Cat Coat Genetics

The genetics of coat color in cats and other furry mammals is complex. During development of the embryo, melanoblasts (precursors to melanocytes, the melanin-producing cells) migrate in a dorsal-to-ventral direction from the neural crest, and end up in the skin. This migration is controlled by at least two genes (w and S). Once at their target location the melanoblasts differentiate into the melanocytes and can start pigment production. The production of, pattern and location of pigmentation depends on additional genes, some of which are discussed below. How all of these genes interact is still being determined; once known, this information may help geneticists to understand the gene interactions of some human diseases, too.

In this laboratory exercise you will examine the coats of cats with respect to certain features such as color, pattern, and texture, and deduce as much as you can about the underlying genotypes. The guide below takes you through what goes into pigment production and the genes involved in that process. Record the results for the cats you observe in the table provided.

Hair length
L or l
LL or Ll cats have short hair; ll cats have long hair. Every kitten is born with all the hair follicles it will ever have. Each hair follicle can produce hair in several rounds. The hair has a distinct growth period followed by a resting period. Eventually, the hair is shed and the follicle will produce another hair. A popular hypothesis to explain long-haired cats is that in ll cats, the growth phase is extended beyond the normal range.

Coat Pigment Distribution Genes
The distribution genes control where in the fur pigment will be displayed. Pigment is displayed in melanocytes. The distribution genes control whether melanoblasts migrate to the skin to produce melanocytes, and how much of the skin contains melanocytes.

W or w
W is called the “dominant-white” gene because it is an epistatic gene. Before melanocytes can start making fur pigment, the melanoblasts must migrate to the skin. Melanoblasts make it to the skin only in ww (homozygous recessive) animals. In WW or Ww animals, the skin lacks melanocytes and the cat is a solid white color (W is a completely dominant allele). The epistatic dominant white phenotype masks other pigment traits. A cat that has any color in its fur will be ww.

S or s
S is the “piebald-spotting” gene – a second gene that affects melanocyte migration. S and s are incompletely dominant alleles. In ss animals, melanocytes migrate evenly to the ventral surface of the animal, so the cat is completely pigmented. Ss animals have less than 50% white fur and SS animals have white patches on more than 50% of the body.

Pigment Production Genes
The pigment producing genes control the production of melanin. Melanin production is a metabolic pathway that starts with the amino acid, tyrosine. There are two forms of melanin: eumelanin (black) and phaeomelanin (orange/yellow).
The C, or color, gene product is tyrosinase, an enzyme responsible for the first step in the synthesis of melanin from the amino acid tyrosine. The C allele is fully dominant and gives full coat color. Alleles cb (Burmese) and cs (Siamese) produce temperature-sensitive versions of the enzyme – the enzyme works at lower temperatures but not at the normal core body temperature of the cat. Consequently, pigment production is restricted to the cooler extremities. cb is less temperature sensitive than cs, so there is more pigmentation with cb cb cats than with cs cs cats. Even at the extremities, however, these temperature sensitive enzymes are not working to full capacity; the color at the extremity tips of a Siamese cat is usually not as intense as the color in a cat with the dominant allele, C. Allele c does not produce any active enzyme; cc animals are albino (white). cb cs cats are Tonkinese, a non-pure breeding hybrid of the Burmese and Siamese. (A litter of kittens whose father and mother are both Tonkinese may have up to 12 different coat patterns ranging from that of the typical Burmese and Siamese as well as intermediates.) The dominance series for coat color alleles is C > cb = cs > c. (C is fully dominant over the other alleles; cb and cs are incompletely dominant with respect to each other, and c is fully recessive to the other alleles.) Note: cc is the only genotype that is epistatic to the W allele; an albino cat cannot produce any pigment at all, no matter whether the melanocytes are present in the skin or not. Albino cats also lack eye pigmentation.

B or b
The B allele is also required for production of eumelanin (the black pigment) and is dominant to b. Homozygous recessive bb animals are brown or chocolate. Because the starting point for melanin synthesis is the amino acid tyrosine, even BB or Bb cats may have a brownish tinge to their fur if their diet is deficient in tyrosine. (Note: The sable (dark chocolate) coat color common in Burmese cats is the result of their cb cb alleles, not bb.)

XO or Xo
The orange gene (O or o) is located on the X chromosome. XO converts eumelanin (black) to phaeomelanin (red/orange/yellow). Xo results in no melanin conversion. Male cats are either orange (XOY) or black (XoY), while females can be orange (XOXO), black (XoXo), or orange – black mixed (XOXo). This is because the orange gene is located on the "X" chromosome, which is affected by the Barr body formation in different cell lines during development.

Tortoiseshell females are orange – black mixed (XOXo) lacking white patches as a result of the recessive piebald spotting genotype (ss – full migration of melanocytes to the skin). Calico females are orange – black mixed (XOXo) with white patches (SS or Ss piebald genotypes).

Color Dilution (or Color Density)

D or d
Color intensity can be full (dense, DD or Dd) or dilute (dd). The pigment itself is not altered in dd cats, but the pigment is deposited in clumps rather than evenly; so the pigmentation appears diluted or pale. Black appears blue or gray, chocolate is seen as lilac, and red/orange as cream.

Color Pattern and Inhibition
Although we will not be determining the T, A or I gene patterns in today's lab exercise, they are genes that affect the intensity of pigment distribution, and hence and pigment patterns in fur, and might be of interest.

A or a – Agouti pattern
Agouti fur (AA or Aa) has a band of yellow near the tip of each black hair. The yellow band results from transient inhibition of the B gene by the Agouti protein during hair growth. The inhibition does not occur in aa animals, so each hair is a solid color. The agouti banding pattern cannot be detected in orange cats.
Melanin inhibition is seen in $II$ or $Ii$ cats. Melanin inhibition is particularly effective in the agouti banding pattern, so the fur looks silver. (Recall that the agouti pattern (yellow banding) is visible only in black-pigmented cats.)

$T$, $T^s$, and $t^b$ – Tabby marking.
The tabby pattern of dark stripes is caused by increased intensity of pigmentation in some areas of the body. It is best represented in cats with agouti pattern where the agouti yellow banding is in stripes (or spots) on the fur – the hair within the dark stripes shows solid color. The tabby pattern is difficult to see in non-agouti cats but appears under certain lighting conditions or in young cats whose pigmentation has not matured. The predominant tabby patterns are **mackerel** ($TT$ or $Tt^b$) with thin, vertical stripes on the side of the body, as in a tiger; **Abyssinian** ($T^sT^s$) with almost undetectable striping on the body but somewhat detectable on the face, legs and tail; or **blotched** ($t^bt^b$) in which the even, vertical stripes of the mackerel are replaced by broader bands that can form spirals or bulls-eye patterns. The mackerel allele ($T$) is dominant to blotched ($t^b$) while Abyssinian ($T^s$) is incompletely dominant to both mackerel and blotched: Some weak striping is seen on legs and tails of $T^sT^s$ or $T^st^b$ heterozygotes.

**Additional cat coat gene information**
The $W$ gene is also known as c-kit. The normal c-kit protein (made by the $w$ allele) is a melanoblast cell-surface receptor. When the receptor receives a signal in the form of a ligand (called Steel), the cell can survive and proliferate. From research on mice, it appears that failure to receive adequate levels of this signal causes cell death. What does this information suggest about the white coat color phenotype of $WW$ or $Ww$ animals? In humans, c-kit mutations are associated with some forms of leukemia and ovarian cancer.

The incompletely dominant gene $S$ codes for “endothelin receptor B” (EDNRB), required for migration of melanoblasts toward the ventral side of the embryo. The $ss$ genotype produces EDNRB. The $S$ allele inhibits production of EDNRB and impedes this migration, resulting in patches of white fur. $Ss$ cats have some white patches of fur while migration is blocked even further in $SS$ animals, causing larger patches of white.

Melanoblasts also differentiate into neural cells involved in sound detection in the ear. Loss of melanoblasts can result in deafness – an example of pleiotropy, where one gene can affect different and seemingly unrelated processes.

**Cat web sites to visit:**
http://bs-biosci.unl.edu/GCMB/Christensen/cats.html
http://www.esb.utexas.edu/hampton/zoo325l/Cats/index.htm

Note: This laboratory exercise was adapted from the University of Washington Genetics 371, Winter 2003, Quiz Section, and updated by Al Farrand, Bellevue College Biology Faculty. The accompanying PowerPoint Presentation was prepared by Joe Shippert, former Bellevue College Biology 211 student, based on the Genetics 371 laboratory exercise,
Using the 5 cats below, complete the table below. Include all possible genotypes for each gene.

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<thead>
<tr>
<th>Cat Coat Genes</th>
<th>Possible Genotype</th>
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<tbody>
<tr>
<td></td>
<td>W  S  C  B  X  D  L</td>
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**Cat #1**

![Cat #1 Image](image1)

**Cat #2**

![Cat #2 Image](image2)

**Cat #3**

![Cat #3 Image](image3)

**Cat #4**

![Cat #4 Image](image4)

**Cat #5**

![Cat #5 Image](image5)

**Bonus Cats – These are the same breed**

![Bonus Cats Images](image6)