeds with the highest genetic propensity for autoimmune thyroiditis remain high over the years (example: 31.4% of English Setters tested), and those breeds with the lowest propensity remain low (example: 1.1% of French Bulldogs). Selection based on thyroid testing (and in the future direct genetic tests for liability genes) should reduce the frequency of this disorder.

In cats, the most frequent genetic disorder seen in practice is feline lower urinary tract disease (FLUTD), also known as feline urological syndrome (FUS). This genetic disorder affects the metabolism of normal levels of magnesium and other minerals in the diet, causing urinary crystals, bladder and urethral irritation, and secondary infection. This disorder occurs in pedigreed and random-bred cats with equal frequency. The most frequent single-gene disorder seen in practice is polycystic kidney disease (PKD), caused by an autosomal dominant gene. This defective gene is present in a high frequency (38% testing positive at the UC-Davis Veterinary Genetics Laboratory) in Persian and Himalayan cats. Due to its dominant inheritance, PKD is also seen in Persian and Himalayan cross-bred or random-bred cats, and is not a rare presentation in clinical

practice. Other common genetic disorders in cats include hypertrophic cardiomyopathy (where direct genetic tests are available for the Maine Coon Cat, Ragdoll, and their crosses), patellar luxation, and hip dysplasia.

The most common inherited disorders for all dog breeds according to the AKC Canine Health Foundation are: cancer, eye disease, epilepsy, hip dysplasia, hypothyroidism, heart disease, autoimmune disease, allergies, patellar luxation, and renal dysplasia. With the exception of renal dysplasia, all of these genetic conditions are routinely seen in mixed-breed dogs.

There are some defective disease-causing genes that mutated so long ago, that the mutation (and its associated disease) is found in evolutionary divergent breeds. The same ancestral autosomal recessive mutation for the progressive rod cone degeneration (prcd) form of progressive retinal atrophy (PRA) is found in the American Cocker Spaniel, American Eskimo Dog, Australian Cattle Dog, Australian Shepherd, Chesapeake Bay Retriever, Chinese Crested Dog, English Cocker Spaniel, Entelbucher Mountain Dog, Finnish Lapphund, Golden Retriever, Kuvasz, Labrador Retriever, Lapponian Herder, Nova Scotia Duck Trolling Retriever, Poodle, Portuguese Water Dog, Spanish Water Dog, Stumpy Tail Cattle Dog and Swedish Lapphund. This list continues to grow as more breeds are discovered with the same defective gene. The question is not, "Which breeds carried this defective gene during their development", but "Which breeds did not lose this defective gene during ancestral development."

It is also not surprising that prcd-PRA affected dogs (who must receive the defective gene from both parents) have been identified in Labradoodles (Labrador x Poodle crosses), and Cockapoos (Cocker Spaniel x Poodle crosses). Labradoodles are also being diagnosed with hip dysplasia, elbow dysplasia, and inherited Addison's disease; all recognized disorders in both parent breeds.

So, if breeders desire to produce the best with their matings, the basic question in dog and cat breeding becomes; "Who is a reputable breeder?" For purposely-bred dogs and cats (both pure-breeding and mixed-breeding), it is those breeders who perform genetic testing for breed-susceptible disorders. Official test results should be made available to prospective breeders, and the pet and breeding-stock purchasing public. It doesn't matter whether a breeder is a large commercial breeder, or only breeds once. In today's environment, not testing for documented breed-related hereditary diseases is irresponsible breeding.

Responsible breeding also involves knowledge of how best to use the results of genetic testing. For pure-breeds there are concerns about the breadth of the available gene pool and genetic diversity. Genetic test results should be used to benefit the overall health of breeds, not to limit it. A discussion of these issues, and breeding recommendations for genetic disorders based on different modes of inheritance are included in the 2007 Tufts' Canine & Feline Breeding and Genetics Conference poster abstract; Genetic Testing and Counseling: A Trojan Horse for Dog and Cat Breeds?

Genetic Test Results and Genetic Registries

For direct genetic tests, official test results of the parents, and/or the offspring (tested prior to placement) should be made available to prospective breeders or purchasers of pet or breeding dogs and cats. For some breed associations, the results of genetic testing are available in on-line, publicly accessible databases.

For disorders where there is no direct genetic test available, the knowledge of phenotypic test results (for affected, or carrier status if possible) should be made available in open health database registries. For most of these disorders, it is only through the open reporting of affected dogs and cats that knowledge of disease risk can be identified through the test results or health status of close relatives.

The Orthopedic Foundation for Animals (OFA: www.offa.org) maintains semi-open health registries for testable genetic disorders of dogs and cats. Applications for all of the hereditary disorders in their databases include a check-off to openly report ALL test results; both normal and abnormal. For many breeds of dogs tracking hip dysplasia for example, over one-third of the applicants check the box for open reporting. **It is important that as breeders and veterinarians we encourage open reporting of health results.** The days of stigmatizing conscientious, health-testing breeders who have produced dogs or cats with hereditary disease are gone. No one wants to produce affected offspring from their matings, and no one should be blamed if this occurs (unless the breeder is not doing the recommended health testing). It should be everyone's goal to produce healthy offspring, but this is not possible if the only available health information is about normal dogs and cats, but not abnormal dogs and cats. Once the majority of owners are initialing the box for open reporting, the OFA can change it to a check-off box for not reporting abnormal test results.

The Canine Health Information Center (CHIC: www.caninehealthinfo.org) was established by the AKC Canine Health Foundation and the OFA to assist breeds with managing breed-specific genetic disorders. The AKC national breed clubs determine the recommended testable disorders for the breed (whether tests of the phenotype or the genotype). If an owner is contemplating breeding their dog,

they can look up the recommended genetic tests to perform in their breed. Veterinarians can also assist prospective breeders by looking up and discussing the recommended genetic tests for the breed. Prospective breeding dogs (in either pure or cross-breeding) can be researched, and their genetic test results, as well as that of their close relatives can be studied.

The benefit of the CHIC system is that dogs gain CHIC certification by completing their health testing, regardless of their test results. **CHIC is about health consciousness, not health perfection.** As more tests for defective genes are developed, every individual is likely to carry some deleterious genes.

Veterinarians should ask for pedigrees and results of parental or early age health testing of pure-bred and cross-bred puppies and kittens on first presentation to their clinics. If the test results were not provided to the owner, many can be immediately searched in on-line databases like OFA or CHIC. **A lack of available test results shows that the puppy or kitten was not purchased from a health conscious breeder, and it may be liable to develop genetic disease.** The general public must be educated to become informed "consumers" when purchasing puppies and kittens. They should spend as much time researching the purchase of what will become a member of their family for 10+ years, as they do purchasing home appliances. Breeder health guarantees that provide for replacement of puppies and kittens with genetic disease are often worthless; as few pet owners will be willing to give up a member of their family once an emotional bond has been established.

Example: Cerebellar Abiotrophy (Ataxia) in Scottish Terriers

The Scottish Terrier Club of America (STCA) has provided all of the tools necessary to determine genetic risk of carrying the defective gene causing the autosomal recessive genetic disorder cerebellar abiotrophy (CA), or for producing affected puppies. CA is a degenerative neurological disease that causes slowly progressive incoordination from several months to several years of age. The defective gene is old, and widespread in the Scottish Terrier gene pool worldwide.

The STCA has an area on their website entitled CA Central (www.stca.biz/GrandCentral/) where a list of all confirmed CA affected dogs and their pedigrees is listed. The club maintains an on-line searchable pedigree database (www.stca.biz/pedigrees) that includes identification of all dogs with obligate CA risk. They also have a relative risk analysis calculator in CA Central that allows breeders to calculate the CA carrier and affected risk of dogs and of proposed matings.

The STCA has funded several studies to identify the autosomal recessive defective gene causing CA, and its members and breeders hope to some day have a genetic test for carriers. However, CA Central allows their breeders to minimize their current risk of producing Scottish Terriers affected with cerebellar abiotrophy, and reduce the frequency of the defective gene now, while waiting for a genetic test to be developed.

Health testing, and the knowledgeable use of test results is now an important requirement for responsible breeding. Breeders, veterinarians, and breed organizations must educate the general public of the need to check for health testing in their dog and cat purchases. As this happens, the overall genetic health of purposely-bred dogs and cats will improve.

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Alethea's Liberty Valance
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Genetic Testing and Counseling: A Trojan Horse for Dog and Cat Breeds?

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Disease-causing genes are searched for by researchers, and the resulting genetic tests are desired by breeders. Once obtained, it is a double-edged sword: its use can enable breeders to improve a breed or devastate it.

Most dog and cat breeds have a closed stud book, which means that there is a finite amount of polymorphic genes and genetic diversity present. They can only lose genes, not gain them through selective breeding.

The primary reaction of a breeder discovering that their breeding stock carries a defective gene is to retire it from breeding. As researchers, we often recommend using a genetic test to eliminate carriers from breeding.

Widespread elimination of all carriers of a high frequency gene can place a strong negative pressure on a gene pool. This can act to decrease the genetic diversity of the breed, cause a loss of other quality genes, and increase the frequency of other defective genes through genetic bottlenecks.

We know that most individuals carry some unfavorable genes. The more genetic tests that are developed, the greater chance that a breeder will identify an undesirable gene in their breeding stock. Making breeding decisions based on a single **testable** gene is inappropriate. **Any quality individual that would have been bred if it had tested normal should still be bred if it tests as a carrier.**

Prospective breeding animals represent the quality of the gene pool. A genetic test that was designed to help a breed and its gene pool should not be used to devastate it. As more genetic tests are developed, the discarding of individuals based on single, testable genes further restricts the gene pool. We should be offering genetic counseling recommendations that eliminates defective genes, but maintains breed lines and genetic diversity.

The best way to utilize genetic tests is to breed quality carriers to normal-testing mates, and replace them with quality, non-carrier offspring. This prevents affected offspring, while maintaining breed lines and genetic diversity in the breed.

Genetic Counseling and Control of Genetic Disease

The primary goal of domestic animal breeding is to maintain and enhance the quality of the breed. This is well understood in livestock production breeding, but often overlooked in dog and cat breeding. Breeders must consider all relevant aspects, which may include various health issues, conformation, temperament, and working ability. Health and diversity issues are important, but they must coincide with, and not replace selection for quality.

The goals of genetic counseling are to:

1) **prevent the production of additional affected individuals**

2) **decrease the frequency of the defective gene(s)**

3) **maintain a genetically diverse pure-bred population**

Genetic counseling recommendations need to take into account the dynamics and epidemiology of both the breed gene pool, and the defective gene(s). Rare or low frequency defective genes require more stringent selective pressure to prevent their spread. High frequency (breed-wide) defective genes require more pragmatic management that does not adversely affect the gene pool.

Historical Examples:

At the onset of testing for the autosomal recessive gene for GM1-gangliosidosis in the Portuguese Water Dog, the carrier frequency was 16%. The breed in America originated from less than ten individuals imported in the late 1960s and early 1970s. The defective gene was brought into the breed by the ancestral Algarbiorum line, which was the dominant breeding line. Breeders recognized that the Alvalade line did not carry the defective gene for GM-1 gangliosidosis, and preferentially selected dogs from this line for breeding, making it the major influence in the breed. Unfortunately, the Alvalade line carried the gene for late-onset prcd-PRA, including several influential affected imports. This defective gene was not present in the Algarbiorum line. The end result of selection was the near elimination of one ancestral line, and a breed-wide carrier frequency of prcd-PRA of 35%.

In cat breeds, genetic testing for the autosomal dominant genes for polycystic kidney disease in Persian and Himalayan cats (38% affected worldwide) and hypertrophic cardiomyopathy in Maine Coon Cats (over 30% affected worldwide) will require careful selection to maintain breed diversity. Obviously, breeders do not want to produce additional affected cats. However, the wide scale elimination of over 30% of the breed would put a significant negative pressure on the gene pool – even in these populous breeds. The amount of quality genes and quality cats that can be lost forever from such selection, and the amount of genetic bottlenecking could be devastating. Concurrently preserving the diversity of the gene pool over the next few generations while at the same time eliminating the defective gene is the most practical and desirable way to manage the disorders.

The American Burmese cat breed in recent years has split into a traditional and a contemporary head phenotype. Unfortunately, the contemporary phenotype that has been desired in the show ring is shown to be caused by the heterozygous genotype for the recessive, lethal, cranio-facial defect. Dr. Leslie Lyon's laboratory at UC-Davis is in the process of identifying the defective gene. Once a genetic test is established, it will be seen how the breeders will utilize the test for the best interests of the breed.

Genetic Counseling Recommendations

- Selection against a **single gene trait with a test for carriers** is based on the individual. Breeders only have to know the results of the individuals they plan on breeding.

- Selection against **disorders that lack a test for carriers, complexly inherited disorders, or disorders with an unknown mode of inheritance**, require knowledge of the carrier or affected status of related animals.

Autosomal recessive disorders:

With a valid genetic test for carriers, breeders should mate quality carriers to normal-testing individuals, and replace the carrier parent with a quality, normal-testing offspring. Carrier-testing offspring should be selected against for breeding. In this way breeders can prevent affected offspring, while eliminating the defective gene from their breeding stock in one generation.

Without a genetic test for carriers, knowledge of the affected or carrier status of relatives is important. This requires testing for the affected phenotype, knowledge of pedigree backgrounds, and relative risk pedigree analysis. An **open health database** is the best method for objectively disseminating this information. Breeders should mate quality, higher-risk individuals to lower-risk individuals. Replace the higher-risk individuals with their lower-risk offspring. Repeat the process in the next generation. If the majority of breeders plan matings with a carrier-risk below the average of the breed, then the frequency of the defective gene will diminish in the population. This has been successfully done in many breeds.

Relative Risk Pedigree Analysis: With simple autosomal recessive genes and no test for carriers, knowledge of affected and carrier relatives can provide an objective risk assessment. Relative risk is the **minimal risk** based on known risk from the pedigree. The following are obligate carrier risk values: Offspring of affected = 100%, Parent of affected = 100%, Phenotypically normal full-sib to affected = 67%, Full-sib to carrier = 50%.

If risk comes down from only one parent, then the offspring's carrier risk is half that of the parent. If risk comes down from both parents, then the **affected risk** is half the sire's risk times half the dam's risk.

S = risk of being carrier from the Sire.

D = risk of being carrier from the Dam.

Risk of being affected = S x D

The **carrier risk** depends on the knowledge of whether the individual can be excluded as phenotypically affected.

If you **do not know** if the individual is phenotypically normal or affected, then the risk of being a carrier is the sum of the risk from both parents, minus the risk of being affected.

$$\textbf{Carrier Risk} = S + D - (S \times D)$$

If affected individuals cannot reproduce, or it is **known** that the individual is not phenotypically affected, then:

$$\textbf{Carrier Risk} = \frac{S + D - (2\,(S \times D))}{1 - (S \times D)}$$

Pros: Relative risk pedigree analysis objectifies risk relative to the population. It allows breeders to understand their own risk, and that of their proposed matings. It allows breeders with higher-risk breeding stock to lower their risk through planned matings.

Cons: Relative risk pedigree analysis selects against entire families, based on relatives with risk. It selects against both carrier and normal individuals. However, without carrier tests it is an effective tool to reduce the frequency of both affected and carrier individuals, and has been successfully used in many breeds.

X-linked (sex-linked) recessive disorders: Replacing affected and carrier individuals with normal male relatives will lose the defective gene in one generation. Avoid breeding high carrier-risk females, as half of the male offspring from carrier females will be affected.

Autosomal dominant and X-linked dominant disorders: Quality affected individuals should be replaced for breeding with a normal-testing parent, sibling, or prior-born offspring. Ideally you do not want to breed affected individuals, as half of their offspring will be affected.

Complexly inherited (polygenic) disorders, and familial disorders with no known mode of inheritance: The knowledge of affected relatives is important in determining risk status. Open health database registries can provide this important information. Three factors should be considered:

1) Complexly inherited disorders should be viewed as threshold traits. A number of genes must combine to cross a threshold to produce an affected individual.

GC Traditional's Melody of Evita
© Preston Phography

2) Increased response to selection can be attained by attempting to break down the phenotype into measurable traits that may be more directly linked to the underlying genes. Example: Measuring joint laxity, acetabular depth, or liability to secondary boney changes in hip dysplasia.

3) The most important method to manage complexly inherited disorders is to select for **breadth of pedigree normalcy**. Phenotypically normal individuals with normal or mostly normal littermates have the greatest chance of carrying normal genes. Phenotypically normal individuals with affected littermates have a greater chance of carrying a genetic load of disease-causing genes. Normal parents who have a preponderance of normal littermates provides even greater confidence. An open health database that shows genetic test results of close relatives can provide this information.

Genetic tests are powerful tools, and as with any tool require an instruction manual for their proper use. When offering these tests to breeders, we need to provide genetic counseling advice that allows their use to be beneficial, and not detrimental to the breeds.

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Understanding Feline Infectious Peritonitis

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What is Feline Infectious Peritonitis – FIP?

FIP is caused by a feline coronavirus; coronaviruses of various species exist in most types of animals and humans and usually cause acute respiratory or enteric disease. FIP is the cause of death of 1 in 100 cats seen at veterinary teaching hospitals throughout the U.S. The incidence can be 5 to 10 times greater among young cats coming from catteries and shelters and is the major cause of abdominal fluid (ascites) and intraocular and neurologic inflammatory disease in cats under 3-5 years of age. FIP is virtually 100% fatal and there is no good prevention. The emotional toll of FIP is especially great, because it strikes suddenly weeks, months and even years after initial infection. Therefore, cat lovers usually experience this disease long after they have developed strong emotional bonds with their new pet.

Does FIP only affect pedigreed cats?

FIP affects both pure- and random-bred cats. However, the disease usually starts in young kittens, so it is closely linked with cat breeding. The disease is also enhanced by improper husbandry, especially resulting from overcrowding (shelters, large multiple cat households). We also know that genetic susceptibility may account for 50% or more of the risk of developing FIP. Although FIP occurs in all breeds, there is no doubt that certain bloodlines, and therefore certain matings, are more apt to produce kittens that eventually die from FIP. These genetic factors are most likely a result of the inbreeding that goes into breed development. Therefore, catteries are at the highest risk because they are subject to all three risk factors (kitten production, dense housing, genetic susceptibility). The likelihood that any given cattery will suffer at least one outbreak of FIP over a five year period is very high, and mortality in catteries can be 5-10 times higher than it is in the general population. Shelters have the second highest risk, and the greatest incidence is among kittens adopted during periods of overcrowding and prolonged stays. Most shelter kittens are random bred, with many coming from the feral cat pool. Random bred kittens are more genetically diverse in general, so husbandry factors are more important in causing FIP in this population than genetic factors. Husbandry factors are greatly influenced by the seasonal influx of kittens.

Why are you particularly interested in cats and FIP?

I was raised on a poultry farm in southern California. My dad fed many feral cats so they would stay around and keep the rodents down. As a result, I experienced and loved cats for as long as I can remember. When people came to the farm to buy eggs, I would sit with a box of kittens to give away, and sometimes I would get 25 cents for a kitten, which at the time I thought was a fortune. At first I wanted to be a cattle doctor, because I also grew up around beef and dairy cows and had show steers in high school. But when I went to veterinary school, I discovered that nobody knew much about cats and cat diseases. In those days there were lots of deaths associated with feline leukemia virus, but of course we did not know this virus existed until several years later. But from the beginning I had the most fascination for FIP. The first reported clinical cases of FIP were in 1963. My first publication on FIP was in 1965. I've been a member of the faculty at Davis since 1972.

What is the History of FIP?

FIP was first recognized as a specific clinical entity in the late 1950's. This timeline was based on decades of meticulous necropsy records kept by pathologists at the Angell Memorial Animal Hospital. There was a steady increase in the incidence of the disease in the 1960's onward, and it is currently one of the leading infectious

causes of death among young cats from shelters and catteries. The reason for the sudden emergence of FIP is not known, but there are at least two possible explanations. First, it is noteworthy that FIP appeared within a decade of the initial descriptions of transmissible gastroenteritis (TGE) of pigs in North America. The causative virus of FIP is closely related to TGEV of pigs and canine coronavirus (CCV), although they are still genetically distinguishable. However, mixtures between these three viruses are known to occur. At least one strain of canine coronavirus can induce mild enteritis in cats and enhance a subsequent infection with FIPV, indicating a special closeness to feline coronaviruses. Therefore, CCV may be a more likely parent of FECV in this scenario. Another related possibility is that the FIP mutation occurs only in a relatively new strain of FECV, and that this new strain only evolved in the 1950's. Coronaviruses such as FECV are continuously mutating as a result of the manner in which their genetic material (RNA) is replicated. Therefore, genetic change, either among themselves or through genetic mixing with closely related coronaviruses from other species, could have either allowed a coronavirus of another species to take up host in cats or to alter a strain that existed prior to the 1950s.

An alternative non-genetic explanation may involve changes in how cats were viewed as pets and their husbandry. There was a dramatic shift in the status, keeping, and breeding of cats as pets after WWII. The numbers of pet cats greatly increased, purebreeding and cattery rearing became increasingly popular, and more cats, and in particular kittens, found themselves in shelters. These large multiple cat indoor environments are known to favor feline enteric coronavirus (FECV) infection and FIP. Interestingly, feline leukemia virus (FeLV) infection also became rampant among indoor multiple cat households during this period, and FeLV infection was a significant enhancing factor for FIP until it was pushed back into nature as a result of testing, elimination/isolation, and eventual vaccination in the 1970s and 1980s.

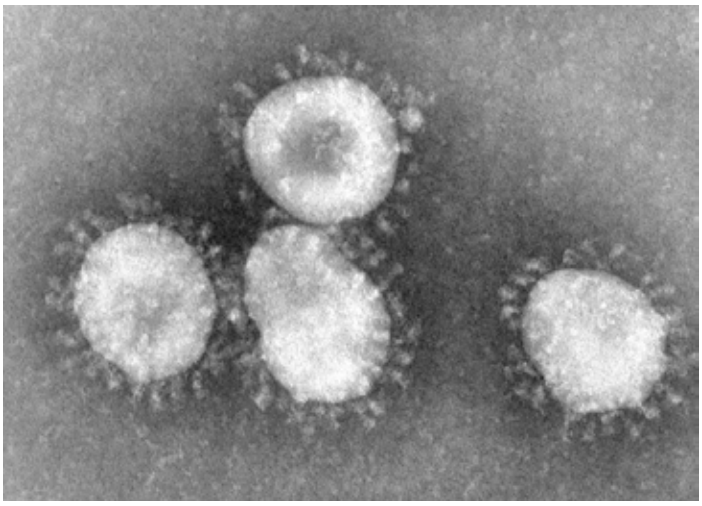
How is the coronavirus spread between cats?

Coronviruses are ubiquitous among all cat populations and the principle one of cats is correctly referred to as feline enteric coronavirus (FECV). FECV is present in virtually all catteries with 6-8 or more cats and in 40% or so of the kittens relinquished to shelters. The enteric virus in the cat population lives in the digestive tract and is shed in feces. Cats can shed the virus for 4-6 months, or for a year or more in a continuous or intermittent fashion. Recurrent infections are also common. FECV is easily spread through litter and litter dust, and can be carried from place to place on people's bodies and clothing. Virus contaminated material is easily transferred to the paws and fur of susceptible cats and then ingested during grooming. Kittens are infected by other cats at about 9-10 weeks of age, although one report places it as early as 3 weeks.

How does the coronavirus turn into FIP?

FIP is caused by a mutation of FECV, which is ubiquitous among cats. Although the mutation of FECV to FIPV is common, it is fortunate that only a small percentage of cats exposed to this mutant virus will get FIP.

FECV is undergoing continuous mutation and several genetic forms of the virus may co-exist in the same animal at the same time. Most of these mutations have very little effect on the behavior of the virus and merely serve to genetically reflect the region from which the virus originated. However, mutations that inhibit or knock out the function of a certain small gene (called 3c) have a pronounced effect on the biologic behavior of the virus. All known isolates of FIP virus that we have studied, and that have been reported by others, have various types of mutations in the 3c gene. Mutations within the 3c gene with the potential of causing FIP are common. One study indicated that 20% of the kittens infected with FECV will produce an FIP mutant. Of course, only a fraction of the mutants will go on to produce FIP, depending on host resistance factors (genetic or non-genetic). This FECV to FIPV genetic change is referred to as the internal mutation theory. The internal mutation theory has two corollaries: 1) that each cat that develops FIP, even if it is a littermate, closely related or commonly housed, has a different mutation in the 3c gene, and 2) that



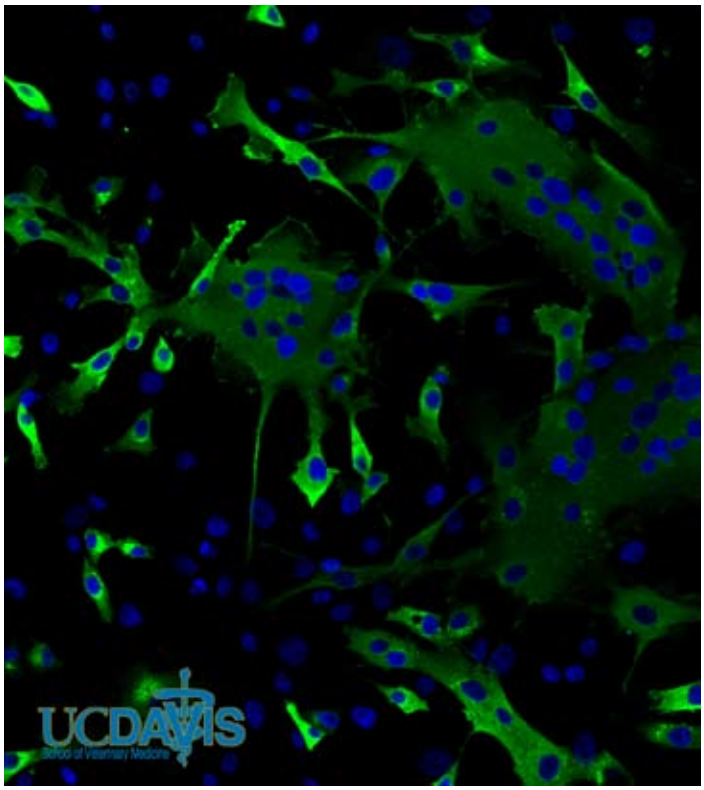
This photo from the CDC (Centers for Disease Control) is of the SARS virus, which is a corona and looks just like feline coronavirus. "Corona" refers to the rays of the sun, and describes the distinctive crown seen around the virus.

horizontal (cat-to-cat) transmission of the FIPV mutant is uncommon. We have reconfirmed corollary 1, and have confirmed corollary 2 in concept but not in fact. Reconfirmation for the internal mutation theory came from a recent outbreak in three kittens in a litter of Scottish Folds and in a half-sibling from a second litter. All four FIPVs had significant, but different, mutations in their 3c genes, but were closely related in all of their other genes to one of two different FECVs detected in the feces of one of the kittens. We have found that many cats with FIP are in fact shedding the same FIPV in their feces that is in their diseased tissues. However, for some reason, it does not appear to be highly contagious.

What are the signs of FIP?

Signs of FIP arise weeks, months, and in rare cases years after initial infection. During this quiescent stage, the cat may be asymptomatic or suffer from vague signs such as stunted growth or increased susceptibility to other common infections. Many breeders and even clinicians believe that FIP can cause upper respiratory disease signs during its early stages; this is not technically correct, because upper respiratory disease is usually caused by herpesvirus, chlamydophilla, mycoplasma, etc., and not directly by FIPV. With time, many cats win their battle with this infection, while others lose. However, "losing the battle" may occur over a very long period of time; only terminally, when the cat's defenses collapse, do the more characteristic signs of FIP develop. This capitulation to the virus explains why cats with FIP seldom recover, because a loss of immunity is extremely difficult to reverse. Cats who develop clinical cases of FIP may initially show nonspecific symptoms such as loss of appetite, depression, rough coat, weight loss, a fluctuating antibiotic resistant fever, and increased susceptibility to secondary infections (such as respiratory disease). More specific signs of FIP vary depending on the form of the disease (wet vs. dry) and the organs that are involved.

The most common form of the disease is referred to as "wet FIP." Wet FIP is caused by inflammation of the linings of the abdominal viscera, and less commonly of the thoracic organs. This inflammation exudes large volumes of a characteristic mucinous, yellow-tinged fluid (exudate). Therefore, the major clinical sign in the wet form of FIP is ascites and abdominal distension (abdominal involvement) or dypnea (thoracic involvement).



This image is of FIPV growing in a laboratory flask containing normal cat cells. The cells that are stained apple green are infected with FIPV, and the nuclei of the tissue cultured cells are blue. Some of the infected cells have many nuclei. Cell cultures are often used to study the behavior and properties of the virus, rather than infecting laboratory cats.

FIP can also take a more chronic form referred to as "dry FIP." Dry FIP, as the name implies, is not associated with fluid accumulations in the abdomen or chest, but rather with more localized masses in the kidneys, spleen, liver and terminal bowel, eyes, and the linings of the lungs and heart, and central nervous system. Uveitis (intraocular inflammation) can affect the eyes, making them look cloudy and changing the color of the iris. Inflammation can enter the brain and spinal chord and cause a spectrum of progressive neurologic abnormalities. FIP accounts for over one-half the cases of inflammatory intraocular and nervous system disease in cats under 3-5 years of age. Although unappreciated in the past, we now know that cats in the terminal stages of FIP are often severely immunocompromised. This explains why common bacterial infections may complicate the disease picture in cats with FIP.

Is FIP contagious?

Cats with FIP do not appear to be very contagious to cats that they come in contact with. Although this has been based mainly on clinical observations, it has also been confirmed by laboratory studies. We have not observed contact transmission in experimental settings. Furthermore, cat-to-cat transmission implies that every FIPV isolated from a group outbreak of FIP will be genetically identical in

its 3c gene mutation. As I mentioned earlier, we have yet to observe this. However, we now know that FIPV is present in the feces of most cats with FIP, so horizontal transmission is theoretically possible, although very uncommon.

How do genetics, stress, and other infections play a role in FIP?

FIP is not a breed specific disease, but does follow certain bloodlines within breeds. Heritability accounts for about 50% of the incidence. Environmental factors influence the other half.

The age of the cat at the time of initial FECV exposure plays an extremely important role in whether a cat dies from FIP. Kittens usually began shedding FECV at around 9-10 weeks of age, which places their actual exposure a few days to a week earlier. The immune system of the kitten is rapidly maturing during the period between 6-16 weeks of age. Therefore, the first exposure of most cats to FIP causing mutants occurs during a time period when their immune systems are still developing. This lack of development enhances the likelihood of a FIPV mutant to gain a strong foothold into the body. Just as there is an age susceptibility, there also appears to be an age resistance. FIP is seldom seen in cats over 3-5 years of age, and most cases occur before 16 months of age.

In the 1970s, when tests for FeLV became available, we discovered that one third to one half of all cats with FIP were also FeLV positive. In later experiments, we showed that cats that had resisted infection with FIP virus would develop FIP shortly after being infected with FeLV. This meant that FeLV infection somehow interfered with the ongoing immunity to FIPV. With the elimination of FeLV as a major infection of cats, we no longer see such a strong relationship, especially among catteries and shelters where FeLV control programs are in effect. Most cats with FIP in the present age, with the exception of a few household pet cats, are not FeLV infected.

Anything that stresses cats can depress immunity and also increase the likelihood that FIPV will establish itself in the body. Stress may also allow an FIPV that is being successfully contained to become active. The effect is even more powerful if the stress occurs at or shortly after the time the cat is exposed to the virus. Stressors can include overcrowding, weaning, spaying or neutering, other infections, being placed in a new and strange household, adding new cats to a household, shipping cats to new owners or other catteries, or stresses of pregnancy, parturition and lactation. Disease caused by feline herpes virus and other common upper respiratory pathogens are good indicators of cattery or shelter stresses. If a cattery or shelter is having a lot of problems with these upper respiratory infections, it is likely that they will also have problems with FIP (especially if the genetics are unfavorable as well). For instance, an area SPCA had a huge FIP problem in the kittens they were adopting out. It was kitten season and the facility was overcrowded with cats and they had to stay for longer periods awaiting adoption. There was also a lot of upper respiratory disease. After limiting their intake of cats, overcrowding was eliminated and cats were adopted after shorter stays. The FIP problem decreased to negligible levels, as did the respiratory infections.

Is there a definitive test for diagnosing FIP?

The diagnosis of FIP should be relatively simple, given its affinity for younger cats, its strong tendency to involve catteries and shelters, the typical physical and historical findings, and numerous characteristic laboratory abnormalities. Nonetheless, it somehow remains one of the most difficult of diagnoses for many veterinarians. The truth is that veterinarians have little trouble in placing FIP high, or at the top, of their diagnostic list, but have great difficulty, and even reluctance, in confirming their diagnosis. This is probably because FIP is viewed as a death sentence, and we are reluctant to confer such a sentence without certain proof.

Although a definitive test result would assist decision making, a certain diagnosis can be based on cumulative odds rather than a single, simple, definitive test result. A young cat from a cattery or shelter with chronic uveitis and/or neurologic signs, high serum proteins, hyperglobulinemia and hypoalbuminemia, fluctuating antibiotic unresponsive fever, leukocytosis with a lymphopenia, and an anemia of chronic disease can have no other disease than dry FIP based on odds alone. Likewise, the same cat with similar history and laboratory findings, but with yellow-tinged, mucinous, inflammatory ascites is highly unlikely to have any other disease than wet FIP. It is interesting that a cattery owner or cattery worker is often the one to cue in on the correct diagnosis based on the simplest of observations and intuition.

In an attempt to reach the elusive definitive diagnosis, veterinarians rely on dozens of tests that claim to highly correlate with the disease or to be diagnostic. I do not have time to go into the dozens of tests that fall into this category, or the good tests that are improperly done, or improperly interpreted, that lead to misleading positive or negative results. In truth, the only good definitive way to diagnose FIP is to identify the virus in macrophages within lesions or ascetic/pleural fluid by a procedure called immunohistochemistry. PCR would work equally well on diseased tissues or fluids, but many

of the current tests are improperly designed and conducted and frequently yield misleading results. In some cases, the proper fluid or tissues cannot be obtained pre-mortem. However, there is no excuse for not doing such definitive tests post-mortem.

This brings me to a final point, a necropsy should be done by a qualified veterinary pathologist on any cat that requires a proper diagnosis. However, even veterinary pathologists will hem and haw about a definitive diagnosis, even when faced with incontrovertible historical, clinical and histological evidence. Make them make a definitive diagnosis, either based on proper reading of the odds or by doing immunohistochemistry on lesions.

What is the best way to care for a cat that has FIP?

There is currently no cure for FIP; therefore, the primary concern needs to be making the cat comfortable and deciding when to quit. Cortisone can help reduce inflammation and encourage appetite. Good nutrition, hydration and non-stressful environments are also important, but in almost all cases they serve only to prolong the inevitable. Therefore, we will encourage some owners to go with symptomatic treatment, but only if the animals are not suffering. There have been reports that the feline interferon omega is effective in combating FIP. We actually tested it against FIP years ago and it did not work. Fortunately, a double blind, placebo controlled study was recently reported from Europe on the use of interferon omega in treating FIP. Cats receiving this very expensive treatment fared no differently than placebo treated cats. This will hopefully stop the use of this treatment in the US and other countries, although some people still believe more in anecdotes than scientific trials. Unfortunately, veterinary medicine is filled with anecdotal treatments.

How do you know when to euthanize a cat that has FIP?

This is a decision only you can make, and it is a difficult one. I would never suggest euthanizing a cat, even with FIP, as long as it looks and acts fairly normal. Miracles do happen, but they can't happen unless they are provided time to happen. However, I also cannot argue with those owners that decide to end suffering at an earlier stage, given the grave prognosis.

I always tell owners to decide to put an animal down when it no longer takes pleasure in life. But cats can feign health to the last moment and you often regret in retrospect not making the decision earlier. I did the same thing with one of my cats that was dying of cancer. He actually was dead one morning, even though I still thought he had time to live and seemed reasonably content. There is also a myth that if a cat is still purring that it is still enjoying life. But research has shown that cats purr even when in extreme pain, it is another way that they mask illness.

After you have had FIP in an environment, how long should you wait before a new cat or kitten is brought in, and what other steps should you take?

Remove any cat related items that you cannot wash or disinfect, such as a scratching post or soft toys. Clean and disinfect everything else in the environment that you can. Time will take care of the rest, because viruses of this type are not long-lived in the environment. We generally recommend a couple of months. These steps are undoubtedly an overkill when it comes to FIP, but these recommendations are standard for most infectious diseases and we try to keep everything simple and consistent.

If FIP occurs in a cattery, how do you decide whether and which cat to spay/neuter?

We know that genetics play a strong part in FIP - at least 50% of the incidence or more has a heritable component. We know that susceptibility is carried both in paternal and maternal lines, but we have suggested, at a minimum, that paternal lines that throw kittens that die from FIP not be used for breeding. This is because toms breed multiple queens and sire dozens or hundreds of kittens, and have the greatest influence on how bloodlines are developed. This is true of a lot of diseases – the "Founder effect." If females are genetically weak, and bred to weak toms, that is when you get into problems. If toms are genetically strong, and queens are genetically weak, the male's resistance genes seem to mask this weakness. The best scenario is to not breed either susceptible toms or queens, but not using problem toms is the best possible alternative in the interest of doing the most with the least disruption of breeding practices and bloodlines. However, if there are multiple losses from FIP in a litter, remember that susceptibility comes both from the paternal and maternal lines. It's also important to consider the cat – if you believe the cat may be at risk for FIP, avoiding the stress of being a breeding cat may help prevent the disease for that individual.

If one kitten in a litter has FIP, how likely is it that other littermates will be affected as well?

We know that if one kitten in a litter gets it, that the others are several times more likely, but this is not absolute. If the overall incidence is 5% across the spectrum of young cats produced in a cattery, it could be 10-50% or higher among the remaining littermates. I have seen one kitten in a litter of 6 killed by FIP and I have also seen 5 out of 6 develop the disease. Time is the only thing that will determine the fate of healthy siblings.

Sometimes both bacterial and viral infections exist in a cat with FIP – which came first?

It is really impossible to say which came first, because it is like chickens and eggs. A given cat could have had some underlying immunosuppression that made it susceptible to a number of common feline pathogens as well as FIP. It is also possible that the cat suffered from FIP for a long time and that this was the cause of immunosuppression and the other infections were secondary.

What is the best way for breeders to prevent FIP?

Resistance is the ability of the immune system to cope with a disease. We know that 50% of the incidence is heritable, and we know that resistance (or susceptibility) factors exist in both toms and queens. However, culling problem toms is the simplest genetic procedure to reduce incidence. Toms produce far more litters and kittens than queens, and therefore have a much bigger effect on the disease. Good judgment and husbandry will influence the other 50% of the equation. Pick the largest and strongest in a litter to keep for breeding and avoid kittens that are slow growing and prone to other infections. Spay and neuter cats that throw FIP and adopt them into good homes. Avoid stress and overcrowding; maintain only those cats deemed necessary for your breeding program and chose mating wisely to limit kitten numbers. Keep cats in small, separate groups. Consider isolating the kittens from the mother at weaning to avoid exposure to the virus. Don't mix very young kittens with older kittens. If you can limit coronavirus exposure until 12-16 weeks of age, when the immune system is better developed, the likelihood of developing FIP will be less. Follow accepted protocols for vaccinations and practice good husbandry to limit other infections. Clean and disinfect cages and litter boxes regularly. The corona virus is easily killed by bleach and other disinfectants.

Is it possible to have a corona virus free cattery?

This is extremely difficult, because the virus is ubiquitous in the environment and easily spread by cats and on people. Isolation of queens and early weaning has been touted in the UK and is used in the US. However, UK catteries are small and such a program can only succeed in smaller catteries and with exceptional isolation facilities and quarantine procedures. Moreover, even if you could produce a virus free cattery environment, the moment a kitten or cat goes to new environments such as a pet home it will most likely be exposed to FECVs. Therefore, we do not recommend this procedure unless simpler husbandry practices totally fail to reduce the problem.

Isn't there an FIP Vaccine?

A vaccine was developed and is available. However, it has to be used in kittens at least 16 weeks of age (most cats are already exposed to coronavirus at this age), it is not effective in cats already exposed to coronavirus (which is most cats), it is not effective against the common serotype of FIPV, and even when all factors are optimal, it has low efficacy. In short, it does not work in the environments where it is needed most (catteries and shelters) and is not justified in older pet cats where FIP is hardly seen. We do not recommend its use.

Why is there hope now for progress on finding ways to prevent and treat FIP?

It is true that there is still no cure, or totally effective prevention. But we understand the virus and the infection much better now. We have new tools that allow us to look at viruses at the molecular level. Any knowledge about the virus and how the host cat responds to it will have influence down the road. The Feline Genome has been sequenced and in 2 years it will be complete. We will be able to identify viral genes responsible for causing disease (which will facilitate antiviral drug development) and host genes that confer resistance/susceptibility (which will facilitate genetic control).

There are only two ways to affect the corona virus at the current time – through husbandry and careful matings. Husbandry techniques can help prevent the spread of the virus – a great deal of research has been done and continues in this area at the Shelter Medicine program at Davis.

Understanding how the immune system affects both resistance and the form of the disease (wet vs. dry) will be

important. Immunity studies focus on how to modify the immune system's reaction to the virus. Understanding how to block inflammation and the development of anti-viral drugs would be ways we could fight it. There is no reason why these can't be developed. In fact this was happening following the appearance of the severe acute respiratory syndrome (SARS) - another coronavirus disease, but of humans. However, this research was curtailed when SARS failed to spread to the general population and was easily contained. But it made human researchers interested in the corona virus. Drugs could be developed and used in FIP with some effect - like HIV/AIDS it could become a manageable disease

If we can discover the genetic basis for susceptibility, we will be able to offer genetic testing and breeders can breed out the trait over several generations, while preserving valuable bloodlines. This is exactly what breeders are doing with many other genetic diseases, such as polycystic kidney disease in Persians and breeds with Persian blood.

How can Burmese and Birman breeders help with FIP research?

Genetic research has great potential, but it takes time and money. Because FIP is a purely animal disease, there will be limited funding from sources such as the NIH. It's important to start banking DNA and assembling pedigrees showing relationships between affected and healthy cats. Breeders should not be ashamed of FIP. If you breed enough cats, long enough, you will experience FIP. The reluctance of breeders to talk about it has been a huge detriment to FIP research. For instance, if every one would cooperate, we could determine whether or not there is a genetic basis for FIP resistance or susceptibility in rapid time. If there is a genetic basis, we could then determine the gene or genes involved and develop tests to predict which cats to breed or not to breed. Fortunately, some breeders are now coming forward and cooperating in raising funds for FIP research and providing information and materials (such as DNA and FIP virus isolates) from the field. Such people have come out in the past, but they usually get frustrated and give up after a while. So if we want to succeed in understanding this disease we must all work on together to collect the DNA, case and pedigree information that will help us advance our research.

FIP affects all breeds, but we cannot spread our genetic studies across all breeds. We only need two breeds, which are inbred to a degree and have relatively small to moderate numbers in their registries. We have seen a number of FIP cases in Burmese and Birmans this year, especially in certain bloodlines. Burmese and Birman breeders seem to know a great deal about bloodlines and certain matings that result in FIP. Because Burmese breeders have cooperated so well on the head defect project, they know what is needed for another genetic study. Birmans do not have head defects, but Birman breeders appear equally involved and knowledgeable about the genetics of their breed. We are trying to get as many breeders of these cats as possible to come together and provide at least three generational pedigrees, with DNA samples (cheek swabs with Q-tips), of families known to produce affected kittens. These families are essential for determining the genetic basis for FIP susceptibility

We need FULL cooperation from breeders of these two breeds. We can code the information we receive – so confidentiality will be maintained. It's my hope this will reassure breeders to provide us with full data and pedigrees on FIP cases and related cats they have experienced in their catteries.

There are two types of studies we want to do. Whole genome linkage mapping requires at least three generations of cats with 30 or more members and with accurate identification of FIP affected and non-affected individuals. Similar information can be obtained by combining information from a number of smaller families, providing breed pedigrees are made available to determine degrees of relatedness among these families.

Whole genome association mapping involves two large groups of cats of the same breed – one group of cats that have had FIP, the second that have not had the disease. This can be two different catteries, one with problems and one without (but please be honest about your status, as false classification will doom this type of study). These two groups can also come from different bloodlines in the same cattery – breeders have strong intuition on which cats are problems and which are not. We need a total of 100 or more cats in each group (affected and non-affected) for association mapping.

Breeders, besides information from your cattery, talk to clients. We want information on siblings and parents and grandparents of FIP cats. Get DNA on everybody in your cattery and from relatives whenever possible. Now is the time to start to collect and bank DNA for when the feline genome is complete. It is also important for future research on other feline diseases.

We will also need fecal samples to collect viral RNA for studies on the origins of FECVs and FIPVs. We need to know if certain strains of FECV are more apt to mutate to FIPV, and if catteries having these strains are therefore more likely to suffer high FIP loses. It is extremely important that FIP is accurately diagnosed. We can help to review veterinary records to see if there is enough evidence to confirm the diagnosis. We can accept proper tissue samples taken at necropsy or biopsy

from your veterinarians and test them for FIP. We can also do necropsies at Davis when possible and necessary.

How much money is needed for this research at Davis?

All money given to Davis will go right into FIP research. Some of this research will be clinical in nature, and some bench top. We need $50,000 – 75,000 a year just for a single technician or graduate student, and the more such people we can engage in research the faster we will reach our goals. The genetic testing will be expensive - the DNA chip arrays will cost $400 or more each just to purchase (once they are developed by commercial companies), read, and analyze. These are certainly daunting figures, but doable if everyone admits to their problems and work together. As demonstrated by our predecessor, SOCK it to leukemia, a lot of money can be raised by ordinary people (cat breeders, cat owners, cat lovers), and a lot can be accomplished with that money if it is concentrated in the hands of knowledgeable and capable researchers.

What about other universities and institutes that are studying FIP?

Though we are trying to focus on U.C. Davis for greater impact, the scientific community is very collaborative and pedigree/disease information and DNA samples will be useful for meaningful collaborations. We are aware that others are interested in this same approach. Our goal is to solve FIP and in the end it really does not matter how it is accomplished or who does it. Scientific competition is always good. We are also aware that other groups may be raising money to study FIP, and this is also respected and accepted. A world full of researchers have studied this disease for over 40 years, and although we know a lot more about it, we still do not have effective ways to totally prevent or cure this disease. Hopefully, this worldwide research effort will finally bear the needed fruit. We can only do the best as our part.

My heartfelt gratitude to Dr. Pedersen for answering my many questions about the disease – questions I know are shared by others. I believe the comprehensive information he has provided will help breeders, rescue and shelter workers, veterinarians, and cat lovers in general to gain a better understanding of FIP and ways to prevent it in our cats. I also hope it will encourage Burmese and Birman breeders and fanciers to participate in this important research.

Nancy L. Reeves, Editor

SOCK it to FIP!

Save Our Cats and Kittens (S.O.C.K.) was a nonprofit organization founded by cat lovers from the San Francisco Bay Area in 1974 to aid in much needed feline leukemia virus (FeLV) research at the University of California at Davis. Other organizations have copied this name, but there is only one SOCK.

FeLV infection was the cause of death of up to one-third of all sick cats during the 1960s and 1970s. The original goal of "socking it to leukemia" was ultimately met, with the development of simple and rapid diagnostic tests to detect carrier cats as well as effective vaccines. Testing and vaccination drove FeLV from our catteries and households and returned it back to nature, where it had existed for thousands of years as an infection of limited importance.

After twenty-four years of dedicated service and the raising of hundreds of thousands of dollars, SOCK passed its torch to the Center for Companion Animal Health (CCAH) at U. C. Davis. SOCK is now being reactivated as a privately run and supported adjunct of the CCAH. Its mission is simple - raise money for research into the cause, diagnosis, prevention, and cure of feline infectious peritonitis (FIP).

SOCK FIP is an organization run by a coalition of cat lovers, pet owners, rescue groups and breeders who have had personal experiences with this terrible disease and whose support is directed to the U. C. Davis CCAH and its experienced group of FIP researchers. Research on FIP at Davis will be supported by SOCK, but data and findings will be shared with other research institutions around the world. SOCK will have a website soon, but in the meantime if you would like additional information about SOCK, please contact:

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The Ins and Outs of Pedigree Analysis, Genetic Diversity, and Genetic Disease Control

by

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As breeders, you engage in genetic "experiments" each time you plan a mating. The type of mating selected should coincide with your goals. Outbreeding brings together two animals less related than the average for the breed. This promotes more heterozygosity, and usually more variation in a litter. A reason to outbreed would be to bring in new genes or traits that your breeding stock does not possess. Outbreeding can also mask the expression of recessive genes, and allow their propagation in the carrier state.

Linebreeding attempts to concentrate the genes of a specific ancestor or ancestors through their appearance multiple times in a pedigree. The ancestor should appear behind more than one offspring in the sire and dam's pedigree. Otherwise you are only linebreeding on the single offspring. A linebreeding may produce an offspring with magnificent qualities. However, if those qualities are not present in any of the ancestors that have been linebred on, the individual may have a wonderful show career, but it may not breed true. Careful selection of mates is important, but careful selection of offspring from the resultant litter is also important to fulfill your genetic goals. Without this, you are reducing your chances of concentrating the genes of the linebred ancestor.

Inbreeding significantly increases homozygosity, and therefore uniformity in litters. Inbreeding can cause the expression of both beneficial and detrimental recessive genes through pairing up. Inbreeding cannot change, or create undesirable genes. It only exposes them through homozygosity. Inbreeding can also exacerbate a tendency toward disorders controlled by multiple genes, such as hip dysplasia and congenital heart anomalies. Unless you have prior knowledge of what milder linebreeding on the common ancestors has produced, inbreeding may expose the offspring (and buyers) to extraordinary risk of genetic defects. Research has shown that inbreeding depression, or diminished health and viability through inbreeding, is directly related to the amount of detrimental recessive genes present. Some lines can thrive with inbreeding, and some cannot. Increased homozygosity through inbreeding can also decrease the diversity of major histocompatability (MHC) genes that affect the immune system, and play a role in auto-immune disorders.

The inbreeding coefficient is an estimate of the percentage of all the variable gene pairs that are homozygous due to inheritance from common ancestors. It is also the average chance that any single gene pair is homozygous due to inheritance from a common ancestor. In order to determine whether a particular mating is an outbreeding or inbreeding relative to your breed, you must determine the breed's average inbreeding coefficient. The average inbreeding coefficient of a breed will vary depending on the breed's popularity or the age of its breeding population.

For the calculated inbreeding coefficient of a pedigree to be accurate, it must be based on several generations. Inbreeding in the fifth and later generations (background inbreeding) often has a profound effect on the genetic makeup of the offspring represented by the pedigree. In pedigree studies, the difference in inbreeding coefficients based on four versus eight-generation pedigrees varied immensely. A four-generation pedigree containing 28 unique ancestors for 30 positions in the pedigree could generate a low inbreeding coefficient, while eight generations of the same pedigree, which contained 212 unique ancestors out of 510 possible positions, had a considerably higher

GC Hullabaloo Olympia Ducatkus CNW

inbreeding coefficient. What seemed like an outbred mix of genes in a couple of generations appeared as a linebred concentration of genes from influential ancestors in extended generations.

Many breeders plan matings solely on the appearance of an animal and not on its pedigree or the relatedness of the prospective parents. This is called assortative mating. Breeders use positive assortative matings (like-to-like) to solidify traits, and negative assortative matings (like-to-unlike) when they wish to correct traits. Some individuals may share desirable characteristics, but they inherit them differently. This is especially true of polygenic traits, such as ear set, bite or length of forearm. Breeding two phenotypically similar but genotypically unrelated individuals together would not necessarily reproduce these traits. Conversely, each individual with the same pedigree will not necessarily look or breed alike. Therefore, matings should be based on a combination of appearance and ancestry.

Some breeds and breeders have concerns about genetic diversity. Molecular genetic research in many of these breeds shows that there is more diversity (heterozygosity) present than breeders realize. Some breed clubs advocate codes of ethics that discourage linebreeding or inbreeding, as an attempt to increase breed diversity. The types of matings utilized do not cause the loss of genes from a breed gene pool. It occurs through selection; the use and non-use of offspring. Regardless of the popularity of the breed, if everyone is breeding to a single stud, (the popular sire syndrome) the gene pool will drift in that individual's direction and there will be a loss of genetic diversity. The frequency of his genes will increase, possibly fixing breed related genetic disease through the founder's effect. If some breeders linebreed to certain individuals that they favor, and others linebreed to other individuals that they favor, then breed-wide genetic diversity is maintained. Animals who are poor examples of the breed should not be bred simply to maintain diversity. Related individuals with desirable qualities will maintain diversity, and improve the breed.

If you linebreed and are not happy with what you have produced, breeding to a less related line immediately creates an outbred line and brings in new traits. Repeated outbreeding to attempt to dilute detrimental recessive genes is not a desirable method of control. Recessive genes cannot be diluted; they are either present or not. If an individual is a known carrier or a high carrier risk through pedigree analysis, it can be retired from breeding, and replaced with one or two quality offspring. Those offspring can be bred, and replaced with quality offspring of their own, with the hope of losing the defective gene.

Trying to develop your breeding program scientifically can be an arduous, but rewarding, endeavor. By taking the time to understand the types of breeding schemes available, you can concentrate on your goals towards producing a healthy and worthy representative of your breed.

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Pedigree Analysis
by Carmen L. Battaglia, PhD

Carmen L Battaglia is an AKC judge, researcher and writer. He holds a Ph.D. and Masters Degree from Florida State University.

The term pedigree is an old word which is derived from the French "pie de grue", meaning crane's foot. The drawn pedigree was first used in the breeding of cattle and other domestic livestock. Now, after more than six centuries, the tradition of using it as a primary breeding tool continues. Over time breeders learned the important uses of a pedigree as a means of identifying carriers along with the strengths and weaknesses of each ancestor. Thus, when the frequency of a trait or disease occurred among the ancestors, it should serve as a signal that something is likely to be heritable.

TRADITIONAL PEDIGREE

The Traditional Pedigree is the most popular of the pedigrees used by breeders. Unfortunately, as a breeding tool the Traditional Pedigree has many shortcomings. The most notable one is the importance it places on memory and knowing the names and titles of the ancestors none of which is heritable. The custom has been for breeders to recognize and associate names and titles with what could be remembered about the traits and characteristics of each ancestor. This approach lacked reliability and it did not capture the information needed to plan a breeding. Another problem associated with the Traditional Pedigree occurred when litters were evaluated. When something worked, credit was given to the pedigree and the breeder. When it didn't, there was no record or source of information to be reviewed. This made it ineffective as a breeding tool. Perhaps its major criticism was that it did not lend itself to collecting the right kinds of information in sufficient detail to be useful to plan a breeding. A review of how most Traditional Pedigrees are used shows that scribbled notes around the edges and in the margins typically serve as the record system. Words such as "beautiful coat", "wonderful type", a title or the name of a famous offspring becomes the information a breed has to use. This approach fails to collect what is relevant or specific to making improvements. In short, breeders had no way to learn from their mistakes.

Two other pedigrees were developed to compensate for the limitations of the Traditional Pedigree. The first was called the Stick Dog Color Chart Pedigree. Its focus is on the traits of conformation. The other is called the Symbols Pedigree. It is used to track and analyze health, performance and other special traits of interest.

FIGURE 1. Traditional Pedigree

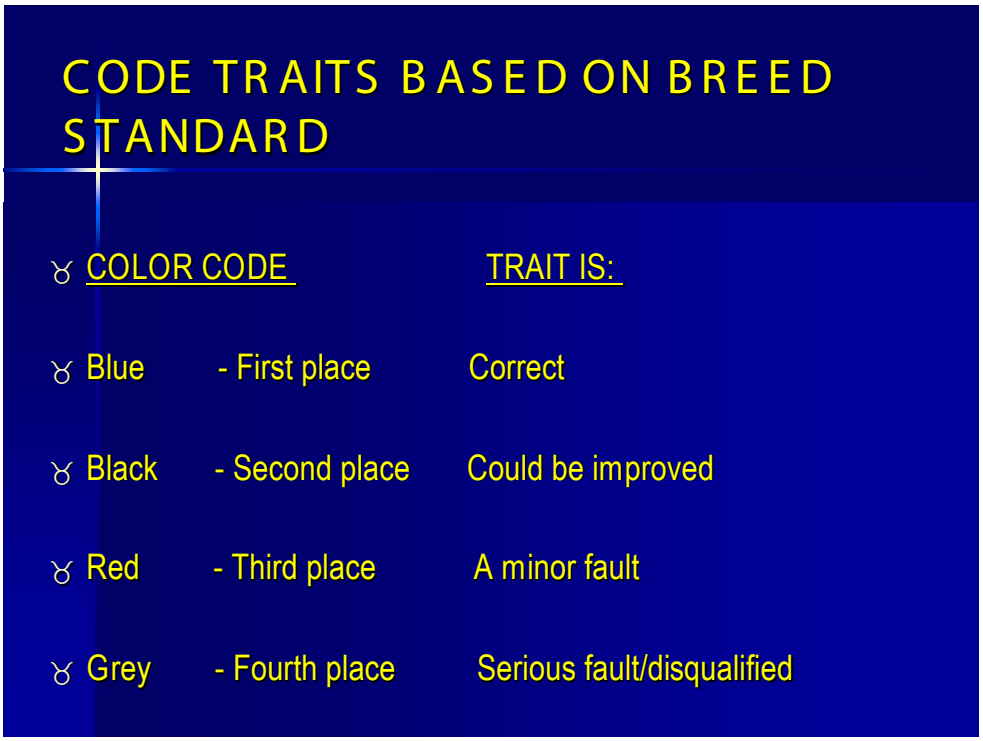
Notice that the Traditional pedigree in Figure 1 is easy to read, but it does not display the kinds of information needed to make decisions. For example, no information is collected about the carriers, normal or affected ancestors. It is not clear which ancestor(s) has the desirable and undesirable traits. The Traditional pedigree, as a record, forces breeders to rely on titles, certificates and winning records, all of which must be remembered.

STICK DOG COLOR CHART PEDIGREE

The Stick Dog Color Chart Pedigree was originally developed for research and computer analysis. Later, it was adapted to meet the needs of breeders. The Stick Dog Color Pedigree allows breeders to see the strengths and weaknesses of each ancestor based on the breed standard. The logic underlying this pedigree is that each ancestor is represented as a stick figure of coded information. Rather than a name and or title, each ancestor is drawn as a stick figure with seven structural parts: ears, head, neck, front, back, rear and tail. Each part is coded for its quality using four mutually exclusive colors. Each color is used to signify the rank or quality of the trait based on the breed Standard.

The Stick Dog Color Chart Pedigree helps breeders to identify and rank the traits of conformation of each ancestor using the seven key traits of conformation. Each trait is assigned a quality (rank) using a color-code. Notes are added to each stick figure to supplement and clarify the color codes.

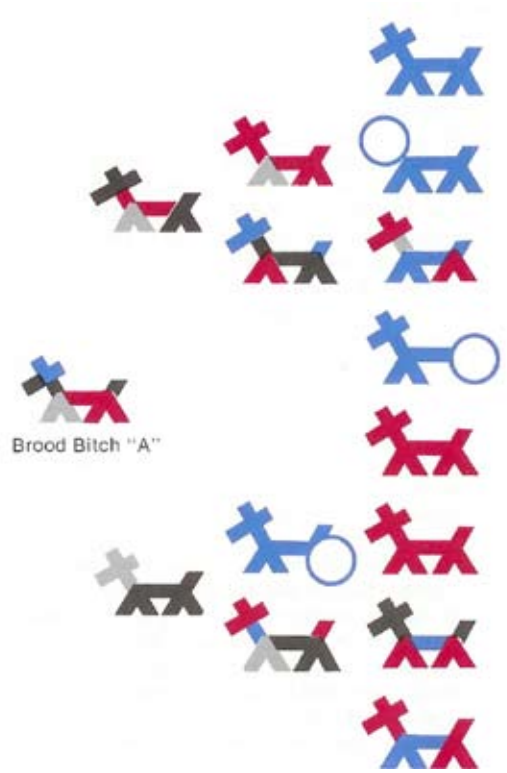
Figure 2. Code Traits Based on Breed Standards



The rules used to code the quality of a trait, or the lack thereof, is straight forward. When a trait is coded with a first place color (blue), it is viewed to be correct or ideal based on the breed standard. For example, if the ears on a sire were coded blue and those on the dam were coded black, the breeder would know that the sire's ears were correct but the ears on the dam were not correct and lacking in some way. Thus, the color-coding of each ancestor identifies their qualities along with their specific strengths and weaknesses. The color codes also show if there are trends or problems and whether they are on the sire or dams side of the pedigree.

Notice that brood bitch "A" has a fourth place front as does her father, grandfather, and her grandmother on her mother's side. Thus, in the first two generations, three out of six ancestors, or 50% of her ancestors, all have the same fourth place front. This suggests that she inherits her faulty front legitimately from her ancestors. It should also be noticed that poor fronts occur on both sides of her pedigree. This is useful information when searching for the right stud dog and traits he is expected to improve.

FIGURE 3. Stick Dog Pedigree



SEARCHING THE GENOTYPES

Researchers and breeders often use the term phenotype and genotype. Phenotype refers to the characteristics that can be seen, meaning their external appearance. Hence, a dog that is observed to be black (phenotype) may or may not produce only black puppies. It could have a genetic make-up (genotype) that includes the genes for other colors. Since genotypes cannot be seen directly, indirect methods must be used to learn about them. Indirect methods are not estimates or guessing games. Instead, they require the collection of detailed information about each ancestor and each of their littermates, usually for three generations. Those who do not collect and code information about the ancestors and their littermates usually rely on "type" breeding. This means they select sires and dams based on their appearance rather than on the traits observed in their offspring or the relationship that exists between them. Many times "type" breeding simply means breeding the winners to the winners. In practice, these breedings fail to take advantage of what the science of genetics has taught us about inheritance. Studying a pedigree for its genotypes means focusing attention on the occurrence of traits found among the ancestors and their littermates. While this approach requires more time, it is far superior to the Traditional Pedigree, which relies on learning names and titles.

SYMBOLS PEDIGREE

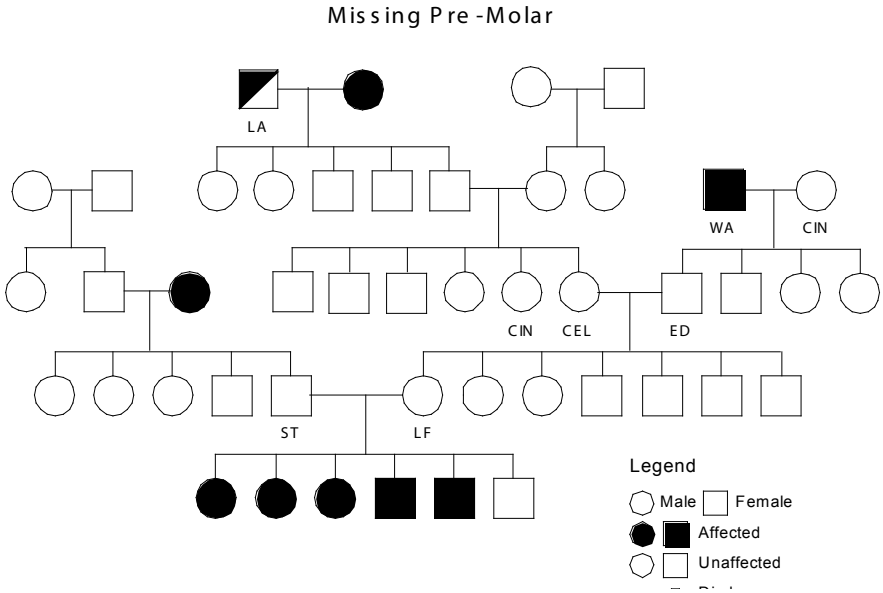
Breeders interested in health, temperament or special traits need a third kind of pedigree. One that is able to capture and display the strengths and weaknesses of each ancestor and all of their littermates. Breeders need a way to identify the carriers of certain undesirable health problems or some special trait of interest on either the sire or dam's side of the pedigree. Knowing where and how often these problems occur increases the probability that the carriers can be controlled or eliminated.

What every breeder wants is an inside look at the genes carried by the ancestors. Since no single method can look directly into the genotype, breeders have to rely on the information they are able to collect. The best pedigree for this purpose is called the Symbols Pedigree. It focuses on the breadth of a pedigree, meaning the littermates. The Symbols Pedigree relies on the logic that a pedigree can be understood by learning about the traits and characteristics observed among the littermates of each ancestor. It is especially effective for making improvements in the core elements: health, performance, temperament and other specific traits of interest.

The Symbols Pedigree gets its name because symbols rather than names are used to identify each ancestor. The inclusion of littermates further distinguishes this pedigree from the others. Its great advantage is that it produces a record of information that can be used to make improvements and eliminate problems.

The Symbols Pedigree is a powerful tool because of the amount of information that can be coded and quickly recognized. Squares are used to represent the males and circles to represent the females. The littermates for each ancestor are also represented as either a circle or a square. As information is collected about each individual it is coded using designated colors that represent specific traits or diseases. Because breeders are interested in many traits and diseases they will use several colors to code this pedigree. Key words and phrases are also added to clarify and further explain the characteristics, conditions, test results etc. for each ancestor. The repetition of a color, key word or phrase usually signals that a genetic trend or pattern may be present.

FIGURE 4. Symbols Pedigree



Notice in Figure 4 that the sire of the litter had three sisters and one brother and that the dam had four brothers and two sisters. This pedigree shows a litter of six pups (3-3). Five of these six pups had missing teeth.

A comparison between what the Traditional Pedigree and the Symbols Pedigree and Stick Dog Color Chart Pedigree should be convincing evidence that pedigree analysis can be improved by using these new breeding tools.

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Partnering with your Veterinarian

by

Lynette Massow with Dr. Jeremy Grossbard

Are you thinking of changing veterinarians, establishing yourself with a new vet, or are you just getting started finding a vet? Whether you are a breeder or a pet owner, here are a few suggestions relayed to me by my dear vet, Dr. Jeremy Grossbard of Companion pet Clinic in Apache Junction, Arizona.

If you need an effective way to find a new vet, other than letting "your fingers do the walking through the yellow pages," your best approach may be word of mouth. Talk to other breeders in your area or communicate with other pet owners. Ask about their experiences, both good and bad. Type your prospective vet's name into Google or Yahoo and see where it takes you. You may be very surprised what you'll find, but keep an open mind.

Another way Dr. Jeremy suggests to begin your search is by contacting your state veterinary society, the American Society of Feline Practitioners (AAFP - aafponline.org), or the American Veterinarian Medical Association (AVMA -- avma.org). One can also contact the vet's state licensing board to check for complaints. In all fairness keep in mind that there are two sides to every story, and a fee dispute is NOT a reason to shrug off a potentially great vet.

Once you have settled on one or two prospective vets, call and arrange for an interview as if it were an appointment. Choose a time with him or her that respects their work and surgery schedule, so that they have time to give you a tour and answer your questions. And use the time wisely to discuss all your concerns.

Dr. Jeremy believes that the biggest problem between breeders and vets is a lack of mutual trust. He feels sometimes breeders think vets don't have sufficient knowledge of their particular breed, and some vets think that breeders have less knowledge than they actually do. Use this interview to establish a foundation of mutual trust. According to Dr. Jeremy, a good vet should trust that the breeder has useful knowledge to share, and at the same time the breeder should trust that their vet is trained and knowledgeable in veterinary medicine, regardless of the breed.

It is obvious that Dr. Jeremy enjoys working with breeders, but he feels a big problem in working with some breeders is they try treating their own animals, and can let illnesses go too long before seeking a vet's advice and help. However, Dr. Jeremy's greatest concern with breeders and pet owners is non-compliance with treatments that have been agreed upon and prescribed. Another issue is financial, particularly when he has treated an animal and the outcome wasn't what the pet owner wanted. Even if the outcome is a negative one the vet still has to collect a fee to pay his or her expenses, such as utilities, employees, medicines, and other vets' services, particularly if he owns the practice.

Personally, I have felt in several instances over the years that some vets I've interviewed had an instant dislike of cat breeders. I asked Dr. Jeremy about this, and he disagreed. In his opinion this is not the case the majority of the time. He feels those kinds of problems can arise as a result of not being clear in the initial interview, or, once a relationship has been established, it can result from a failure of communication. When you are working with your vet don't be afraid to be honest about bad as well as good experiences. Discuss all options and recommendations relevant to what is going on. Explain why you feel the way you do.

If you are feeling comfortable in your interview with a new vet, you should also consider discussing subjects such as what he or she prefers for vaccination protocols, the use of antibiotics or overuse of them, holistic treatments either separately or in combination with medical treatment, and what he or she thinks of raw diets versus commercial foods.

A very important topic that comes up frequently between vets, breeders, and pet owners is anesthesia for their cats. From years of giving anesthesia I have learned that it isn't the anesthetic that is the problem, but the monitoring of the feline under anesthesia. All anesthetics present risks, and no anesthetic is any better than the person administering it. It is fair to inquire how he or she does their inductions, what agents are used, who monitors the animal, what equipment is used to monitor them, such as EKG, oxygen saturation monitoring, in some cases blood pressure monitoring, and most of all WHO watches them while the surgeon is operating. Last but not least, who watches them and for how long upon emergence. Dr. Jeremy feels that the most critical safety net for animals under anesthesia is a trained pair of eyes at the head of the table to watch the animal and the monitors while the surgery is being performed.

I discussed with Dr. Jeremy those pet owners or breeders who may also wish to explore the use of naturopathic medicine, either alone or in combination

with traditional veterinary medicine. Most vets in the United States are trained in Western medicine, but slowly alternative medicine is working its way into vet practices. Dr. Jeremy feels that a good vet will be willing to explore with you holistic alternatives, and not brush them off.

One important thing that Dr. Jeremy recommended was to take note on your tour of the condition of a new vet's facility. Remember that a vet clinic can get busy and be untidy. But a clinic that seems dirty and odorous MAY reflect their quality of care. Also, have a back up plan and ask your vet where he or she sends emergency cases when his or her clinic isn't open.

You may find that you in turn are also being interviewed. Turnabout is fair play. I asked Dr. Jeremy what his idea of a good breeder is. He likes a breeder that doesn't overbreed their animals, but retires females at around 4 years of age having had 3 or 4 liters, and the males at around 6 years old. He prefers a breeder that spays and neuters their kittens and retirees, and shows compassion and caring for their animals regardless of their earning abilities. Breeders who aren't afraid to suggest options for treatments based on their personal experiences, and also to discuss what hasn't worked and when they have been wrong, are great assets to their vets.

I asked Dr. Jeremy if he had one word that would help vets, breeders and pet owners to establish a great working relationship -- and that magic word is communication.

Lynette Massow
AZ Mews Cattery

AZ Mews Vandy
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UBCF Member Survey

Tips for Good Health for our Burmese

Thank you to the 22 members who responded to the survey. We received a wealth of information, and it is impossible to include it all in this issue. Members also had additional questions they wanted posed to the membership – so we'll do more surveys in the future!

DRY FOODS

What kind of dry food do you use?

Only two members used one brand of food exclusively, the rest use multiple brands. The most popular food used by nearly half of respondents is **Innova Evo**. But the most popular brand is **Royal Canin** which has a variety of formulas. Many members use **RC Baby Cat** and **RC Kitten**, and a couple use **RC Persian**. The third most popular food is **Science Diet**, followed by **Avoderm** and **Wellness**. Other brands with more than one respondent are **Bench and Field's, Costco, Iams, Medi-Cal, Natural Choice, ProPlan, Eukanuba,** and **Purina One**. Various other individual brands mentioned are Advance, California Natural, Fancy Feast Gourmet, Good Life, Natural Balance, NutraChoice, Nutro, Orijin, Perfect Fit, Prism, Solid Gold, and Walmart Kitten.

Do you vary the dry foods you use, if so how often?

Eight members do not vary the dry food(s) they use, and four not usually or only periodically. One changes once a week, one every three months, one every six months. Several change to kitten formulas when kittens are on the ground or when kittens are fussy. Two use a consistent base brand but add different brands or flavors for variety. Several put different brands in separate bowls to let the cats choose or to see which brand is preferred.

Do you mix two or more dry foods together?

Thirteen respondents do not mix food, one sometimes, eight regularly mix foods.

Do you add any supplements to your dry food?

Twelve members do not add any supplements to dry food. Six add **L-Lysine powder** on a regular basis. Other additives include **powdered milk replacer** when

starting kittens on solids, **acidophilus** for kittens, **NuVet** and **Oxyfresh** vitamin supplements, and one member recommended **Pancrea Powder Plus** for kittens that are failing to thrive.

CANNED FOOD AND RAW MEAT

Do you use canned food -- if so, what brands?

The winner in this category – **Fancy Feast**, used by nine members who responded. Next most popular canned brands are **Medi-Cal** (several formulas), **Evo, Innova, Avoderm, ProPlan, Nutro,** and **Friskies**. Other brands used by more than one member include **Prescription Diet A/D** (when needed), **Science Diet Kitten, Wellness, Trader Joe's,** and **Whiskas**. Other individual brands include Authority, California Natural, Eagle Pack Holistic, Edel Cat (Germany), Evanger's Holistic, Evolve, Iams, Pet Gold, Sheba, Solid Gold Tuna, Sophisticat, Tiki Cat, and Weruva. One member uses as needed for kittens baby meats with rice cereal mixed with KMR.

How often do you give canned food?

Half of respondents give canned food twice a day. Other members give wet food once a day, usually every other day but occasionally once a day, one intermittently, and one only when medically necessary. Two members feed three times a day. The remaining have varied feeding schedules – once a day for adults, or none for adults or spays and neuters, but kittens and pregnant or nursing moms are fed between 2 and 4 times a day.

How much canned food per cat?

Of those who feed canned food, eight let cats and kittens free feed and eat as much as they want. Those members who specified, amounts ranged from ¼ can/packet, 3 ounces or 200g to ½ can/packet or 6 ounces per cat per feeding.

Do you feed raw meat?

Not all members answered this question as it was added on after the survey was first sent out. Of those who did answer, six do not feed raw meat, four of those because the cats wouldn't eat it. Others who do feed raw meat recommend **Nature's Variety**, **Aunt Jeni's** home made, **Bravo** beef organs with calcium, and **fresh meats** including ground sirloin and rabbit. Additives to raw meat include **calcium** and **Platinum Plus**. Others give cats and kittens, and pregnant or nursing moms in particular, cooked meat drippings, cheddar cheese, and Live-a-Littles freeze dried chicken pieces.

SUPPLEMENTS

Do you give any other supplements such as vitamins, if so what kind?

Eight members mentioned they do not give any supplements at all or not routinely. Those that do (frequency was not indicated), the most popular are **Lixotinic** or **Pet-Tinic** and **L-Lysine**. Second most popular are some kind of milk replacer, such as **KMR** and **Just Born** or **goat's milk**. Other supplements used include 8-in-1 Excel brewer's yeast, acidophilus, B vitamins, Cosequin for older cats, KittyBloom Wate, Linatone Oil, Missing Link Platinum Performance, Oxyfresh Primorye and Oxyfresh Pet Antioxidants, raspberry liquid for pregnant queens, Spirulena, Vetamino, and Purina FortiFlora.

How often do you give supplements?

Of those giving supplements, seven give them daily or at least once daily. Three members not often, once a week, or two to three times weekly for kittens. One mentioned giving as long as necessary for kittens and sometimes sending a mixture with kittens to their new homes to help with the transition.

WATER

Do you provide tap water or bottled water?

Six members use bottled water, six use tap water, four tap water from wells, four filtered tap water, and of the remaining two one uses both tap and bottled water, and one tap water with Oxyfresh pet oral hygiene added.

Do you use anything to boost immune systems?

Eleven members do not use anything to boost immune systems. Five use **L-Lysine (Viralys),** although one member commented that L-Lysine does not actually boost the immune system but rather interferes with viral replication. Other products used by members include **Equi-Stim** from Revival, **Spirulina**, **yogurt**, **Oxyfresh Primorye** and **Oxyfresh Antioxidants**.

BATHING AND CAT SHOWS

Do you wash your cats regularly (outside of shows)? If so, how often?

Ten members do not wash cats regularly. Eight wash as needed, either if the cat got into something, to reduce excess hair, or to cool the cats off in summer. Other members wash periodically -- once a month, every couple of months, occasionally with waterless shampoo, twice a

year. And one member washes kittens a couple of times before they go to new homes.

What shampoo or other bath/conditioner products do you use?

Those who wash their cats use a variety or products. Most popular, **Chris Christensen** products. Next, **BioGroom**, **Davis products**, and **Johnson Baby Shampoo.** Other products used include Allergroom, Aveda conditioner, F2R2 (pink bottle), Furminator de-shedding shampoo, Goop for stud tails, Hylight shampoo and conditioner, Jerob, Orange Cleansing shampoo, Oxyfresh Pet shampoo, Pure Pet, Ronalle, human shampoo, and vinegar.

Do you brush your cat's coats regularly? If so, with what tools?

Only a couple members don't brush their cats regularly. The most popular tools used by more than half the members who do brush regularly are **a combination of a rubber brush and a fine tooth flea comb**. Several others used only the flea or other fine toothed comb, or rubber, bristle or plastic-tipped slicker brushes. Other tools recommended were the **Furminator** and **Zoom Groom**.

How often do you wash cat bedding?

Sixteen respondents wash bedding at least once a week, more often for kittens. Others wash every couple of weeks to once a month. One washes bedding daily for newborns, and another uses cotton diapers on top of bedding, changed daily.

What products do you use for washing bedding/rugs/ cage covers?

Nearly all members use standard laundry detergent – several use **Arm and Hammer** detergent, and **Oxyclean** and **Oxyfresh** laundry detergents are also recommended, as is **Trader Joe's Detergent with Petzyme**. More than half also use other products as laundry additives or in a pre-wash: most use **bleach** or color safe bleach, other products used include **baking powder**, **borax**, and **Odoban**. Other products added to the wash are Shaklee Basic G as a disinfectant, Anti Icky Poo for urine odor, Nature's Miracle, and Health Guard Laundry Additive as a disinfectant/anti-fungal in the rinse cycle.

Do you wash your cats when you bring them home from shows?

Only five respondents wash cats after shows, one of those ASAP. Two don't show, two don't usually wash, and the rest do not wash cats after shows.

What products do you use during or after shows for bathing or other disease transmission prevention?

Several mentioned using **hand sanitizer** before anyone at a show touches their cats. Products used to disinfect cages included **bleach**, **Shaklee Basic G**, and **A3 by Airkem**. Products used on cats include diluted **Listerine**, **hexachlorophine** spray, **Health Guard** spray and waterless shampoo, **Malaseb**, **Nolvasan**, and **Oxyfresh** shampoos. Two also mentioned treatment for fleas after a show.

CAT LITTER AND OUTSIDE EXPOSURE

What type of cat litter(s) do you use?

The majority of members use **Feline Pine** pellets, and several use other wood pellet products including Tact Cat Litter, wood stove pellets, or horse stall pellets. Three members use **Yesterday's News** (recycled newspaper) litter mixed in with Feline Pine, and one uses exclusivesly Buckerfield's Crown Animal Bedding which is also from recycled paper. Four members use **World's Best Cat litter**, and one uses non-medicated poultry grower corn crumble as a less expensive alternative. Two members use Nature's Miracle cat litter, and the rest use clay litters such as Costco Scoopable, Dr. Elsey's multi-cat, Johnny cat, Tidy Cat Scoop, and Publix white clay.

Do you use any kind of litter deodorizer in addition to/added to cat litter?

The vast marjority do not use litter deodorizer, five use or have used **baking soda** to cut down on odor. One uses Anti Icky Poo, and one Oxyfresh Pet Deodorizer. And one member reminds us that the secret to keeping litter fresh is to scoop, scoop, scoop!

Do your cats have exposure to the outside – indoor outdoor runs, for example?

The majority of members do not expose their cats to the outside except through screened windows or doors when weather conditions are good. Two have screened porches the cats can go into, and four have or are working on building indoor-outdoor runs, mostly for studs only. One has a garden their cats enjoy. Two members noted reasons why they don't expose cats to outdoors – including bears, cougars, raccoons, snakes, and poisonous frogs!

What other questions do you have for UBCF members? Please email your questions to burmapearl@mac.com

Next Issue & How to Join UBCF

Planned for the Next UBCF Newsletter:

RAISING BURMESE KITTENS

Possible articles may include:

- How kittens develop and helping them on their way
- Looking at the new vaccination protocols
- Perspective from a new Burmese breeder
- Educating clients and tips for client contracts
- Our favorite kitten photos
- And the 2007-2008 Show Season Reports with the editor's apologies for not being able to fit them into this issue!

The Mission of the United Burmese Cat Fanciers is:

- To create and develop interest and knowledge of the Burmese cat, and in the care, health and breeding thereof.
- To seek to establish markets for the breed.
- To cultivate acquaintanceship among members.
- To promote and advance in every way possible the interest of owners, breeders, and exhibitors of Burmese cats.
- To determine standards of the Burmese cat.
- To maintain, develop, and publish information concerning the breed.

If you would like more information about UBCF, or to become a member, please contact:

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