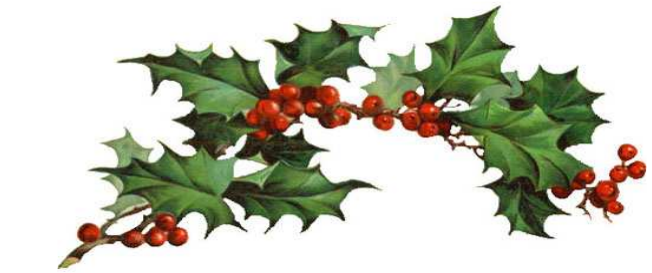
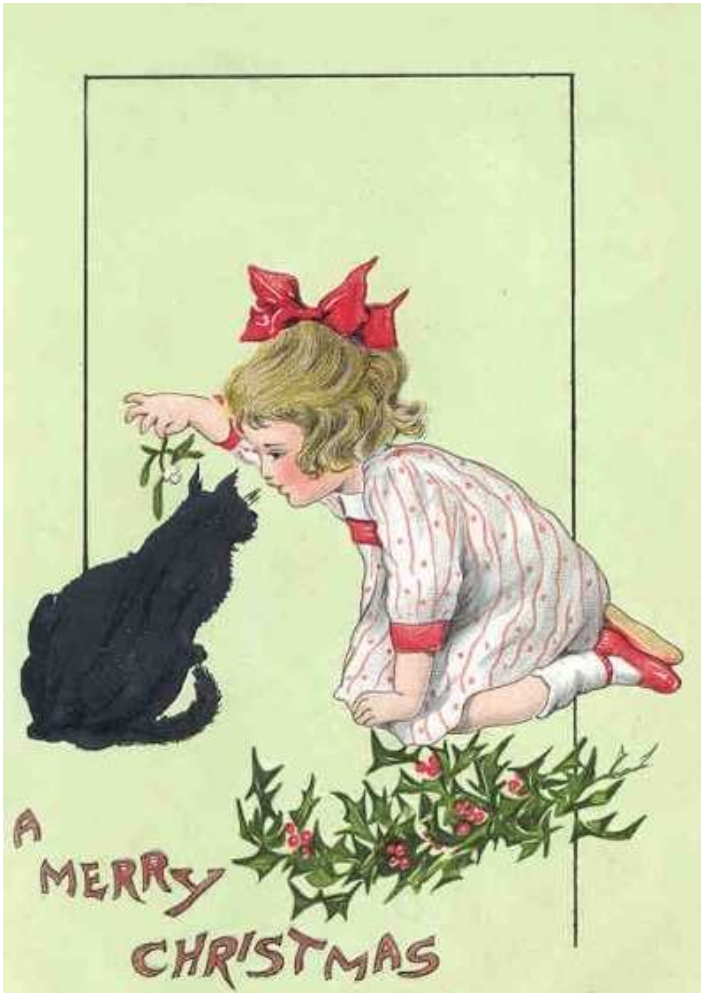
December 2008 Issue 111

GLOVE and GAUNTLET

Newsletter of the Birman Cat Club of Canberra Inc





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Letter from the President

Hi All,

I can't believe how quick the year has gone. I would like to thank the committee for their hard work and commitment for making our 2008 show another success. I have just been informed that Whiskas are happy to sponsor us for our shows in 2009. This is wonderful news. Thanks to Alison for sending out letters showcasing our shows and how successful they were in 2008.

Christmas will be here soon and then we will all be getting ready for the show season. I hope everybody had a successful show year and are looking at the babies they have at the moment, seeing if they may be show winners for 2009. The Birman Cat Club will look forward to seeing you all again in Canberra for our shows in May. I am sure it will be another great weekend.

We have some interesting judges coming for the Specialist Show and All Breeds Show in 2009. They are Judy Lewis, Sandra Perry, Rita Wiseman, Jane Vermulan, Vickie Shields and Adriana Kajon. I think Judy, Sandra and Rita will be doing the Specialist show and then all are doing the All Breeds Show. The schedule will be up on the website in the new year.

The show again will be held at the Burns Club with new caterers so I am looking forward to talking to the Functions Manager in 2009.

The club's new website is now complete, however there are a few things to correct and update (committee and show information). Please could you have a look at it and let me know if there are any errors or changes you think necessary.

I would like to wish everyone of you a very Merry Xmas and a safe New Year and that all your kittens find happy and loving homes.

Merry Xmas,

Carol

Letter from the Editor

Hi Everyone,

I will endeavour to be your editor for the next year and hope to keep you informed with updates of events, news of members and relevant information. To do that I also rely on you to send me letters, any snippets of news or information and articles you have found interesting or amusing. Please share at least your litter arrivals, show results and new titles. It will be very good to hear from interstate members too! I would also like to include a few photos of cats - of your Birmans or photos of other cats and kittens which are appealing (not difficult for a cat!) or funny. There will be a "Cover Cat", "Centrefold Cat", "Cute Cat", "Comical Cat" etc. Please let me know if there are other items, sections or topics you would like covered or would contribute to in the newsletter.

The article on " Sneezes, Snuffles and Sore Eyes", included in this newsletter, contains some practical advice as well as information about this widespread problem in catteries. The second article "Reds, Creams and Inbetweens" not only discusses the genetics of the "O" gene but also the effects of the silver and density modifier genes and resulting issues arising for judges and breeders.

Have a wonderful Christmas and all the very best for 2009,

Karen.

Members' News

ACT Cat of the Year Awards

Congratulations to members who have won "ACT Cat of the Year" awards for 2008. Carol Cootes won for her prefix "Bindura" and was runner up for Breeder of the Year. Tracy Stewart's Sarika Simply Red won Desexed Cat of the Year and Rosemary's Bindura Toberlone was runner up for Best Kitten. Well done!

New Litters

Several new litters have arrived so far this season. A special mention and congratulations go to Tracy Stewart whose girl, Sarika Belladonna, gave Tracy her first litter of Birman kittens. Good luck with these babies, Tracy, and hopefully the many litters to follow! Carol Cootes' Sarika Miss Tee had four babies in October - 2 males and 2 females, all seals and Bindura Embers had 5 babies in November - 3 males (1 seal, 2 red) and 2 seal tortie females. Carol's Kirashan Ciarra Tia (Townsville) has just had 4 babies.
Good luck to everyone with the potential show or breeding careers of these new arrivals.

Show News

Birman Cat Club Specialist and All Breeds Shows

The judges are being finalised and the schedules will be available early next year as downloads from the club's new website at www.actbirmanclub.com.au
Whiskas will be sponsoring both shows and possibly OzPet Litter will sponsor as well.

Canberra Royal Agricultural Show

Schedules for the show, to be held on 28/2 -1/3/09, are on the CCI website at www.cci.asn.au/shows . Entries close on 19/1/09.
Tracy Stewart is Cat Section Head (0408 461 348 Email - stewtr@westnet.com.au)
Carol Cootes is Show Secretary (0407 442 256 Email – bindura@netspace.net.au)

Cute Cats: Atu Goldrush and Atu Stardust are best of mates

Sneezes, Snuffles and Sore Eyes

Understanding Cat flu and what it means for my cats and cattery.

Dr Patrick Brogan and Dr Jason Stayt

Does your cat occasionally get sore inflamed eyes, do any of your cats sneeze regularly, have you bred litters of kittens that seem to have chronically mucky eyes? Then it is possible that some of your cats are cat flu carriers and indeed it may be endemic in your cattery. Don't be alarmed, it is important that one is informed and then once empowered with the information one can systemically put in place procedures to minimise re-infection and eventually eradicate it from your cattery.

What is Cat Flu?

The term 'Cat Flu' is lay terminology referring to, or lending description to the infectious upper respiratory tract pathogens that lead to symptoms that we humans would commonly associate with the flu. Symptoms include sneezing, coughing, runny eyes, nasal discharge, fever, malaise, oral and ocular ulceration. Interestingly the term 'cat flu' is actually merely a descriptive term for a syndrome that is caused by a number of infectious viral and bacterial organisms and has nothing in common with an influenza virus.

To break it down, put simply there are three main causes/components that come together to result in clinical cat flu – **Feline Herpes Virus, Feline Calici Virus** and a bacteria called ***Chlamydophila felis*** commonly referred to as **chlamydia.** Almost 50% of upper respiratory tract infections are caused by feline Herpes Virus, almost 50% by feline Calici Virus and a few by feline Chlamydia infection. Each of these pathogens will be addressed individually covering route of infection, symptoms, outcome, diagnosis, treatment and prevention.

Feline Herpes Virus - FHV

Route of infection

- Inhalation – from direct nose to nose contact or contact with virus infected particles like saliva, mucous left in dirty cages or litter trays etc.
- Feline Herpes Virus is not particularly resilient and will survive no longer than 24 hours outside the body. The drier an environment is, the shorter it will survive.
- Incubation period is anywhere from 1-7 days.

Symptoms

- Lethargy and fever
- Sneezing and nasal discharge initially clear and watery but becoming copious and pussey.
- Swelling of conjunctival membranes of eyes
- Ocular discharge and ulceration
- Anorexia due to oral ulceration and fever
- Symptoms can vary between cats and according to the strain of virus and vaccination history of the cat.
- Sticky eyes seen in litters of kittens at around 10 days of age is commonly FHV related, but can also be non-specific bacterial problems that resolve with symptomatic therapy.

Outcome

- Initial disease is usually self-limiting within 7-20 days with supportive care.

- Life-long infection results. The virus becomes dormant in the base of the spinal cord at the trigeminal ganglia. So systemically the cat recovers with no systemic evidence of inflammation, infection or presence of the virus.
- Later in the cat's life, episode of viral reactivation may occur, leading to viral shedding. At these times of viral shedding cats may show no → mild → marked clinical signs.
- In some cases infection may be so severe that lesions in the nasal bones and permanent damage to the nasal tissue may result. These cats are called 'chronic snufflers', but this is more commonly seen in kittens infected as young kittens.
- Pregnant queens may abort but this is more likely as a result of fever as there is no evidence that the virus crosses the placenta.

Diagnosis

- Clinical signs – but can be very similar to Calici virus and Chlamydial infection.
- Feline Respiratory Panel – Tests for Herpes, Calici and Chlamydia by PCR (polymerase chain reaction). Sample of tears or exudate. Offered by Vetpath, Gribbles, Idexx and Symbion Laboratories. But remember this test may miss the virus if shedding has stopped and the virus is currently dormant.

Treatment

- Supportive care – fluids and soft food for cats not eating, anti-inflammatory medication for fevers, anti-biotics if secondary infection is present.
- Anti-viral drugs can be used but seem to have little effect.
- Isolation of affected individuals especially in a colony situation.

Prevention

- **Vaccination** – Part of the F3 vaccination. Current vaccines reduce clinical disease by stimulating antibody response but **does not stop infection.** Current controversy in the veterinary profession as to how often cats should be vaccinated – we choose to vaccinate ALL our breeding girls prior to the breeding season as it offers us peace of mind that they are producing adequate anti-bodies in their milk to protect their kittens.
- We also vaccinate our kittens at 6 weeks with an F3 ONLY and again at 10 weeks.
- **Hygiene** – Feline herpes virus is highly labile and will survive less than 12 hours in a dry environment but up to 24hours in a moist environment. People can spread the virus on their clothing and hands. Inadequately cleaned food bowls are also a potential source of infection. Make sure stewards/judges at shows spray their hands with an appropriate virucidal spray. Don't allow cats from different catteries to have contact at shows, and don't allow the public to stick their fingers in your cats' cage after going from cage to cage! Remember each time you show and each time you take a girl out for a mating there is a risk you will be bringing home more than just ribbons or a belly full of kittens.
- Minimise stress – boarding cats, hospitalisation, pregnancy, introduction/departure of members of the household (human or otherwise) – these are times of stress that may lead to a re-emergence of the clinical signs and/or shedding of feline herpes virus.
- Quarantine new stock – All new stock MUST be isolated from your other cats for minimum of 1 week and preferably 2 weeks. Remember place them in an area where there is minimal risk of nose to nose contact or chance of sneezing spreading droplets in the air. If you isolate them in a cattery next to other cats place plastic sheeting between the cages. Always feed and clean them last and once finished change your shirt and wash your hands and if you're extra keen disinfect your shoes simply by wiping soles or spraying the shoes with disinfectant like **F10 or Virkon.**

Feline Calici Virus – FCV

Route of infection

- Transmission of the virus occurs via the respiratory, oral or conjunctival routes via aerosolised virus or infectious particles. Animal handlers most often carry the virus to susceptible cats.
- The virus is relatively stable and can survive 8-10 days in humid conditions.

Symptoms

- Fever and anorexia is commonly seen – fevers often greater than 40°C.
- Lesions are usually confined to the oral cavity, respiratory tract and eyes.
- Oral cavity – inflammation of gums and ulceration of tongue and back of mouth. Can cause complete anorexia and severe salivation from pain from ulcers.
- Respiratory tract – ulceration of nose pad and inside nose resulting in sneezing and nasal discharge.
- Eyes – conjunctivitis – but no ulceration of cornea, unlike cases of FHV.
- SOME cats have been reported to develop a 'limping syndrome' or 'ouchy grouchy cat' syndrome seen as fever, muscle soreness and shifting lameness associated with FCV following natural infection and in some recently vaccinated cats. This resolves without treatment.

Outcome

- Maternal antibodies usually lasts until 12 weeks so FCV usually causes disease in kittens older than 12 weeks (whereas FHV can cause disease from 8 weeks)
- A viraemia is present during the acute phase of the infection, and infected cats usually recover in 7-14 days in the absence of secondary bacterial infections.
- Infected cats that recover from the disease can carry and shed the virus for as long as two years.
- Chronically bad teeth, gingivitis and stomatitis can be associated with calici virus.
- Chronic rhinitis
- There are MANY different strains of FCV and they vary greatly in virulence. This results in a variation in the extent and severity of clinical signs with some strains resulting in pneumonia, viraemia and even localisation in the brain.

Diagnosis

- Based on clinical signs
- Feline respiratory Tract panel- PCR test from most commercial labs.

Treatment

- Supportive care, antibiotics to prevent secondary bacterial infection.

Prevention

- A vaccination for FCV as part of the F3 vaccination.
- Kittens usually vaccinated at 6-8 weeks, then 10-12 weeks, and at 16-18 weeks.
- A major problem is that FCV mutates regularly and there are many strains around. So selection of suitable strains to be included in the F3 vaccination as antibody produced from vaccination against some strains will not protect against ALL strains.
- Common sense hygiene as mentioned for FHV.

Feline Chlamydia – *Chlamydophila felis*

Route of infection

- Contact with fresh or dried infected bodily fluids
- Routes of entry are usually oral, respiratory or ocular pathways.
- Poor survival outside of the cat- less than 24 hours.

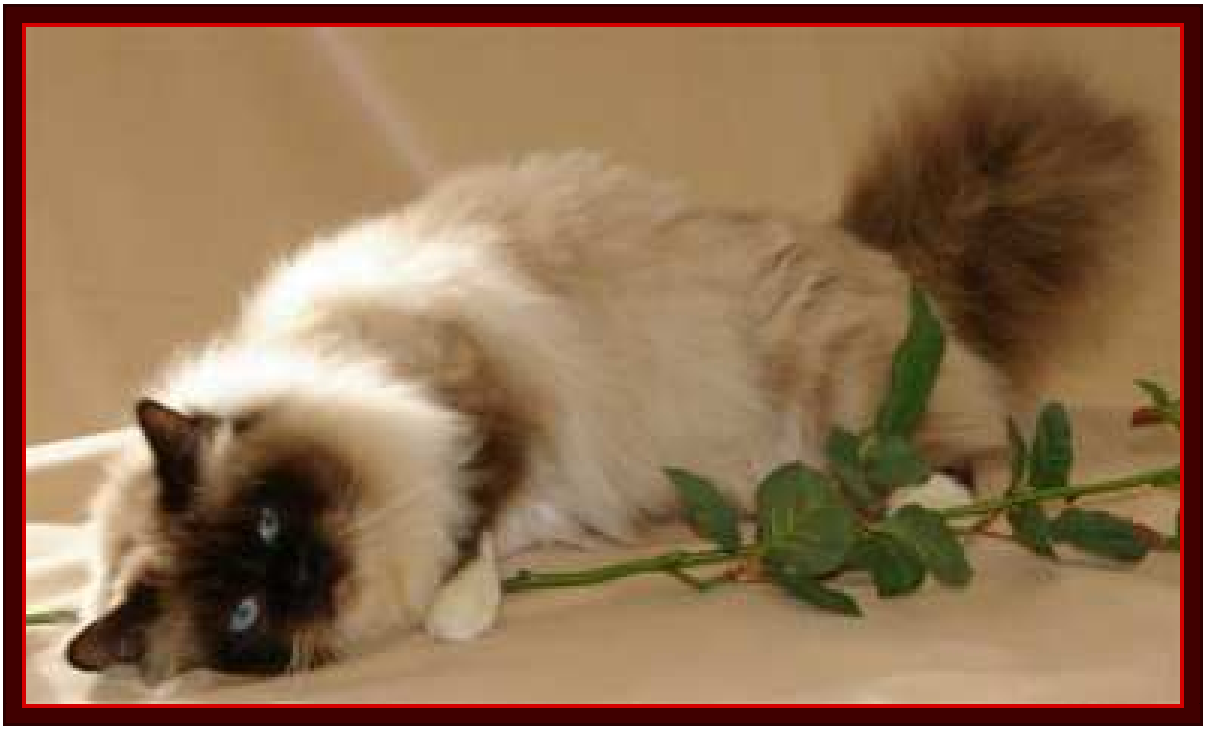
Symptoms

- Incubation period 4-14 days
- Primarily an infection of conjunctival membranes.
- Respiratory signs are rare and are usually indicate co-infection with a respiratory virus.
- Begins in one eye initially with a typical serous or purulent discharge.
- Eyes are often extremely reddened with swollen membranes and very watery.

Outcome

- Maternally derived protection seems to protect kittens for about 5-8 weeks of age.
- About 6% of cats carry this organism without apparent signs
- 50% of cases are in cats less than 6months old.
- Cats can continue to excrete Chlamydia for 8 months following infection and occasionally longer.
- Most cases are self-limiting and will eventually improve without treatment. It may play a role in infertility and abortion in cats as it can be isolated from the vagina of queens and aborted foetuses.

Ed's note: This article is reproduced here with the kind permission of Patrick and Jason, who are in the most fortunate position of being both veterinary surgeons and Birman breeders. This article and others like it, can be found on their website at ***http://www.zaeshinwabirmans.com/health.html***



Centrefold Cat: Bindura Phoenix Rising is a photographer's dream!

Reds, Creams and Inbetweens

Dr Kerry Fowler MSc PhD Grad Dip Ed

Published in NSW CFA Catching Up 5(3/4):5-6(2003)

Background

Current registration systems in most Australian cat councils work on the concept that two solid cats cannot produce a tabby kitten. In the main this policy is absolutely correct however in the case of the orange or 'O' gene cats ie creams, reds and the less vibrant reds known as 'gingers' and 'marmalades', sticking to this hard and fast rule often results in no end of controversy and confusion. Breeders are obliged to register red or cream tabby marked kittens from solid parents as red or cream selfs and in turn judges are required to assess them as such despite some of these red or cream kittens and cats having an abundance of clear tabby markings. Being a sex-linked gene, the 'O' gene coat colour issues extend to tortoiseshells, blue-creams and tricolours. This article identifies some of the misunderstanding surrounding breeding and judging 'O' gene cats including cameos and apricots, and draws on current genetic knowledge, registration policies and show standards from other councils to help resolve some of the re-occurring questions.

Genetic principles

In order to help put some of the debate and perplexity aside it is worth keeping in mind that there are four basic genetic rules that help us to understand the appearance of red or cream tabbies and solids. First, the tabby pattern gene is present in all cats including solid or self cats. Second, the tabby pattern is only revealed when the dominant agouti gene product or protein is present. Third, the recessive non-agouti gene product results in solid coloured cats eg black or blue cats despite the tabby gene being present in their genetic blueprint. Fourth, the non-agouti protein does not work on the dominant 'O' gene pigment colour often resulting in a non-agouti cat with defined tabby markings. These non-agouti red or cream tabbies are often difficult or impossible to distinguish from agouti tabbies and as such fall into an 'inbetween' tabby category that is technically unique to the 'O' gene cats. The issue is further compounded by the tendency of some genetic tabbies to develop a broad solid spine line with age regardless of coat colour.

Historically the agouti gene is named after the fur colour of the agouti mammal that is related to the guinea pig (Nature Com, 2002). The agouti gene protein product creates bands of black and yellow pigmentation on individual hairs. Collectively these coloured bands divulge the nature of the tabby pattern ie ticked, spotted, mackerel or classic. When a black cat has the agouti gene present we see a brown or black tabby†. However, when a black male cat has the dominant 'O' gene present we are likely to see a red tabby regardless of whether the agouti or non-agouti gene is there making it very hard to breed solid red or cream cats that are free of tabby markings.

Clear-coated reds and creams

The idea that breeding clear-coated non-agouti reds or creams is almost a genetic impossibility may be difficult to grasp because occasionally we see a red solid Persian or red Burmese that show little or no tabby markings. These magnificent cats are thought to have come about through selective breeding that has enriched for gene modifiers or polygenes that diffuse the tabby pattern. Of the tabby patterns, the ticked tabby is the most amenable to giving rise to a more solid-looking or clear-coated cat. The red Burmese appears as a red self due to being a selectively-bred, non-agouti, ticked tabby that has the added advantage of the Burmese colour lightening gene known as $c^b c^b$ (Vella et al, 1999). However the ticked tabby pattern is unavailable in the Persian gene pool (Brown, 1992). Nonetheless many

breeders have been able to diffuse agouti-induced classic or mackerel tabby markings to the point of not being able to easily see banding or tabby pattern by carefully breeding some of the non-red Persian coat colours (Vella et al, 1999). Fine examples of this phenomenon are seen in the chinchilla or golden coat colours (Brown, 1992). Besides being enriched for a special set of polygenes, these colours like the agouti or non-agouti red solid Persian have the advantage of coat length which helps dissipate the classic or mackerel tabby pattern (Vella et al, 1999).

Dense tortoiseshells

Being a sex-linked gene, the 'O' gene colour anomalies continue into the tortoiseshell range of coat colours. Some dense tortoiseshell standards call for an equal mixture of primary colour, red and cream (CCCA Show Standards, 2002). This wording is fundamentally wrong on two accounts. How can a dense coloured cat have cream coat colour when there is no pair of dilute genes in the cat's genetic composition. Understanding why the word cream features in the dense tortie standards is not hard. Obviously when looking at the dense tortoiseshell the 'O' gene coloured patches are often not uniform in colour distribution. Indeed red or lighter shades of red are often present and perhaps for the sake of convenience these paler shades of red have been called cream. Cream is the same pigment as red, only it is spaced further apart by the recessive dilute density gene. Using the word cream in dense tortie standards creates confusion because it is genetically incorrect. This could be simply avoided by using another description for tortoiseshells such as 'Presence of several shades of red are acceptable' (CFA Show Standards, 2000-2001) or 'The colours black and red (pale and dark shades)' (ACF Show Standards, 2002).

The second issue with the black or non-dilute tortoiseshell standards is the requirement of black torties to have an equal balance of black, red and cream. This wording is flawed, not just because of the word cream being there but because it is actually asking for 2/3 of the coat to be generated from the 'O' gene pigment product. A much better description would be for black torties to have an equal balance of black and shades of red coat colour suggesting a 50:50 ratio of black and shades of red.

Sex chromosomes, linkage and X chromosome inactivation

Understanding why the latter description is an improvement requires grappling with genetics and the inheritance of the sex chromosomes. It entails grasping a genetic theory that Dr Mary Lyon, a mouse geneticist from Oxford University was clever enough to work out. What is remarkable about Dr Lyon's discovery is that it applies to all female mammals and as such it is equally relevant to women. Like women and other female mammals, cats are warm blooded, give birth to live young and feed them with milk. Female cats and mammals have two X sex chromosomes whereas males have one X and one Y chromosome. In males and females one of the X chromosomes is always inherited from the mother. The other sex chromosome will be from the father. If the father contributes his Y chromosome then the offspring will be male. If the father contributes his X chromosome then the progeny will be female.

In the tortoiseshell cat the dominant 'O' gene coat colour gene resides on one of the X chromosomes thus enabling a pictorial display of Dr Lyon's discovery that in the female mammals only one X chromosome works in any one cell of the body (Lyon, 1999). Dr Lyon worked this out when thinking about why males do just fine with only one set of X chromosome genes and why females do not have problems with gene dosage by having two X chromosomes. So throughout the bones, organs, muscles, skin and coat of the female cat the genes located on only one X chromosome are producing gene products whereas the genes on the other X chromosome are inactivated early in development. When the X chromosome that has the dominant 'O' gene on it is active then red or phaeomelanin pigment

converts black pigment to red revealing red fur and pink skin pigment including pink paw pads and nose leather. On the other hand if the X chromosome that does not have an active 'O' gene is working then no phaeomelanin pigment is produced instead eumelanin or black pigment colour develops in the skin and coat. The net effect is a female cat with random patches of black and red coat colour.

Shell, shaded and tabby cameos

When considering the 'O' gene and Persians the 'inbetween' colour known as 'pink' is also worth discussing. This colour has been used to affectionately describe the coat colour of the shell cameo (Crawford and Crawford, 1993). The definition of cameo is basically a silver cat tipped or shaded with red. The shell variety is tipped with red whereas the shaded cameos have a mantle of red shading. The usage of the word shell in Australia has been strongly linked to cameo with many fanciers overlooking that any tipped silver cat falls into the shell category regardless of hair tip colour. For example the chinchilla being a black tipped silver cat is also technically a black shell. Likewise a shell cameo is a red chinchilla or red tipped, a shaded cameo is a red shaded and a smoke cameo is a red smoke (Gebhardt et al, 1979). Given this, our current CCCA Group 1 standard needs the umbrella headings of shell cameo and shaded cameo to be amended to shell and shaded, respectively, in order to encompass all the non-red and red colour varieties.

In addition to the shell and shaded cameo categories, there also exists the cameo tabby. These cats have come about by mating red tabbies to shaded silvers or smokes (Krzanowski, 1993). The cameo tabby has caused confusion due to the misconception that the shell cameo as well as other tipped varieties of Persians, has the dominant ticked tabby gene. Instead the Persian tabby gene pool is predominantly classic tabby followed by the gene for the mackerel tabby pattern (Vella et al, 1999) making it quite possible to breed a red silver or cameo tabby.

Apricot – a dominant density modifier gene or a misspelt recessive dilute density gene

The last 'inbetween' that comes to mind when dealing with the 'O' gene cats is the colour apricot which is cream under the influence of a dominant density modifier gene known as $Dm^{\dagger\dagger}$. This dominant modifier gene has also been stated to give a metallic patina to other dilute colours such as blue and lilac, converting them to caramel and taupe respectively (Vella et al, 1999). While there is much phenotypic support for the existence of colour variation in the dilute coat colours, there is considerable debate among Australian cat fanciers as to the genetic basis of these variant colours. The original proposal put forward by Patricia Turner, the foundation breeder of caramels in UK, relies on the inheritance of a single dominant density modifier gene that spontaneously arose in one founder cat in the 1970's (Turner, 1992). While this explanation may turn out to be correct for some phenotypic apricots, caramels or taupes it does not adequately explain the claimed prevalence of these controversial coat colours in many different, independently-bred cat breeds found simultaneously in Australia over the past 3 years. On the other hand, DNA evidence in mice would suggest that there are numerous alternative gene forms or alleles of the recessive dilute density gene that can also tone or tarnish coat colours (Davis and Justice, 1998). These observations have lead to the hypothesis that there may be a series of alleles for the feline dilute gene similar in principle, to the albino series: cc, c^sc^s, c^bc^b and c^sc^b that alter the black gene in cats to albino, seal point, Burmese brown and natural mink in Tonkinese, respectively (Fowler, 2000). Given that there is little evidence from some Australian cat pedigrees for the direct inheritance of the dominant Dm gene from cats in the UK, a series of mutant alleles or different forms of the dilute gene and/or other related genes that share the same or related pigment pathway, may well explain some of the observed colour variation. Further evidence that different alleles and/or gene modifiers can alter the warmth of coat

colours comes from the breeders of Australian Mist cats who have successfully established a relatively new breed of cats with a characteristic rufous mist over their coats. Interestingly, a show colour category for 'caramel' Australian Mists has been recently developed by the Australian Cat Federation (ACF) despite these selectively-bred cats bearing no direct pedigree ties to the foundation caramels described in the UK (Dr Truda Straede, personal communication). If these cats are indeed true genetic caramels then it would suggest that a new spontaneous mutation analogous to the Dm gene has independently occurred in the Australian Mist breed.

At present it would seem reasonable to keep an open mind as to the genetic basis of apricot, caramel and taupe cats from Australia and the UK until scientists undertake DNA sequencing of candidate genes. Meanwhile it would be helpful if visiting experienced judges from overseas could share their knowledge with Australian judges and fanciers when judging cats of the Dm phenotype, highlighting those cats that have correct apricot, caramel or taupe coat colours. This would enormously help local judges and fanciers in recognising proper Dm-based coat colours as opposed to 'bad blues or lilacs or creams'.

Colour speciality breeding - a bygone era?

It is also worth noting that established cat breeders in Australia have observed odd-coloured blues, lilacs and fawns as well as 'hot' or 'brown paper bag' creams in unrelated breeds for decades. Many breeders have tried to minimise them by reducing the effect of possible allelic variants and/or polygenes by not radically mixing colours when breeding. Also breeders note that the intermating of colours has increased in latter years with specialist colour breeding diminishing. Some fanciers believe that this has been due to decreasing gene pools, the introduction of new coat colours into established breeds and the putting into practice judges' powerful message that 'Type is paramount'.

Time to revisit registration and show standards

In summary, our CCCA show standards give an inconsistent message to breeders and judges of 'O' gene cats. For example, in the dense tortie we ask for red and cream patches to be present, yet we expect their red solid male offspring to show no unevenness of coat colour. It is time that we recognised that many reds and creams with tabby markings have been registered as solids or selfs simply because their parents were registered as such. To overcome this anomaly, overseas governing bodies have allowed registration of red or cream kittens according to phenotype rather than whether there is a registered tabby parent or not. This has some merit given the difficulty in knowing whether a red or cream cat with tabby markings has the agouti or non-agouti gene present. At present our show standards and registration policies for 'O' gene cat needs an overhaul so as to reflect our current knowledge of genetics and minimise confusion. Regardless of whether the CCCA adopts the policy of allowing red or cream tabbies from solid parents to be shown in the tabby section, the pattern of all tabbies ie classic, mackerel, spotted or ticked should be clearly stated on registration papers and in the judges' books. Hopefully, at the end of the day the right changes to registration policies and show standards will result in less controversy when breeding, registering and judging the delightful 'O' gene cats.

Footnotes

† The use of black tabby (CCCA Show Standards, 2002) or ebony tabby (CFA Show Standards, 2000-2001) terminology instead of brown tabby has caused some confusion of late with the Group 2 cats in Australia. Conventional coat colour categories for tabbies are generally described by the colour of the tabby's markings however the brown tabby with its black markings and brown ground colour has in many breeds adopted the colour of its ground colour in the naming process. This has largely come about to distinguish the brown tabby from the silver tabby, which also has black markings (Brown 1992). We need to be mindful that when judging

the black or ebony tabby we are actually judging a brown tabby. The simple explanation for this is that the governing body has elected to use the black tabby markings to define its colour category rather than the brown ground colour. Under no circumstance should these black or brown or ebony Oriental tabbies be referred to as seal tabbies because they do not have two copies of the recessive himalayan coat pattern gene known as $c^s c^s$, which acts on the black gene to form seal pointed cats. Neither do they have the Burmese colour gene known as $c^b c^b$, which modifies black to form Burmese brown, also described in Australian standard as seal brown (CCCA Show Standards, 2002). It should be noted that Burmese brown is described in the USA as sable in recognition of the genotypic difference between $c^b c^b$ and $c^s c^s$ (CFA Show Standards, 2000-2001).

†† The term apricot frequently appears in colourpoint standards as a colour description of red pointed cats that do not have the dominant density modifier gene. If the colour apricot as described by Patricia Turner stands up to DNA scrutiny then another description for the red colourpoints is required.

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Wrapping Presents 101 for Cat Owners

1. Clear large space on table for wrapping present.
2. Go to wardrobe and collect bag in which present is contained, and close door.
3. Open door and remove cat from wardrobe.
4. Go to cupboard and retrieve rolls of wrapping paper.
5. Go back and remove cat from cupboard.
6. Go to drawer and collect transparent sticky tape, ribbons, scissors, labels, etc.
7. Lay out present and wrapping materials on table.
8. Go back to drawer to get string, remove cat that has been in the drawer since last visit.
9. Remove present from bag. 10. Remove cat from bag.
11. Open box to check present, remove cat from box, replace present.
12. Lay out paper to enable cutting to size.
13. Cut the paper to size, trying to keep the cutting line straight.
14. Throw away first sheet because cat tried to chase the scissors and tore the paper.
15. Cut second sheet of paper to size by putting cat in the bag the present came out of.
16. Place present on cut-to-size paper.
17. Lift up edges of paper to seal in present, wonder why edges now don't reach, and find cat between present and paper. Remove cat and retry.
18. Place object on paper, to hold in place, while cutting transparent sticky tape.
19. Spend next 20 minutes carefully trying to remove transparent sticky tape from cat with pair of nail scissors.
20. Seal paper down with transparent sticky tape, making corners as neat as possible.
21. Look for roll of ribbon; chase cat down hall and retrieve ribbon.
22. Try to wrap present with ribbon in a two-directional turn.
23. Re-roll up ribbon and remove paper that is now torn, due to cat chasing ribbon end.
24. Repeat steps 12-22 until down to last sheet of paper.
25. Decide to skip steps 12-16 in order to save time and reduce risk of losing last sheet of paper. Retrieve old cardboard box that you know is right size for sheet of paper.
26. Put present in box, and tie down with string.
27. Remove string, open box and remove cat.
28. Put all packing materials in bag with present and head for lockable room.
29. Once inside room, lock door and start to re-lay out packing materials.
30. Remove cat from box, unlock door, put cat outside door, close door and re-lock.
31. Lay out last sheet of paper. (Admittedly this is difficult in the small area of the toilet, but try your best!)
32. Seal box, wrap with paper and start repairs by sealing down tears with transparent sticky tape. Now tie up with ribbon and decorate with bows to hide worst affected areas.
33 Admire your handiwork, congratulating yourself on making good of a bad job.
34. Unlock door, and go to kitchen to make drink and feed cat.
35. Spend next 15 minutes looking for cat, before coming to obvious conclusion.
36. Unwrap present, untie box and remove cat.
37. Retrieve all discarded sheets of wrapping paper, feed cat and retire to lockable room for last attempt, making certain you are alone and the door is locked.
38. At time of handing over present, smile sweetly at receiver's face, as they try and hide their contempt at being handed such a badly wrapped present.
39. Swear to yourself that next year, you will get the store to wrap the darn thing for you.

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