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Poultry Genetics for the Nonprofessional

Brief introduction

The purpose of this document is to provide a poultry genetics reference to interested poultry enthusiasts who may not have any formal training in genetics. My personal goal is to provide the enthusiast with '*the document that I wish I had had when I started poultry keeping'*.

Literature sources are primarily *Poultry Breeding and Genetics*, Elsevier 1990, R.D. Crawford, Editor, books by F.P. Jeffrey, W.F. Hollander, F.B. Hutt and poultry science journals like *Poultry Science* which are available in most university libraries. Care is taken to keep the information in these pages current and to correct any errors.

Part III is a list of chicken genes with comments regarding gene functions and related information which we hope will be an easy-access reference for poultry enthusiasts.

I. Basic Genetics

DNA, genes and chromosomes:

I am intentionally avoiding jargon. However, there are a few basic terms that are necessary.

A *gene* is a piece of DNA that carries information about a specific trait.

A *chromosome* is a string of genes connected together (although most of the chromosome is DNA that has no known function or no genetic activity).

An *allele* is a gene that is a member of a set of genes that all belong to the same locus, or location, on a chromosome. These genes are often thought of as being related to each other through mutations (one allele could be a mutation of another allele) or they could be mutations of an ancestor gene.

Chicken*s*, like people, usually have two of every chromosome. The chromosomes in a chromosome pair are not identical, since one comes from each parent. A gene is said to be *dominant* when only one gene (rather than two) is sufficient for the expression of that trait to which the gene corresponds. Some genes are referred to as *incompletely dominant*. The expression of these genes is inhibited by (usually unknown) modifying genes. When the inhibiting, modifying genes are not present, the incompletely dominant gene expresses. This interaction with modifying genes is responsible for the seemingly random nature of the expression of incompletely dominant genes.

The sex chromosomes are unique in that there are two types, a long sex chromosome, the Z chromosome, and a short sex

chromosome, the W chromosome. The female has one long and one short sex chromosome, she has ZW sex chromosomes. The male has two long sex chromosomes, he has ZZ sex chromosomes. For this reason, the female has only one copy of some genes that are on the long, Z, sex chromosome.

The genes that are not on the sex chromosomes are called 'autosomal' or autosomes. Both male and female chickens have two of these genes. Chickens have 39 pairs of chromosomes (78 individual chromosomes). Most of them are tiny and referred to as 'dot' or micro chromosomes.

An important point is that, when we talk about adding or removing a gene, say frizzle, F, we don't intend that the chromosome is lengthened or shortened by the addition or deletion of that gene. Rather the frizzle gene, F, replaces the gene of the wild-type jungle fowl, f^+, when it is added, or, it is itself replaced by the wild-type jungle fowl gene, f^+, when frizzle is removed. I used the frizzle gene as an example here, but the statement applies to all genes.

Generation notation:

The original members of a mating are referred to as the parental (P) generation. The first generation of progeny from the parental cross is referred to as the first filial generation, F1. The progeny of a cross in which one or both of the parents are from the F1 generation is an F2 generation (F1 x F1 = F2) and so on.

Homo / hetero / hemi – zygous…genotype and phenotype

For the interested reader who might like to know the meaning of these terms, I have included this brief description. A bird that has one gene, rather than two, for a specific trait is said to be heterozygous for that trait. A bird that has two genes for a given trait is homozygous for that trait. The genotype is the actual set of genes. The phenotype is the appearance or visual characteristics…what you can see. For example, a bird that is heterozygous (has one gene instead of two) for a given dominant trait may look the same as, or similar to, one that is homozygous (has two genes) for that trait. They both have the same appearance or phenotype. Because the female fowl have differing sex chromosomes, the long one, Z, and the short one, W, the Z chromosome has gene locations that the W chromosome does not (see above). Sometimes when referring to these genes that have no counterpart on the W chromosome, the female is said to be hemizygous. Since the female can have only one copy of these genes, there is an apparent overlap in the meanings of 'heterozygous' and 'hemizygous'.

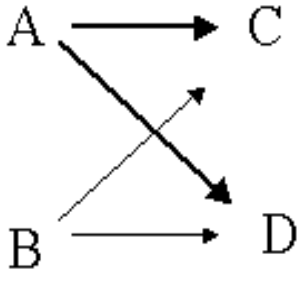
How to predict the outcomes of breeding events for non-sex-linked and sex-linked traits

Non sex-linked traits:

Both parents have two genes for a given trait. Let's consider the gene for frizzle plumage, F, and agree that we will represent the lack of the frizzle gene with f^+. The superscript '+' indicates that the gene is present in the wild-type fowl which, with respect to chickens, is the red jungle fowl. Here, I apply the jargon immediately above, but will minimize the use of it from now on. A bird is said to be heterozygous for frizzle if her genotype is (F, f^+) and homozygous if her genotype is (F, F). Since frizzle is dominant, both genotypes will have the same (or similar) appearance or phenotypes. (In this particular case, frizzle shows a 'dose effect' and the frizzle homozygote has brittle feathers that usually break off so the homozygotes can be almost bare. There is a common recessive modifying factor, mf, that reduces the influence of the frizzle gene.)

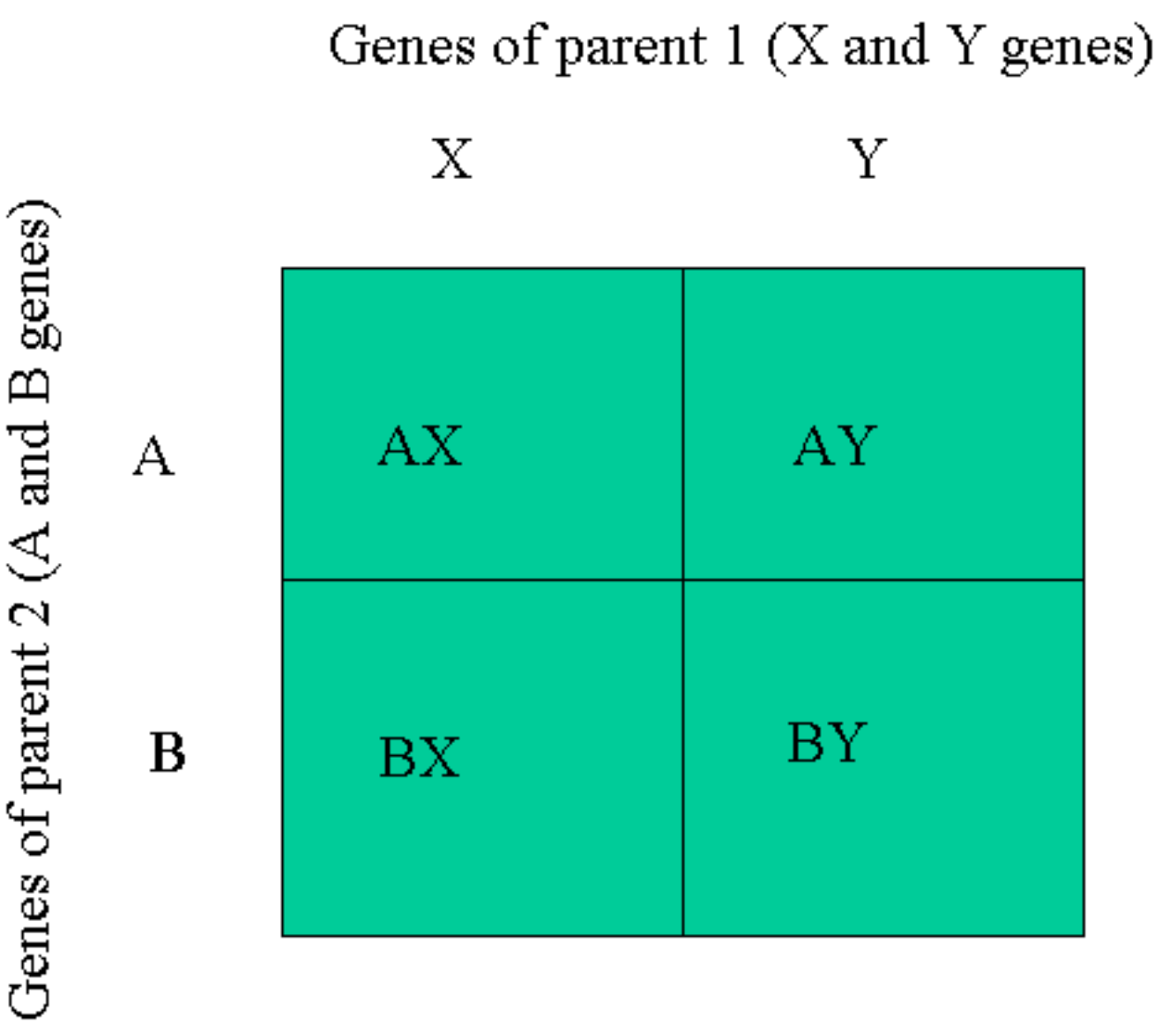
To determine the genetics of the offspring, one takes the four possible combinations of the genes of one parent with the genes of the other parent. For example, let's consider a cross between a bird that has two frizzle genes, homozygous for frizzle, (F, F) and one that is without frizzle, (f^+, f^+). It helps with the bookkeeping for our purposes here if we (artificially) number the genes: (F1, F2) and (f^+1, f^+2) so that F1 is the first frizzle gene of the first parent, F2 is the second frizzle gene of the first parent and so on. The four possible pairs that can be made by combining these genes are: (F1, f^+1), (F1, f^+2), (F2, f^+1) and (F2, f^+2). Since frizzle is a dominant trait, these four gene combinations will result in chickens with frizzle plumage (they will all have the same or similar phenotypes). In practice one would not number the genes as I have done in this paragraph. I numbered them to distinguish the four combinations, since they are all genetically the same. One would normally write: (F, F) crossed with (f^+, f^+) gives (F, f^+) times 4.

So, in order to get the four combinations of the genes of the two parents, just take the first gene of the first pair with each gene of the second pair, then do the same thing with the second gene of the first pair. The figure below illustrates how to get the combinations of genes of one parent, (A, B), and the genes of another parent, (C, D). The four possible combinations are (A, C), (A, D), (B, C) and (B, D).



The Punnett Diagram

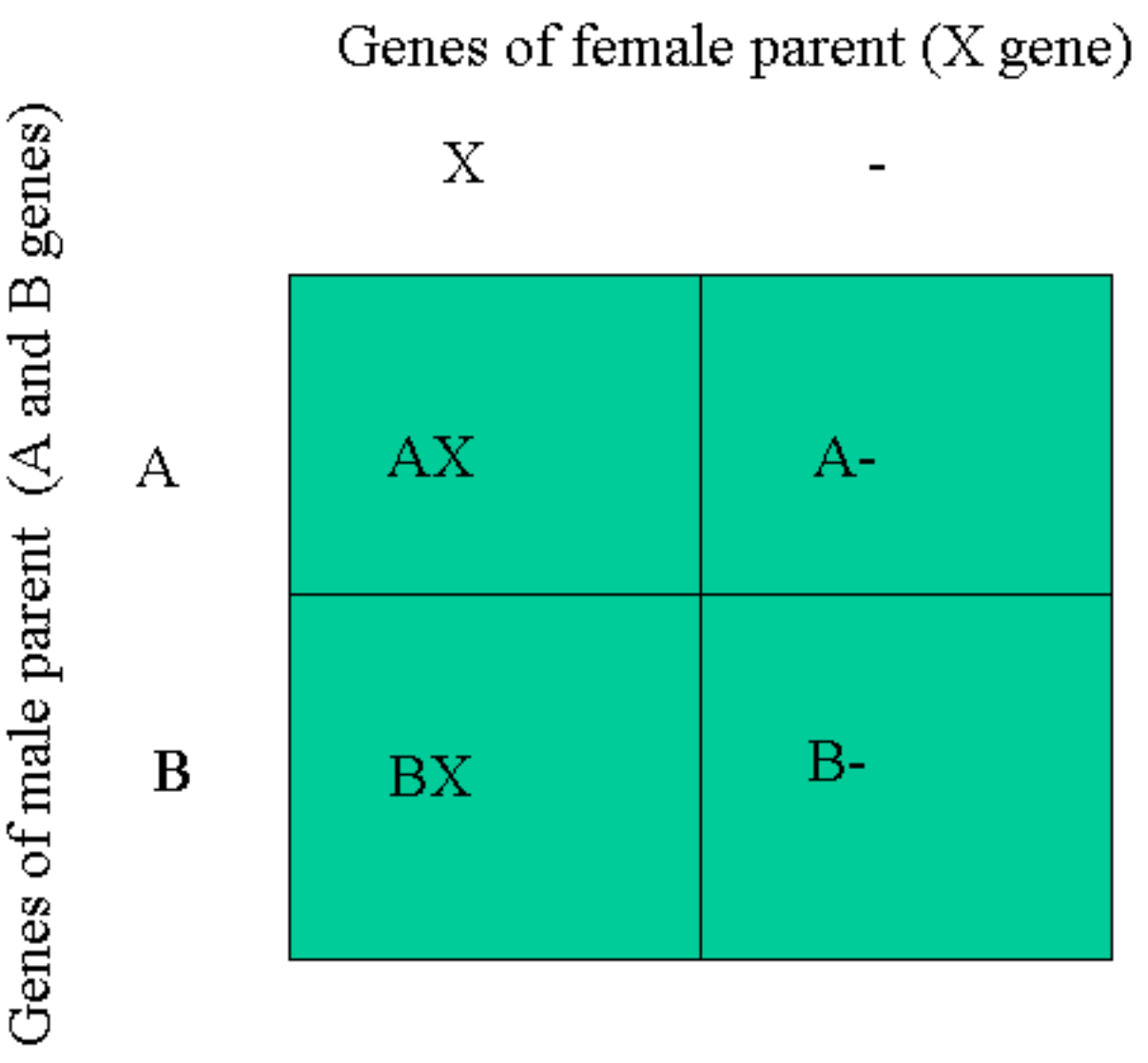
Another way to determine the 4 different combinations of the genes for a trait (two from each parent) is to use a Punnett diagram. In this method one makes a square that is divided into four quadrants. Write the genes of one parent across the top with one gene over each quadrant as in the figure below. Write the genes of the second parent along the left side. In the figure below, one parent has genes X and Y, while the other parent has genes A and B. This is a slightly different notation than I used in the previous drawing. Now, in each quadrant write a gene from each parent. Write the gene from each parent that is physically nearest the quadrant you are presently working in. Another way to say this is to put the 'A' gene in the top row of quadrants, the 'B' gene in the bottom row of quadrants, the 'X' gene in the first column of quadrants and the 'Y' gene in the second column of quadrants. Hopefully the figure below makes this process clear. The Punnett_Monkey is an application that will do the Punnett square for you.



Sex-linked traits:

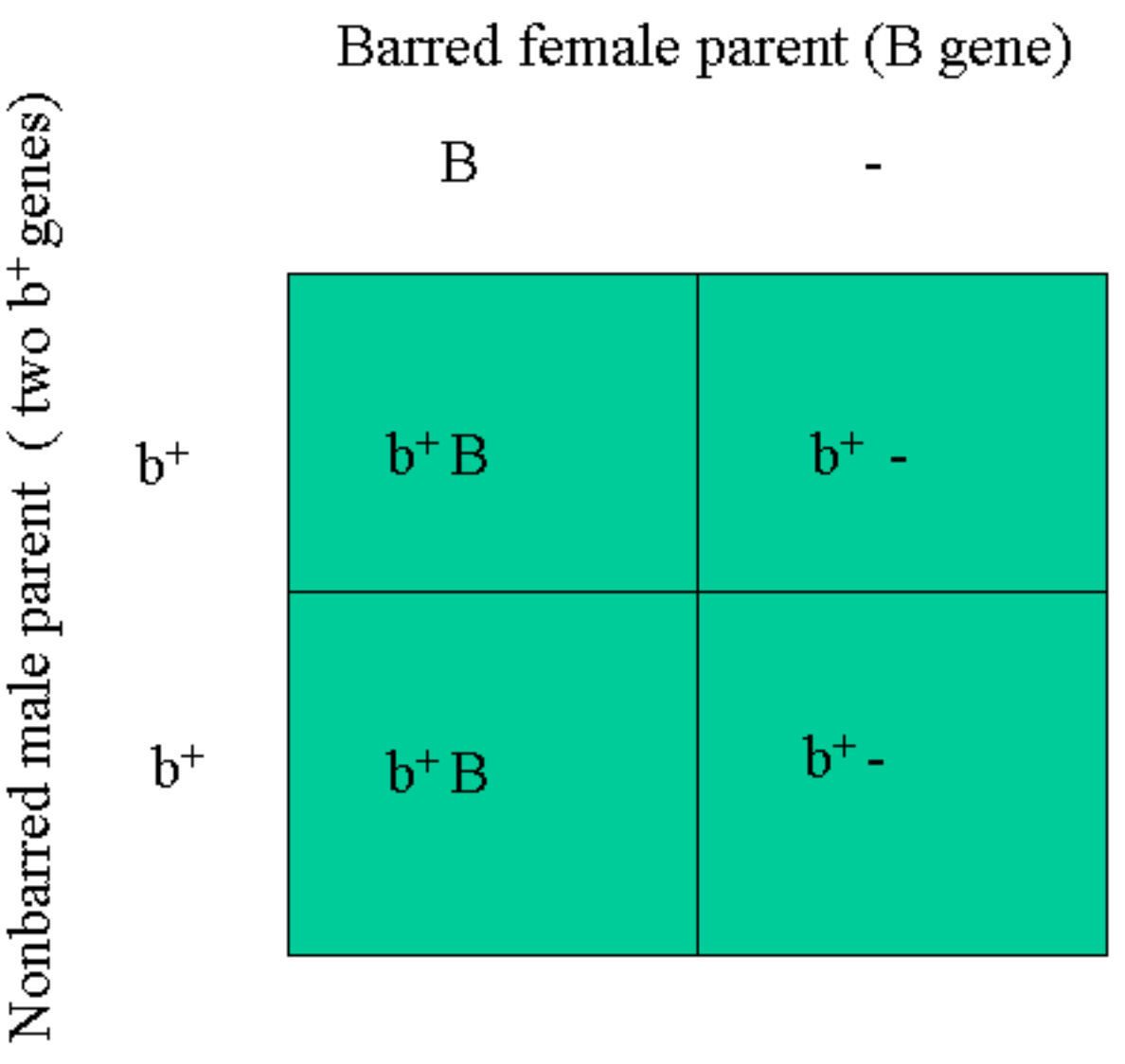
Genes responsible for sex-linked traits are on the long sex chromosome, Z, but not the short sex chromosome, W. For that reason, I prefer to use a blank or underscore to represent the missing gene when representing the genes (genotype) of the female. Some people write a 'W' to indicate that the presence of the W sex chromosome which lacks the locus for the gene in question. Rather than writing (B, -) to represent a barred female, some authors write (B, W) or B/W with the slash separating the gene symbols for the different chromosomes. I include a clickable link to the chicken gene tables so the reader can look up the genes that are used in these examples.

A Punnett diagram (or Punnett square) for determining the mating outcomes for sex-linked traits is below:



In this example the fact that the female can have only one gene is represented with a dash or underscore in one column of quadrants. The dash or underscore actually represents the W chromosome which is what makes her female. The other gene symbols represent genes that are all allelic to each other (that means that they all belong to the same location on the chromosome). The chicks that 'inherit the dash' really inherit the W sex chromosome and are therefore female. In the example in the Punnett square above, the (A,_) and (B,_) chicks are the females.

As an example of sex-linked barring (I use it a different way below), the A and B gene could both represent the wild-type gene, b^+, which is lack of barring. The X gene could represent the barring gene, B. So this is a mating between a barred female and a nonbarred male. The Punnett square predicts that all the male chicks will be barred [(B, b^+) genotype] and the females will all be nonbarred [(b^+, _) genotype]. Punnett square below is this mating example (The Punnett_Monkey is an application that will do the Punnett square for you.):



Saying the same thing a little bit differently, Cuckoo barring or sex-linked barring, B, is one of these genes that is located on the Z chromosome (recalling that the male has two Z sex chromosomes and the female has one Z and one W sex chromosome and there is no barring gene on the W chromosome so females can have only one). A male with two copies of the barring gene might be represented as (B, B). This is his 'genotype' with respect to barring. A male with one copy is represented as (B, b^+) where lower-case b^+ indicates a lack of the barring gene and the b^+gene is the gene that the wild red jungle fowl has instead of the barring gene, B. A male with no barring is represented as (b^+, b^+). Since the female has one long chromosome and one short chromosome and the barring gene is on the long chromosome and not the short one, a female can have only one copy of the barring gene. A female with barring is represented as (B, _). A female without barring is represented as (b^+, _). The underscore indicates her short chromosome lacks the locus (location) of that gene.

I distinguish between the terms 'sex-linked' and 'sex-indicating'. A gene is sex-linked when that gene is on the long sex chromosome and not the short sex chromosome. A sex-indicating trait is one that arises from a sex-linked gene if the cross is carried out properly. The following example indicates this.

When a barred female, (B, _) is crossed with a non-barred male, (b^+, b^+), the four possible outcomes (see the figure above) are: (B, b^+), (b^+, _), (B, b^+) and (b^+, _). Here I have written all four combinations even though some are the same. The order of writing the genes of the pair is usually to write the dominant gene first and the blank last. Of the four possible outcomes, the males are barred and the females are non-barred. So, when the cross is carried out this way, barred female x non-barred male, the barring is a sex-indicating trait, and indicates male offspring.

When a barred male, (B, B) is crossed with a non-barred female, (b^+, _), the four possible combinations of the genes are: (B, b^+), (B, _), (B, b^+) and (B, _). Therefore all the chicks will be barred. The barring is still a sex-linked gene, but the cross was carried out in a way that leads to both males and females being barred. In this situation the barring is not indicative of the sex of the offspring.

The Sil-Go-Links (for silver-gold-sex-link) are similar except that the dominant sex-linked gene is the silver gene, S, which has the function of inhibiting the red pigment, pheomelanin. The lack of the silver gene is represented with lower-case s^+. Here again, s^+ is the gene that the wild-type fowl or red jungle fowl has instead of the silver gene. Crossing a red male lacking silver, such as a Rhode Island Red (s^+, s^+), with a silver female, such as a Delaware (S, _), gives (S, s^+), (S, s^+), (s^+, _) and (s^+, _). So, the males are silver (which means mostly white) and the females are red and can be sexed after hatching. Carrying out the cross the other way, a silver male (S, S) and a red female (s^+, _) gives as possible combinations: (S, s^+), (S, s^+), (S, _) and (S, _) so that the red pigment in both males and females is inhibited and they will be mostly white.

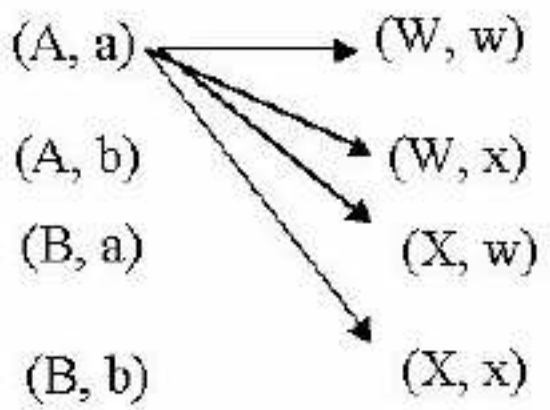
Black sex-links can be made by crossing Barred Rock females with a red male, such as Rhode Island Red or New Hampshire Red. The Barred Rock females have one barring gene, (B, _) and should have two nigrum genes (E, E). The nigrum gene extends black by changing red to black. The red male will be (b^+, b^+) for barring and lacking in nigrum, (e^+, e^+). Here the lack of nigrum is represented with the symbol, e^+, which indicates that, instead of the nigrum gene, E, the bird has the wild-type gene that the red jungle fowl has.

With respect to the barring, the four combinations of the genes are: (B, b^+), (B, b^+), (b^+, _) and (b^+, _). So the males are barred and the females are not. With respect to the nigrum gene, all four combinations are the same, (E, e^+). So, for this set of genes, all the chicks will be black and the males will be barred. The Punnett_Monkey is an application that will do the Punnett square for you.

How to predict the outcome of a breeding event when two pairs of genes are involved:

This is relevant in poultry genetics because there are traits that depend on two or more gene pairs. The Advanced Punnett_Monkey is an application that allows the user to select as many as four independent traits and, with a few mouse clicks, determine the frequency, in percent, that a given genotype will appear in the progeny. The comb type is an example. Shank and foot color are traits that depend on three pairs of genes. What we do in a case such as comb type that is determined by two pairs of genes, is to determine the combinations of the genes of each parent for both sets of genes. We then realize that the combinations of the first genes can occur with any combination of the second genes. So, we have to consider all the possible combinations of the genes of the first set with the genes of the second set.

An example illustrates this. Suppose a trait is determined by two sets of gene pairs on different chromosomes. A male with a genes (A, B) and (W, X) for this trait is crossed with a female having genes (a, b) and (w, x) for the same trait. The possible combinations from the first gene pair are: (A, a), (A, b), (B, a) and (B, b). The possible combinations of the second set of genes are: (W, w), (W, x), (X, w) and (X, x). We're not finished because each combination of the first set of genes can occur with any combination of the second set of genes. To determine these 'double' combinations it is helpful to make a drawing:

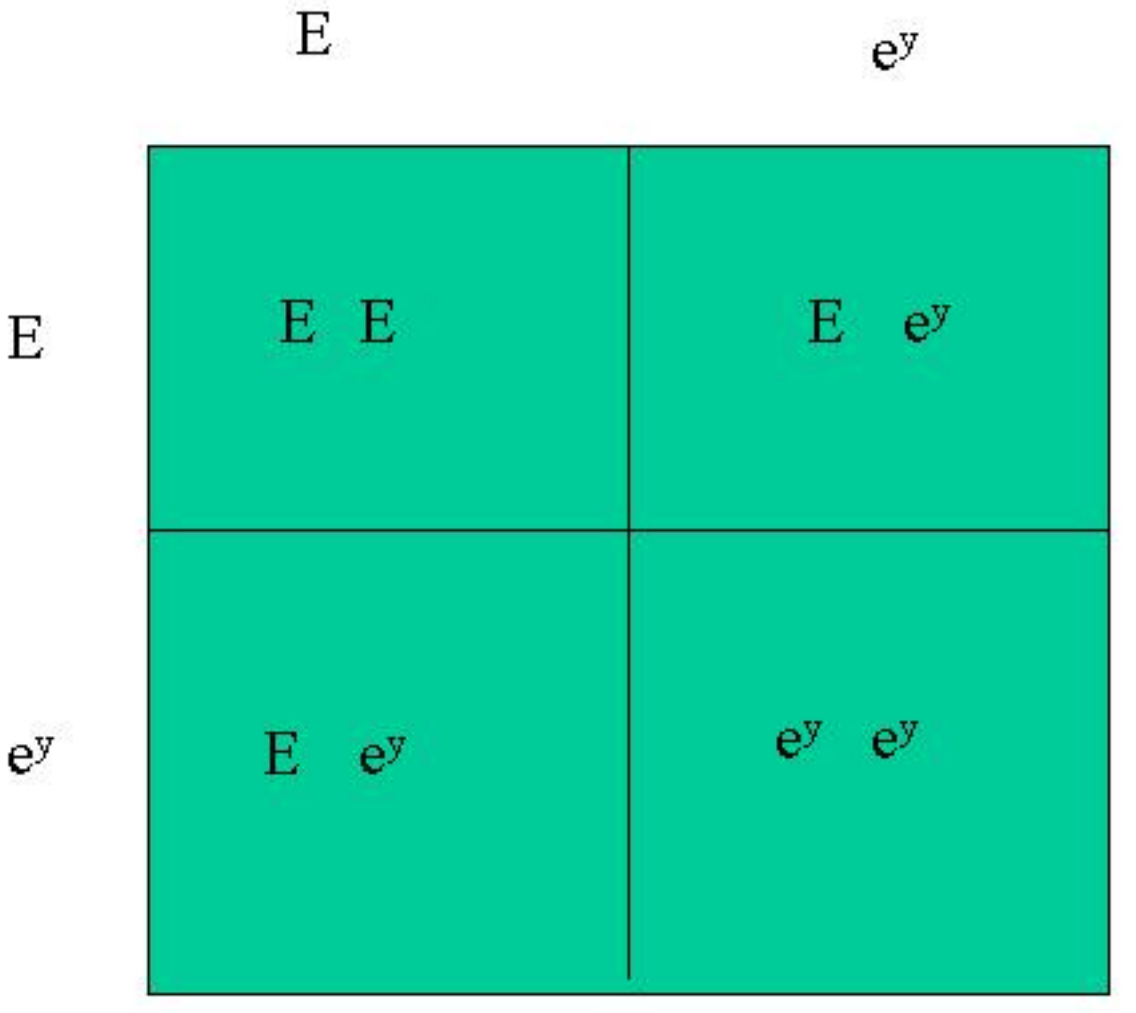


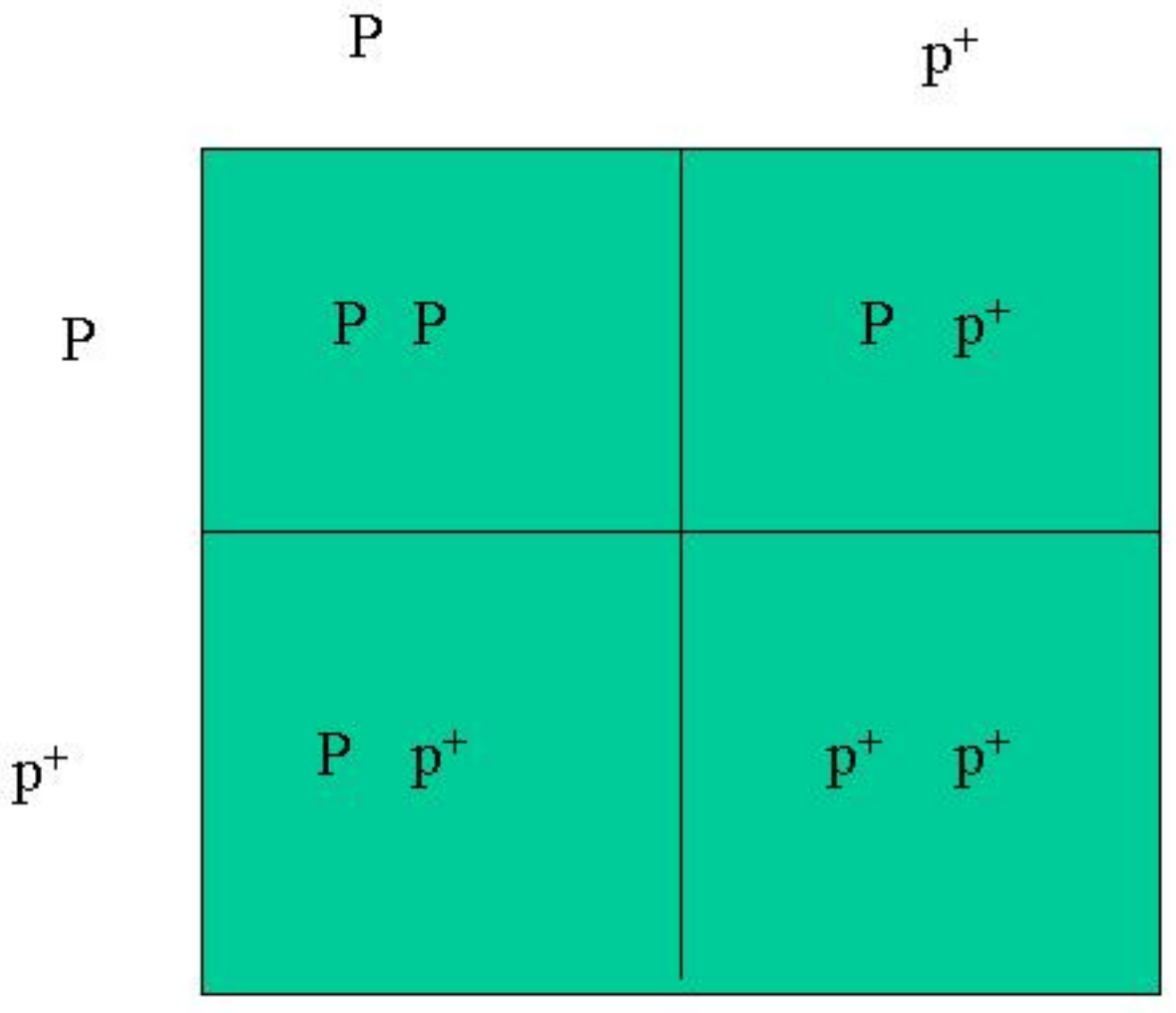
The arrows in the figure above indicate the combinations of the (A, a) gene pair with the four combinations of the second genes: (A, a) with (W, w); (A, a) with (W, x); (A, a) with (X, w); and (A, a) with (X, x). Next we do the same thing except using the (A, b) gene pair instead of the (A, a) one. Then again with the (B, a) gene pair and lastly with the (B, b) gene pair. This gives 16 'double' combinations.

Punnett diagram for the inheritance of two traits

The inheritance of two sets of gene pairs can be determined by the use of a Punnett diagram. In these examples, I start with single traits and work with Punnett diagrams. The strategy is to use Punnett diagrams to determine the combinations for the single traits separately, then use those Punnett square combinations to make the larger Punnett square for the inheritance of both traits together. The Araucana black large fowl is homozygous for both dominant black, E, and pea comb, P. The Rhode Island Red is homozygous for recessive wheaten, e^y and single comb, p^+ (which is really the lack of the pea comb gene). A mating between an Araucana black large fowl and a Rhode Island Red gives sons and daughters that belong to the F1 generation and are all heterozygous for dominant black E, recessive wheaten, e^y, P (pea comb) and p^+ (lack of pea comb). A mating between two of these F1 chickens is what I consider here. So, this is what you would get in the F2 generation.

The F1 sons and daughters are heterozygous for both traits. The F1 fowls are (E, e^y) and (P, p^+). Since we know this already, we don't need to use a Punnett square to determine the distribution of traits in the F1 generation. We do, however, need to use a Punnett square to determine how the genes combine with each other in the F2 generation (F1 x F1). Then we use a Punnett square again to determine how the two separate traits appear with each other. So the Punnett square for first trait has E and e^y on both edges. The other one has P and p^+ on both edges. These two Punnett squares determine how the genes for these traits are inherited separately in the F2 generation from F1 matings. The Punnett square for the inheritance of the individual traits are:


E
e^y
E
E E
E e^y
e^y
E e^y
e^y e^y



Now we make another Punnett square to determine how the two traits appear with each other in the F2 generation. The combinations from each of these single-trait Punnett squares above is used along an edge of a new and larger Punnett square to determine how the genes will appear together in the F2 chickens that come from mating F1 chickens to each other:

Results of Punnett Square for E-locus

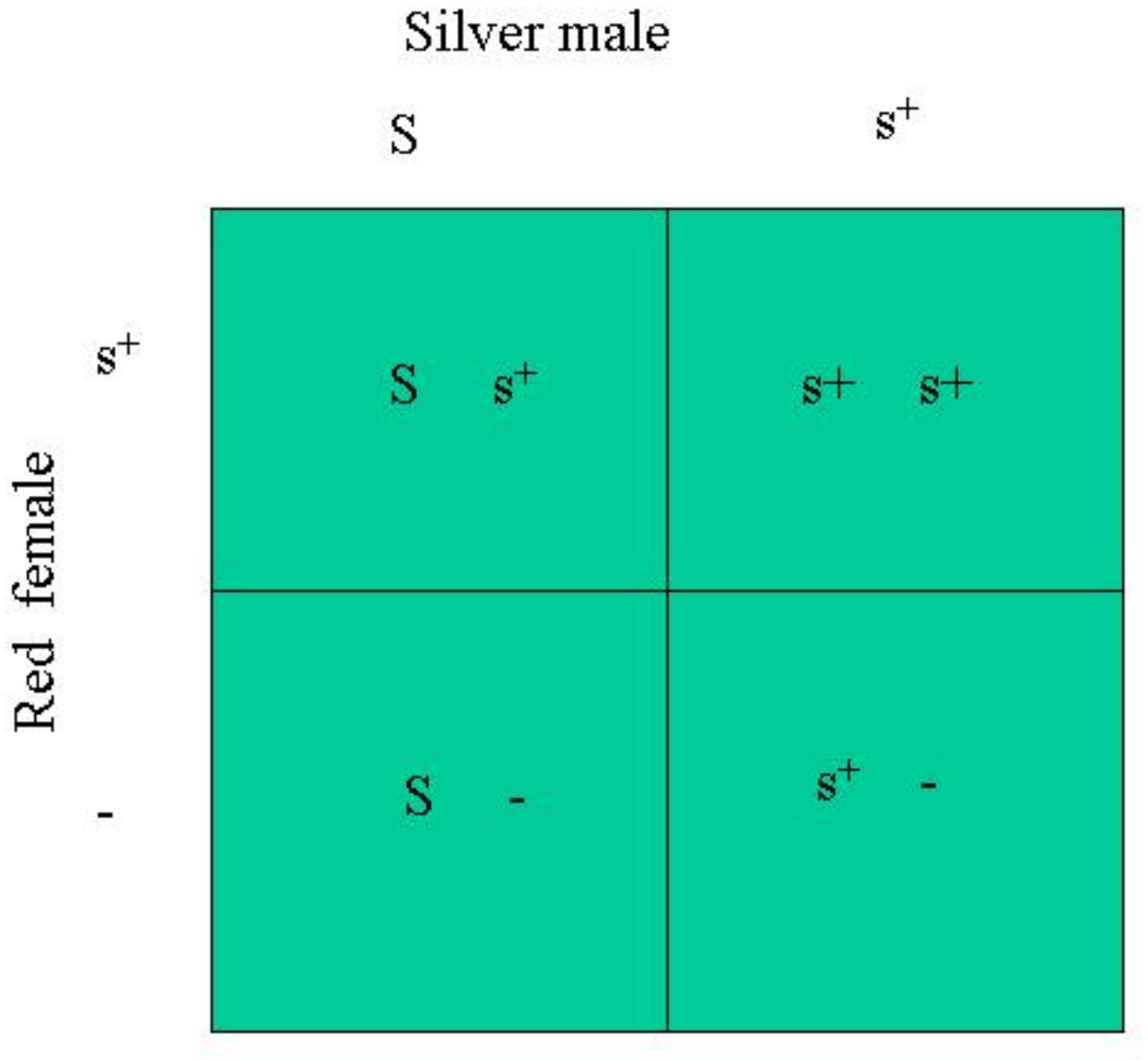
Results of Punnett Square for P-locus

	E E	E e^y	e^y E	e^y e^y
PP	E E PP	E e^y PP	e^y E PP	e^y e^y PP
Pp^+	E E Pp^+	E e^y Pp^+	e^y E Pp^+	e^y e^y Pp^+
p^+P	E E p^+P	E e^y p^+P	e^y E p^+P	e^y e^y p^+P
p^+p^+	E E p^+p^+	E e^y p^+p^+	e^y E p^+p^+	e^y e^y p^+p^+

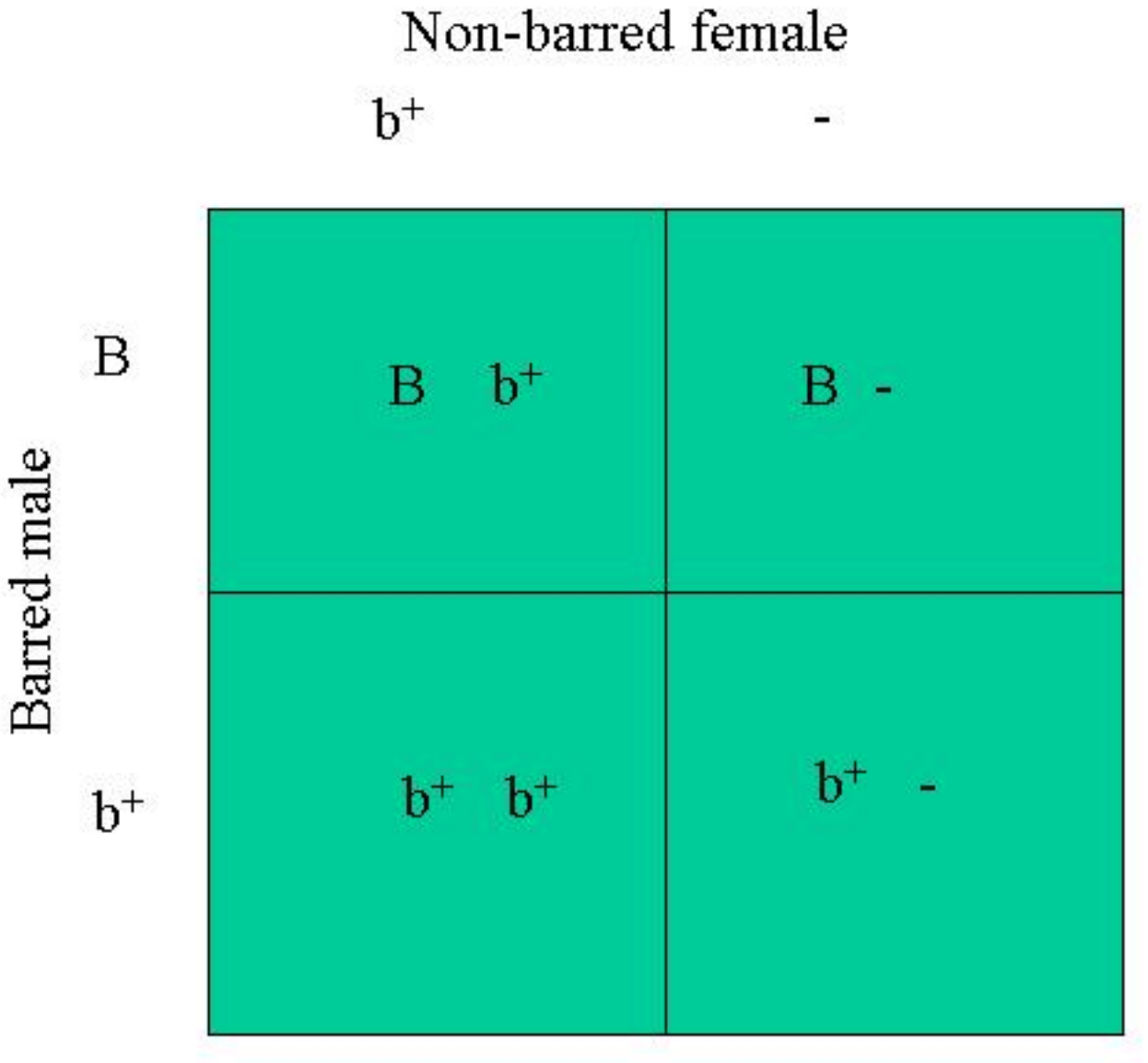
The gene combinations that give the same phenotype (appearance) have the same color in the Punnett square above. So, the F2 generation from a mating of Araucana and Rhode Island Red will have one fourth that are double heterozygotes (heterozygous for both traits). These are the four center squares above. This gene combination occurs four times out of 16, so the percent is 25%. These will probably be black with a 'poor' pea comb. It may be difficult or impossible to distinguish them from the EE Pp^+ birds. Only 1/16 will be black with a good, homozygous pea comb (the EE PP combination in the corner).

Because sex-linked traits are popular with fanciers, this next example considers the inheritance of two sex-linked traits. Consider the cross between a male that is heterozygous for silver and barring and a female that is red, non-barred. His genotype is (S, s^+) with respect to silver and (B, b^+) for barring. Her genotype is (s^+, -) with respect to silver and (b^+ -) with respect to barring. First, I make a Punnett square for the inheritance of the silver trait and another for the barring trait. I then make another Punnett square with the results of these Punnett squares.

For the silver trait, the Punnett square is:



The Punnett square for the barring trait is:



The results of these two Punnett diagrams are used to make a larger one. In the Punnet square below, I have written the results of the Punnett square for the silver trait across the top and the results of the Punnet square for the barring trait down the left-hand side. The interior squares of the Punnett diagram are filled the same way they are for the smaller Punnett diagrams.

Results of Punnett square for silver

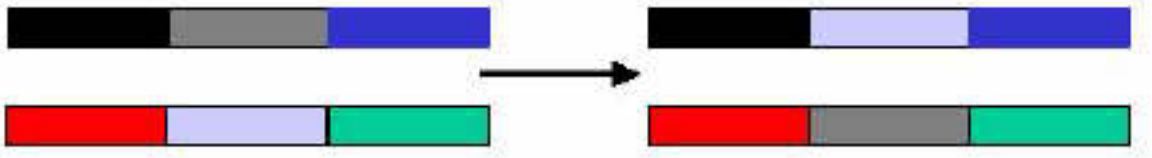
Results of Punnett square for barring	S s^+	S -	$s^+ s^+$	s^+ -
B b^+	S s^+ B b^+	S - B b^+	$s^+ s^+$ B b^+	s^+ - B b^+
B -	S s^+ B -	S - B -	$s^+ s^+$ B -	s^+ - B -
$b^+ b^+$	S s^+ $b^+ b^+$	S - $b^+ b^+$	$s^+ s^+$ $b^+ b^+$	s^+ - $b^+ b^+$
b^+ -	S s^+ b^+ -	S - b^+ -	$s^+ s^+$ b^+ -	s^+ - b^+ -

Since there are 16 interior squares in this Punnett Diagram, there are 16 possible combinations of the four genes. Because these genes are *linked*, the appearance of each combination is not equally probable. See the discussion of linkage below. If the two traits, silver/gold and barring/non-barring had not been on the same chromosome, then we could consider each combination in the Punnett diagram above to be equally probable with the probability being the number of times a specific combination appears in the Punnett diagram divided by the number of squares inside the Punnett diagram.

Adding a third gene pair, as one would have to do to consider shank/foot color (assuming those genes are on different chromosome), is essentially the same procedure just expanded by a third gene pair. This gives 64 total combinations.

Linkage

Genes on the same chromosome are 'linked' and usually inherited together. Two genes that are always inherited together would be linked 100% of the time. However, linkage is never 100% because crossover events occur when the body manufactures sperm and egg cells. The figure below illustrates a crossover event.

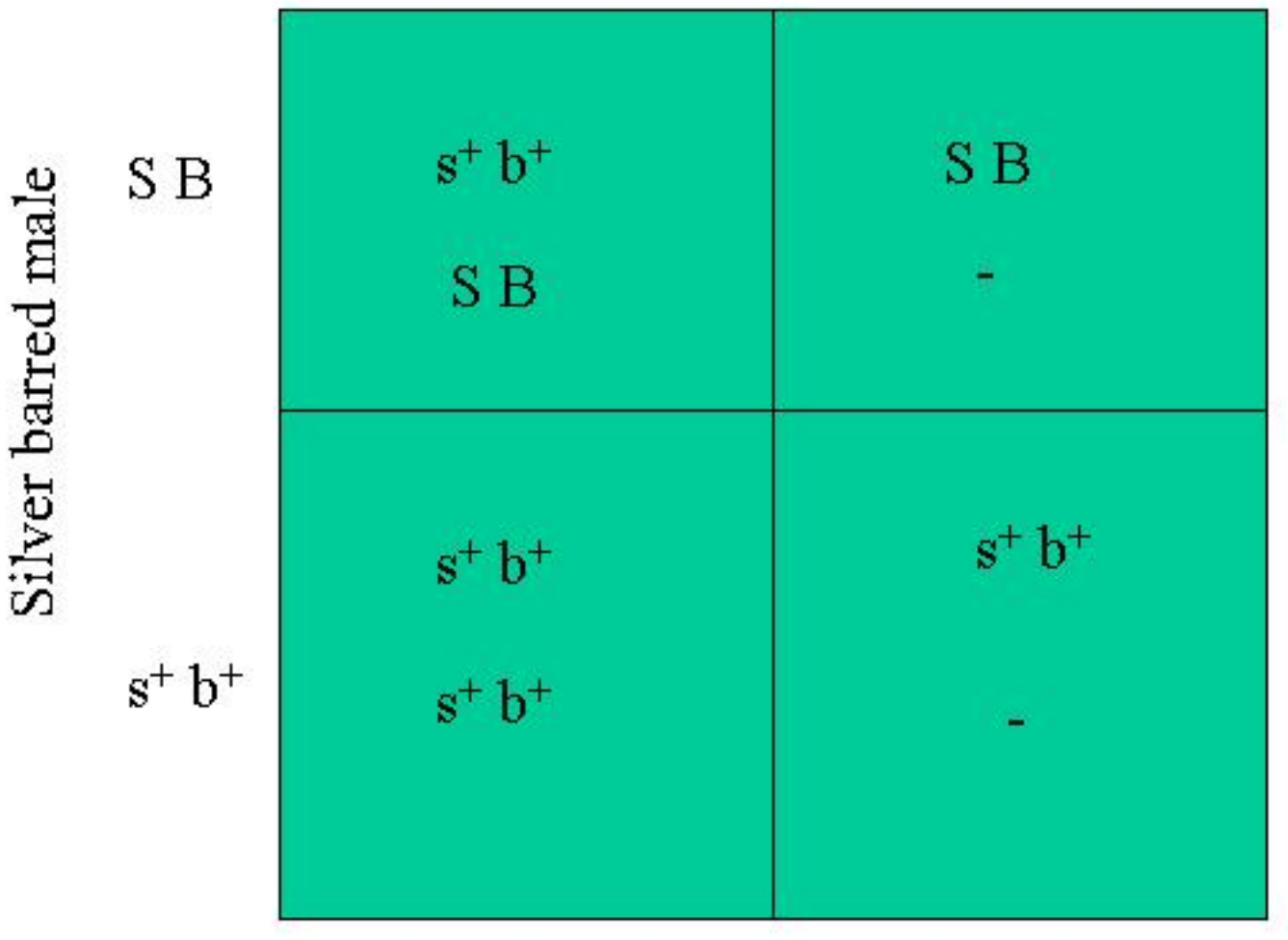


The two bars on the left side represent two chromosomes having three genes each. The genes in the middle 'cross over' during the process of sperm or egg cell formation. The end result is that new 'linkage' relationships exist for the genes on the chromosomes on the right.

Crossover events are actually very common. The rate of crossover events occuring between a gene at locus A and a gene at locus B is proportional to the distance between the two genes on the chromosome (or equivalently, the crossover rate is proportional to the distance between the two loci). A rule-of-thumb for the rate of crossover events in poultry is 1% for every 10 map units in separation between the genes. A map unit is a distance along a chromosome. The actual distance in length units is not really relevant since all chromosome maps are written with distance expressed in map units rather than more familiar units of length.

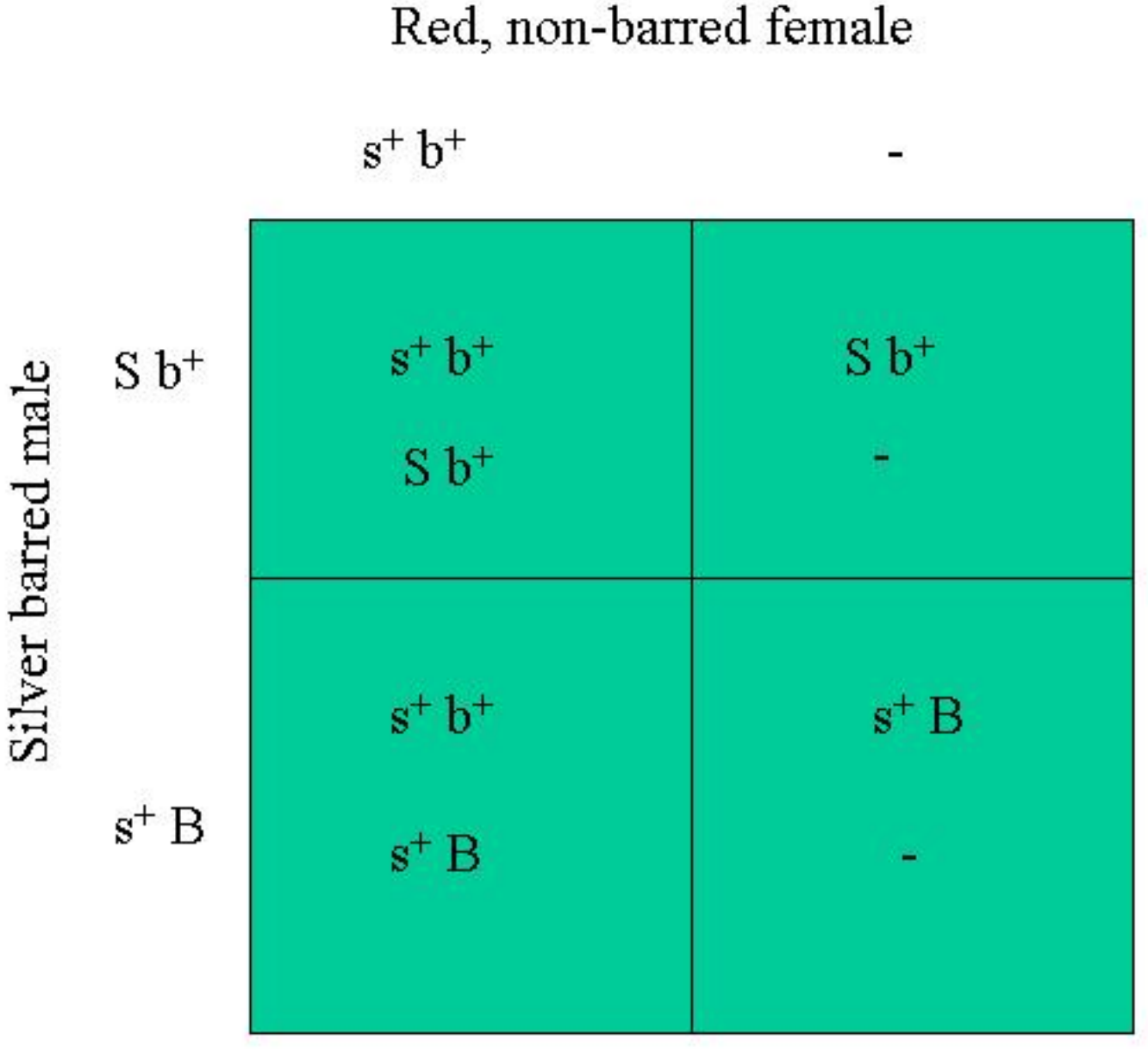
In the Punnett diagram above describing the gene combinations for two traits, silver and barring, the loci of the genes are linked because they are both on the same chromosome, the Z sex chromosome. This means that the wild-type genes, s^+ and b^+ genes of the red and non-barred female will be inherited together most of the time. Occasionally a crossover event will occur and the genes will be inherited separately. So, while the Punnett square above gives all the possible combinations of the four genes, it can not be used to determine the percentages that the gene combinations will appear in the progeny that arise from a cross between a male that is a heterozygote for silver and barring and a red, non-barred female.

Since linked genes are inherited together, they can be treated as single entities in the Punnett square. The Punnett diagram below shows the inheritance of silver and barring in the mating of a red, non-barred female and a male that is heterozygous for both barring and silver. This corresponds to the larger Punnett diagram above, but here I consider that fact that the genes are linked and I treat them as a single object:



The results of this Punnett square (the gene combinations inside the square) are what one would get from the mating *if no crossover events occurred*. The number of times a given gene combination appears inside the Punnett diagram divided by the number of squares in the Punnett diagram is the probability or percent frequency of occurrance of that combination in the progeny. For example, the males are 50% barred and silver and 50% red and non-barred. The females are also 50% barred and silver and 50% red and non-barred. The red, barred male progeny (B b^+ s^+ s^+) that is indicated in the large Punnett diagram above only occurs when a crossover event has taken place, if the parents have the genotype we assumed.

This is an important point, namely that we assumed that the barred, silver male had the B and S genes on the same chromosome. If his genotype had been: Z_1 = B s^+ and Z_2 = b^+ S for his two Z chromosomes, the red barred male would have been present in the progeny without requiring a crossover event. The Punnett square for this mating is:



In this mating, half the males are red and barred.

Inbreeding

There are varied opinions regarding the issue of inbreeding. One school of thought contends that inbreeding is a negative thing and brings about depression in traits such as fertility, hatchability, rate of lay and others. Another school of thought maintains that the negative aspects of inbreeding can be controlled and even eliminated to a large extent through intelligent selection.

Several studies were conducted in the early part of the twentieth century (for a brief synopsis, see Crawford, Elsevier, 1990, Chapter 39) that showed essentially disasterous results when full sibling fowl were mated for several generations. However, even in the first generation of progeny from full sibling matings in these early studies, traits such as hatchability and rate of lay were seriously depressed. These early studies are largely responsible for many people believing that inbreeding in poultry is universally negative.

Other poultry enthusiasts are aware that inbreeding in plants is a very successful strategy in developing hardy strains with desirable traits. They also recognize that most lines of show-quality poultry are inbred. Research performed in the 1970s and later (see Crawford) on inbreeding in chickens (Leghorns), turkeys, quail, pheasants and partridge fowl showed that desirable traits such as rate of lay, hatchability and fertility can be selected for in inbred lines. These traits can recover from the initial depression due to inbreeding, sometimes even to the same level as the non-inbred lines. A 1988 study by Ameli and co-workers showed that long-term selection against the negative effects of inbreeding can be successful in recovering traits such as high rate of lay and fertility in Leghorn populations.

The depression in traits seen in (random, nonselective) inbreeding, such as fertility, hatchability and rate of lay, is often due to recessive genes. If the depression of these traits were due to dominant genes, the depression would be expressed and observed in non-inbred lines and would not be a phenomenon associated with inbreeding. Epistasis or Epistacy (the

interaction of genes at different locations on chromosomes) is sometimes invoked to explain aspects of inbreeding depression.

As of this writing, inbreeding experiments ongoing at the University of Arkansas have associated the greater part of inbreeding depression on hatchability to the male. The evidence for this is the following. Inbred females were mated to a range of different males and the hatchability of their eggs was observed. Inbred males were bred to a range of different females and the hatchability of their eggs were observed. The hatchability of eggs from inbred males was substantially lower than the hatchability of eggs from inbred females, regardless of the cross. So, for example, the hatchability of eggs from a father-daughter cross in which the father is an inbred individual was about the same as the hatchability of eggs from a mating of the same male with non-inbred females. This is strong evidence that the inbreeding depression of hatchability is largely a property of the male birds.

The fact remains that, if the backyard fancier allows inbreeding to take place and does not actively select against the negative effects of inbreeding, the entire population will perform at a lower level with respect to fertility, hatchability, rate of lay and and so on. On the other hand, the objective evidence is convincing that it is possible to develop successful inbred lines of poultry through active selection for desireable traits.

Gametes, meiosis, mitosis

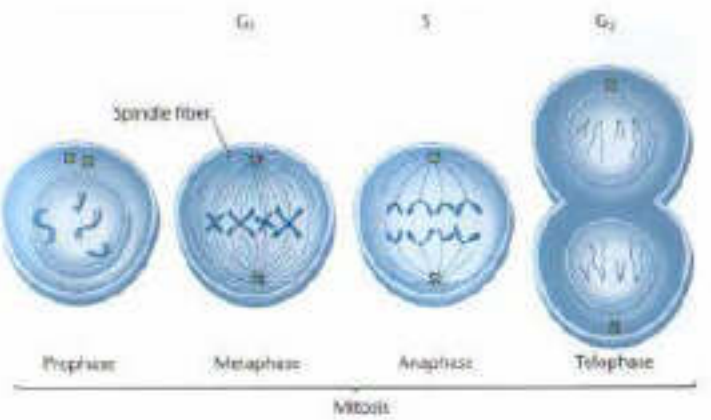
This presentation of genetics tries to limit the use of jargon. However, the interested reader may well want to participate in discussions on the Poultry Genetics discussion board, for example, and will need to know the meanings of some basic terms. Some have already been defined, but others have not.

Cell Differentiation and Reproduction

In the early stages of the development of the embryo, the cells proliferate as they must to grow the early embryo, but they remain essentially identical in that there is no difference among the cells. At a certain point, cells begin to differentiate into specific tissues. Some make heart and circulatory cells, some make kidneys, liver, intestines and so on. What controls cell differentiation is not well understood.

A *gamete* is the 'sex cell'. In other words it is the sperm of the male or the unfertilized egg (ovum) of the female. In general, the gamete has only half the chromosomes of a mature individual.

Mitosis: There are two types of cell division processes. One process, mitosis, is the division of mature cells in the body… cells that have the full compliment of chromosomes (two pairs of chromosomes). The process of mitosis has four identifiable phases:



The prophase is an initial organization phase in which the ‘centrioles’ (small centers from which fibers originate…small yellow squares in the figure above) form and become organized. In the metaphase spindle fibers emminate from the centrioles and attach to the chromosomes. The anaphase is characterized by the separation of the chromosomes by the spindle fibres and the centrioles…they essentially pull the chromosomes apart. In the telophase the cell wall closes and new cells are evident.

Meiosis: The process of cell division that produces *gametes* or ‘sex cells’ (sperm and ovum) . The cells that initiate meiosis contain the full set of chromosomes. However, the process of meiosis yields gamete (sperm and ovum) cells that have half that number of chromosomes. Which chromosomes of the original ones find their way to the gamete cells is essentially a random process. In this process, the chromosomes (of the chromosome pairs of the parents) get mixed or ‘scrambled’ in a random fashion. This is also the point at which crossing over of genes from one chromosome of a chromosome pair to the other chromosome can occur.

Click here on the picture to get to the Table of Contents (frontdoor page) of this site.

II. Discussion of Poultry Genetics

The sex of your chicks:

The sex of a chick is determined even before the egg is fertilized. Each pair of chromosomes in the fertilized egg has one chromosome from each parent. The father always contributes a long sex chromosome (the Z chromosome) to the fertilized egg.

Before the egg is fertilized, it has only those chromosomes from the mother. If the mother contributes a long sex chromosome, Z, to the unfertilized egg, the chick from that egg will be male because it will have two long sex chromosomes after fertilization, since it *always* gets a long sex chromosome from the father. If the mother contributes a short sex chromosome to the unfertilized egg, then the chick will be female because it will have one long and one short sex chromosome after fertilization. So, in this way the egg can be thought of as already having a sex (gender) even before it is fertilized.

The sex ratio of baby chicks:

On the basis of extensive research, it is now accepted as fact that female chicks are equally probable as male chicks. There is no bias toward one sex or the other. Given good incubation techniques, one should hatch equal numbers of male and female chicks if a statistically valid (large enough) sample of eggs is incubated. However, it is believed that female embryos are preferentially killed by fluctuations in incubation conditions.

Feather sexing baby chicks:

In order for rate of feathering to be an indicator of chick sex, the mothers of the chicks have to have a slow feathering gene (see the table) while the fathers have normal feathering or rapid feathering genes. A cross between these males and females will give pullets with rapid feathering and cockerels with slow feathering. This is a sex-linked trait that can be a sex-indicating trait in the same way that sex-linked barring can.

How to breed for a trait for sexing day-old chicks:

In the gene table in Part III the first listing is a set of sex-linked genes. Some common sex-linked traits are Cuckoo barring, gold, silver, slow feathering and dwarfism. Gold, s^+, and silver, S, are allelic,which means that they are found at the same locus (on the long, Z, sex chromosome). In order to breed for a trait that will useful for sexing day-old chicks, the trait must be visible in the hatchling. The brown eye trait is not a good choice because chickens don't get their final eye color until they reach sexual maturity.

To breed a trait that is present in male chicks and absent in female chicks, *the trait must be dominant and on the Z sex chromosome (sex-linked), the female parent must have the trait and the male should be lacking the trait*. Please see the sex-linked genes in the table in Part III. Any of the *dominant* sex-linked genes listed there can be exploited to give birds that are sexable at a very young age. The silver gene, S, is often expoited in varieties like Red Sex-Links for sexing day-old chicks. For example, we might choose to cross a red Rhode Island Red male (s^+, s^+) with a silver Delaware female (S, _) where this means that her long Z chromosome has the silver gene, S, and her short W chromosome is lacking that locus and is represented by an underscore or dash. The four possible gene combinations of the parent genes from this cross are: (S, s^+), (S, s^+), (s^+,_), (s^+,_). Here the dominant gene is written first and any gene is written before the underscore.

In this example of the red male mated to the silver female, there are really only two unique gene combinations since two of the four gene combinations are identical to the other two. The 50% of the chicks that inherit the gene combination, (S, s^+), are silver males (male because they inhereted two copies of the long Z sex chromosome) and are essentially white birds with some possible coloration because silver can be a leaky gene. The other half of the chicks that inherit the (s^+,_) genes are red females (female because she inherited the short W sex chromosome). So the pullets are red and the males are primarily white (yellow down). It is common that Delaware dams and Rhode Island Red sires are used in a cross like this to obtain a Red Sex-Link. This cross is sometimes called Sil-Go-Link for 'silver-gold-sex-link'. The silver gene used this way (the *female* parent having the dominant gene and the male parent having the *recessive* genes), will always give sons that have the dominant gene and daughters that do not.

If the cross is carried out the other way, a silver male on a red female, all the chicks will be essentially white if the male has two copies of the silver gene (homozygous for silver). In this case it is not possible to determine the sex of the day-old chicks by their color.

Any *dominant* sex-linked trait can be used in this way for the purpose of sexing day-old chicks so long as that trait is visible in the chicks. The slow feathering trait can be a good choice because it does not change the basic color or pattern characteristics of the birds (see Part III under Cuckoo barring) so that their appearance (phenotype) can be maintained.

Auto-sexing breeds:

An auto-sexing breed is a breed in which the male and female day-old chicks can be distinguished. Some physical characteristic must be observable that is different in males than females. The important difference between a sex-link hybrid and an auto-sexing breed is that the auto-sexing breed is a pure, true-breeding strain and not a hybrid. Hybrids don't breed true in the sense that phenotypes of male individuals are similar to each other and female phenotypes are similar to each other.

An example of an auto-sexing breed is the Barred Plymouth Rock. In this breed, the auto-sexing property arises from the dose effect that the barring gene, B, exhibits. The B gene is on the Z choromosome so the male Barred Rocks have two B genes while the females have one. The males and females hatch with white spots on top of their heads with the male spot being larger and less sharply defined. Also, the females tend to have darker shanks because the B gene is an efficient inhibitor of shank color. These two traits, based on the B gene dose effect, allow sexing of day-old chicks with a high accuracy rate.

Lethal genes:

Some genes are lethal. A dominant gene that is lethal when a bird has only one of that gene (heterozygous for that gene) is immediately taken out of the gene pool, since no bird survives with it. Some dominant genes are letal only when the bird has two copies of the gene. The creeper gene, Cp and the ear tuft gene, Et, are lethal to a chicken with two copies (homozygous). I am aware of an exception to this in which someone claims to have a male with two ear tuft genes that has survived. This should be considered to be a rare exception. The short leg genes in other breeds are often lethal. Some traits, like frizzleness and rumplessness are known to reduce hatchability but are not explicitly lethal.

Genetics of ear lobe color:

Most breeds have red ear lobes. The red color is due to the blood of the bird and is visible because the skin of the ear lobes, comb and wattles has a rich blood supply that is not masked in any way. These skin areas are so highly vascularized that squeezing a comb between your thumb and forefinger will more than likely squeeze out some of the birds blood onto your fingers. Mosquito bites often leave a small amount of dried blood on the comb. Breeds of the Mediterranean Class (Leghorn, Minorca and Spanish) have 'white' ear lobes.

The white ear lobe is due to the purine pigment which is controlled by a number of genes. The trait is said to be polygenic. The red ear lobe is due to the lack of the genes that invoke the purine pigmentation. Sometimes the white ear lobe can have a greenish or yellowish tinge. The number and location of the genes responsible for white ear lobes is not presently known.

Genetics of eggshell color:

Brown eggshell color is a complex trait and as many as 13 genes have been proposed to account for the range in eggshell color. The white eggshell color is due to an absence of blue and brown, and perhaps some modifying factors (genes), since there are different shades of white. The blue eggshell gene, O, expresses if it is present which is why it is considered to be dominant. The gene symbol for the recessive, wild-type gene is o or o^+. My understanding at present is that the locations of the brown eggshell genes are not known and it is not known how many brown modifying genes there are or where they are in relationship to the genes of known locations. Brown may itself be just an array of white modifiers. There is a recessive sex-linked gene, pr, that inhibits the expression of brown eggshell genes and can be used to help remove the brown tint from white eggs, for example.

The brown pigment, ooporphyrin, is deposited primarily on the outside of the eggshell and is a chemical compound resulting from hemoglobin metabolism. In fact, much of the brown pigment can be buffed off with a common kitchen (plastic) scrubbing sponge and warm soapy water. The blue eggshell pigment, oocyanin, is a byproduct of bile formation and is present throughout the eggshell.

The eggshell color genes interact in the following way. The effect of the blue gene is dominant over white. The effect of the brown gene is dominant over white. When blue and brown genes are both present, both genes contribute to the eggshell color making the eggs appear green. In this case, the inside surface of the eggshell will be significantly less green and more blue than the outside surface, which is where most of the brown pigment is.

Since the blue and brown eggshell color genes should be at different locations, we need at least two pairs of genes to describe the genotypes of the blue, white, green and brown layers. For the purposes of this discussion, I use the fictitious symbol, Br, to indicate a brown eggshell color gene. I represent the complementary recessive gene that takes the place of Br when it is absent as "br" (lack of brown gene). We can represent the genotype of a blue eggshell layer as (O, O) with (br, br). Blue and white genes, (O, o) with (br, br) also yields a blue egg, but perhaps a lighter blue. The pair of eggshell color genes, (O, O) with (Br, Br), are the genes for producing a green egg, (o, o) with (Br, Br) produces a brown egg and (o, o) with (br, br) yields a white egg. Females having one blue gene and one or more brown genes will lay eggs having a greenish color. My personal experience with eggshell color makes me believe that this genetics picture of eggshell color is oversimplified (there are certainly more than one gene for brown eggshell color. In order to account for the wide range of shades of brown eggs we see in our Sil-Go-Link line, there must be a relatively large number of eggshell color modifying genes that are not yet known. Most people accept a rule of thumb to the effect that a daughter will lay eggs that are a color between that of the parent lines.

To explore the genetics of eggshell color, let's cross a green egg layer (faux-Araucana or Easter Egg Chicken) with a white egg layer (Leghorn). Here as before, I will use the fictitous symbol "Br" to represent brown eggshell genes. The genes of the green egg layer are (O, O) with (Br, Br) assuming the locations of the blue and brown genes are not the same. The Leghorn is (o, o) with (br, br) for eggshell color (white). In this example, the daughters will all have one gene for blue eggshell color and one gene for brown. They will all be green egg layers! My personal experience with eggshell color genetics leads me to believe it is more complex than this. There certainly must be a number of brown eggshell genes and once you have them, it is difficult to breed them out completely.

Genetics of comb type:

Comb type in chickens is due to two genes, the rose comb gene, R, and the pea comb gene, P. These two genes are on different chromosomes. The lack of these genes is represented with lower-case letters, r and p. More correctly stated, r and p (or r^+ and p^+ to indicate they are the wild-type genes) are the genes that replace R and P when they are not present. A chicken with a single comb is lacking both R and P genes and so could be represented as (r, r) for rose comb and (p, p) for pea comb. Some authors will combine this 'notation' and write (rrpp) to represent the genes for single comb. I prefer the first way of writing the genes for the purposes of this text.

A chicken with a rose comb will have one of the gene combinations: (R, R) with (p, p), or (R, r) with (p, p). A bird with a pea comb will have (r, r) with (P, P), or (r, r) with (P, p). Since one copy of the rose or pea gene is sufficient for that comb type, these genes can be thought of as dominant. However, they act together to create the walnut comb when both rose and pea comb genes are present.

Poultry with a walnut comb have at least one copy of both the rose comb gene and the pea comb gene. The gene combinations that give walnut comb are: (R, R) with (P, P), (R, r) with (P, P), (R, R) with (P, p) and (R, r) with (P, p).

To explore the genetics of comb type, let's cross a pea comb chicken, (r, r) with (P, p), and a rose comb chicken, (R, r) with (p, p). Because two genes on different chromosomes are involved, there is more bookkeeping than if there were only one gene involved, but the principle is the same and no more difficult. We first have to consider the combinations of the rose comb genes of the two parents, then the combinations of the pea comb genes of the two parents. Then we realize that each of the rose comb combinations can occur with each of the pea comb combinations. In the end there are 16 combinations in all.

The four possible combinations for the rose comb genes from the two parents are: (R, r), (R, r), (r, r) and (r, r). The four combinations for the pea comb genes from the two parents are: (P, p), (P, p), (p, p) and (p, p). Since each of the four rose comb combinations can occur with any of the pea comb combinations, we now have to consider each of the rose comb combinations with each of the pea comb combinations (16).

The figure above shows how to make a helpful drawing. Make a list (column) of the four rose comb gene combinations on one side and the pea comb genes on the other side. The combinations of the first rose comb gene pair with all the pea comb gene pairs is shown in the figure by the connecting arrows. Considering the (A, a) of the drawing to be (R, r), the possible combinations of

the first rose comb gene pair with the pea comb gene pairs are: (R, r) with (P, p) twice [we get this combination twice], and (R, r) with (p, p) twice. The second rose comb gene pair with the pea comb genes gives the same combinations: (R, r) with (P, p) twice and (R, r) with (p, p) twice. The third rose comb gene pair with the pea comb gene pairs gives: (r, r) with (P, p) twice and (r, r) with (p, p) twice. The last rose comb gene pair with the pea comb gene pairs gives the same: (r, r) with (P, p) twice and (r, r) with (p, p) twice.

So, of the 16 possibilities, four of them are (R, r) with (P, p) and is walnut comb, four are (R, r) with (p, p) and is rose comb, four are (r, r) with (P, p) and is pea comb, and four are (r, r) with (p, p) which is single comb. We have four out of 16 chances (25% chance) to get a walnut comb from this cross, four out of 16 chances to get rose comb, four out of 16 chances to get pea comb and four out of 16 chances to get single comb.

Genetics of shank/feet color:

The shank/feet color is controlled by genes that affect the skin at different depths. The visible color is due to the combined effect of the different colors of the dermis and the epidermis. So, the shank/feet colors are a combination of upper skin and deeper skin pigmentations. The following table gives the shank/feet colors that result from the major gene combinations (the bird has two copies of each gene). It is important to remember that other genes can modify shank and foot color. For example, the sex-linked barring gene, B, is a potent inhibitor of dermal melanin. The Barred Plymouth Rocks, for example, would *not* have light shanks and feet if it were not for the fact that they have sex-linked barring. The female Barred Rocks tend to have darker shanks due to the dose effect of the barring gene. The following table is intended as a guide but should not be considered to be absolute, since (as mentioned) other genes, such as sex-linked barring, can modify shank/foot color.

Some Basic Shank/Feet Color Genetics

Shank/Foot Color	Genes
Near black with white soles	W^+, Id, E
White shanks and feet	W^+, Id, e^+
Black shanks, white soles	W^+, id^+, E
Blue shanks, white soles	W^+, id^+, e^+
Near black with yellow soles	w, Id, E
Yellow shanks and feet	w, Id, e^+
Black shanks with yellow soles	w, id^+, E
Green shanks with yellow soles	w, id^+, e^+

Genetics of dark skin color:

The hypermelanic condition of some breeds, such as the Silkie breed, is due to a pigment cell activator, which was named by F. Hutt as "fibromelanosis" to emphasize the fact that the gene causes pigmentation of connective tissue. The inheritance of the dark skin phenotype involves the fibromelanosis gene, Fm, as well as dermal melanin inhibitors, such as the sex-linked Id dermal melanin inhibiting mutation. The fowl with Fm and wild-type dermal melanin, id^+, will have darkly pigmented skin and connective tissue. The combination of Fm and Id gives a bird that has little or no observable skin pigmentation. There are other dermal melanin inhibitors that may have an influence on the degree of melanization due to Fm (or the degree of expression of Fm). Some genes influencing plumage color have an effect on dermal melanin, such as the E-locus alleles, which may influence the expression of Fm. However, fibromelanotic Silkies exist with black, white, blue and partridge patterns.

Genetics of feather color:

The genetics of feather color and patterns is an active topic of poultry science research. Much of the work that was done prior to the late 1980s is now considered out of date. Because a number of genes interact to determine feather colors and patterns, it might seem to be too involved for the average enthusiast. I don't believe that this is the case, however, the topic of feather color and patterns may be beyond the interest and motivation of some enthusiasts.

White is actually all the colors combined and black is the lack of reflection of light in the visible range, so one might argue that black and white are not really 'colors' technically. However, if we count black and white as colors, chickens have only three basic colors: black, white and red (gold).

The colors of chickens are achieved by diluting and enhancing or masking black and red (gold). For example, Rhode Island Reds have the gold gene with the dominant mahogany (red enhancing) gene. A blue chicken is a black bird that has the blue gene which dilutes black. Two copies of the blue gene give a splash effect. A white chicken can be achieved in a number of ways by inhibiting black and red pigmentation with combinations of genes (dominant white, recessive white, silver, Columbian, Cuckoo barring).

Some perceived colors of feathers are due to the structure of the feather and not any pigmentation. The purple and the 'beetle' green sheen that can be seen in some poultry is due to the way the feather structure reflects light rather than the presence of a pigment.

First, we need to define a couple of terms. In poultry there are primary and secondary color patterns. Perhaps it is better to define secondary patterns first. A secondary pattern is a pattern that appears on individual feathers. These are patterns like single and double lace, mottle, and so on. Primary patterns are color patterns that involve the entire body of the bird. An example is the silver Columbian pattern. In the Columbian bird, black is restricted to the hackles, wing bow and tail. The silver Columbian is a white bird with some black in the neck, wing and tail areas. Because this pattern is *not* manifest on individual feathers, it is a primary pattern.

To 'construct' a chicken having a particular color scheme, one begins with the 'background' or the E-locus gene(s). The other color and (secondary) pattern genes essentially modify this 'background'. Please refer to the table at the end and the pattern table below to see the choices and comments (other E-genes have been proposed but they are not yet well accepted). Some of these are: E, extended black or nigrum; E^{R}, birchen; e^{Wh}, dominant wheaten; e^{+}, wild type; brown, e^{b}; speckled, e^{s}; buttercup, e^{bc}; and e^{y}, recessive wheaten. These genes cause recognizable chick down color and influence the adult feather color, sometimes male and female feather colors are influenced differently. For photographs of chicks with an assortment of E-genes the interested reader is directed to *Poultry Breeding and Genetics,* R.D. Crawford, ed., Elsevier, 1990 pages 115-117.

As an elementary exercise, let's 'build' a white chicken. We can start with wild-type background, e^{+}, and require our bird to have two copies of this gene. We can suppress the red in the chicken by adding the silver gene, S, which has the effect of changing red to white. Black is suppressed (changed to white) by the dominant white gene, I, however this gene is 'leaky' (see the table for comments) and allows black specks through. A good 'helper' gene in this situation is the Columbian gene, Co, since it is a restrictor of black. Although this set of genes is not the only set that will yield a white chicken, it is one of the ways a white chicken can be obtained.

Please see the table of chicken genes (Part III) for more comments about plumage color genes.

The influence of one versus two genes for a color trait:

Feather color genes often display a 'dosing' effect: "Two genes are stronger than one." For example, since the locus of the sex-linked barring gene is on the Z sex chromosome, females that have Cuckoo or sex-linked barring (the barring that Barred Rocks have) can have only one barring gene and have barring that is less well defined than the barring of males that have two barring genes. Also, Sil-Go-Link males that have only one silver gene (silver inhibits red) often have some red color on their wings. So, in the Sil-Go-Link male, the one silver gene does not completely inhibit the red pigment. The silver gene is dominant but still some red is visible when only one silver gene is present. This 'dose effect' in which two genes for a trait reinforce or strengthen the expression of a trait is common in poultry.

Genetics of patterns

New research has indicated that several major patterns in chickens are not due to independent genes as previous believed. The single lace, double lace, autosomal barring (horizontal penciling) and spangling are now thought to be due to interactions with the Pg gene (pattern gene). For a review of the original literature, see W.C. Carefoot in Crawford's volume. Single lace pattern can be obtained with the genes: Ml (melanotic), Pg (pattern gene) and Co (Columbian) on either e^{b}, e^{y} or e^{Wh} background genes. Double lace pattern is obtained from the single lace pattern by removing the Co gene. Spangling is obtained with Db (dark brown), Ml (melanotic), Pg (pattern gene) on either the E or e^{b} background. Penciling is obtained with the Pg gene on the e^{b} or e^{+} background. Autosomal barring (sometimes called horizontal penciling) is obtained with the Db and Pg genes. The table below, originally constructed by Brian Reeder, is intended to serve as a guide to the patterns seen in chicken plumage. A similar table is found in *Poultry Breeding and Genetics*, R.D. Crawford, ed., Table 5.3, page 127.

Please see the gene tables (Part III) for more discussion of these pattern genes and others. Also there is an application for viewing

the feather patterns, Link to Feather Images

Chicken Plumage Patterns

Pattern	**Pg**	**Ml**	**Co**	**Db**	**E-locus**	**Hf**
Partridge (or pencilled)	x	/	/	/	e^{b}	/
Salmon-breasted pencilled (female)	x	/	/	/	e^{+}	/
Autosomal barring - Hamburg	x	/	/	x	e^{b}	/
Autosomal barring - Fayoumi	x	/	x	x	E^{R}	/
Autosomal barring - Buttercup	x	/	/	x	e^{bc}	/
Single Lace (as in Wyandotte)	x	x	x	/	e^{b}	/
Single Lace (as in Polish)	x	x	x	x	E^{R} (or E)	/
Single Lace (as in Seabright)	x	x	x	x	E^{R} (or E)	x
Single Lace (as in Andalusian)	x	x	x	/	E	/
Double Lace (as in Dark Cornish)	x	x	/	/	e^{Wh},e^{b},e^{y}	/
Spangling (as in Hamburg)	x	x	/	x	E^{R} (or E)	sometimes

Genetics of eye color:

The genetic basis of eye color has not been extensively studied as have other aspects of phenotype. However, some things are accurately known. First of all, the wild-type eye is characterized by the Light Brown Leghorn. Eye color is a result of pigmentation of a number of structures within the eye (iris, retina, uveal tract, ciliary).

The bay-color eye (various shades of reddish brown) is due to carotenoid pigments and the blood supply of the iris. Brown eyes are increasingly melanized with the darkest eye color due to the fibromelanotic gene characterized by heavy eumelanin deposits throughout the eye. Little is known about pearl eye and Smyth has speculated that it has the same eumelanin distribution as the bay but without the carotenoids.

Eye color is modified by a number of genes that are known to be associated with shank and plumage color. The sex-linked dermal melanin genes, id^{+} and id^{M} enhance dermal shank and eye pigmentation. The inhibitor of shank dermal melanin, Id, also inhibits eye pigmentation. Smyth hypothesized that the id^{M} gene together with extended black, E, is responsible for dark brown eyes. id^{M} also darkens the eye on the e^{+} background.

A dominant sex-linked inhibitor of eye pigmentation is known, Br. This trait is not useful for developing sexable day-old chicks because chickens do not get their final eye color until they reach sexual maturity.

In the absence of other melanin inhibitors, the E-locus alleles, E (extended black) and E^{R}, birchen, result in a brownish eye with the E allele making the darker eye. Sex-linked barring, B, and eumelanin inhibitors at the E-locus, like e^{Wh} have an effect on eye color. Recessive white seems to have no effect on eye color and dominant white, I, has a strong ability to inhibit eye pigmentation. The genetics of pearl are not known, however, it is known that the white skin gene, W, is not the genetic basis of pearl eye, since Cornish have yellow skin and can also have pearl eye.

Genetics of chick down color:

Virtually everyone who hatches their own baby chicks wants to know what the chick down color tells them about the genes of that individual chick. This section is an effort to give some guidance in this direction.

Some Basic Chick Down Color Genetics

Chick Down Genotype	**Phenotypic Remarks**
E, Extended Black	basically black down. Variation includes gray and cream bellies. The cream can invade the head and face. Often Australorps have cream in their faces and heads although the adults are standard black color. Sex-linked barring puts the cream head spot on the black down. Recessive mottling (mo) makes these chicks look like penguins
e^Wh, Dominant Wheaten	Without columbian (Co) Wheaten down is a light cream for both sex-linked silver and gold. One cannot distinguish silver from gold with any accuracy on dominant wheaten. Heterozygotes can have varying amounts of striping. Some New Hamps have light reddish stripes on their backs at hatch. This may be due to heterozygousity or some other modifiers in these lines. With Co wheaten downs are easily sexable. There is a high degree of difference between silver and gold wheaten down when columbian is present. This is why commercial white-tailed reds have dominant wheaten and columbian.
e^+, Wild-type	The dark eye stripe is characteristic of wild-type. The wild-type is often referred to as the 'chipmunk' look. The buff color of the face and back stripes are affected by sex-linked silver and gold. You can easily tell Silver Duckwing chicks from Light Brown Leghorns, but you tend to have trouble in crosses, involving Ss heterozygotes, so there are probably modifiers that affect the gold color of the pure line chicks.
e^b, Brown	Can range from a solid dark mink brown to light brown with stripes. The most accurate typing for e^b is that they have brown heads with no stripes on the head like e^{bc}.
e^{bc}, Buttercup	more yellow than e^+. This dilution may be due to e^{bc} or Db that seems to be in all the crosses and pictures involving this allele. Adult females are like eb females and do not have salmon breasts.
e^y, Recessive Wheaten	Sometimes pictured as being yellow but more brown than e^Wh. Both are said to be cream in color. Recessive wheatens are often called dark wheatens because the adult females have more stippling on their backs than dominant wheaten females usually have.
e^b,Co	Chicks having sex-linked silver in addition to the above genes are cream colored with varying amounts of gray on their backs. Their backs can be nearly black. Sex-linked gold chicks show buff on their flanks and faces and cream bellies with the same varying amount of gray on their backs. The less gray the more buff on the backs of sex-linked gold chicks.
e^b, Co, Db	Mostly light (yellow) body with brown head and back stripe. Buff breeds may have wheaten, Co, and Db.

Some additional genes that affect chick down color are: Dominant and recessive white with extended black at the E-locus gives yellow chick down. If black spots leak through it is usually because the chick is heterozygous for dominant white. Dark prown, Db, makes the black down of extended black to be a reddish brown. Blue or grey chicks can be extended black and Bl (blue) heterozygotes. These chicks can also look black. Chicks that are homozygous for recessive lavender are blue / grey.

A fun exercise:

It is fun to consider the genetic make up of some popular breeds. From what we know at this point about poultry genetics, we can make some judgments about the genes that birds have to have in order to look the way they do.

Let's consider Rhode Island Red as an example. With respect to feather color, the Rhode Island Red (RIR) has gold (on the sex chromosome), s^+ (sometimes called G), and the mahogany (red enhancing) gene, Mh (see the tables below). A Rhode Island Red without any black in her hackle or tail may have a black suppressing gene such as dominant white, I, and Columbian, Co, serves as an additional black suppressing influence. She will have no black extending genes, such as nigrum, which would change her to a black bird. Rhode Island Reds come in both single and rose comb types so she may have at least one copy of the rose comb gene. The single comb RIRs have neither the pea comb nor rose comb genes. RIRs have yellow skin, shanks and feet, which requires two copies each of the w, Id, e^+ genes. RIRs can have either one or two genes (hetero- or homozygous) for eggshell color giving

the brown egg.

White Leghorns are white chickens that lay white eggs. The white could be achieved in a number of ways. The task at hand is to effectively inhibit both black and red. We construct a Leghorn by having two dominant white genes, I, which are black inhibitors, but the dominant white gene allows some black flecks through (see the table at the end) and needs help. Sex-linked barring, B, acts as a black inhibitor in white birds and is used as such in Leghorns. The red inhibitors in Leghorns are the silver gene, S and birchen gene, E^R, or nigrum, E. To get the single comb, the Leghorn has (r, r) and (p, p) for comb genes and she needs (o, o) for eggshell color genes for the white eggs. The Leghorn has yellow skin, shanks and feet which requires two copies each of the w, Id, e^+ genes.

Genotypes of common interest:

Below is a table of partial genotypes for breeds of common interest. There may be other genotypes and so I do not claim that any given breed *must* be exactly the genotype specified. If a gene does not appear in the table, it is intended that wild-type be assumed although sometimes wild-type genes are listed for emphasis. I continue to collect information to update this table....

Partial Genotypes of Breeds of Common Interest

Breed	Autosomal Genes	Sex-Linked Genes	Comments
Australorp, Black	W/W, E/E, co^+/co^+, db^+/db^+, (Ml/Ml), i^+/i^+,(Pg)	S, Id	Evidence of unknown black enhancers and the pattern gene have been observed in Black Australorps.
Silver Spangled Hamburg	E^R/E^R, Co/Co, Db/Db, Ml/Ml, pg^+/pg^+	S, id^+	The combination of dark brown and melanotic may be responsible for the white undercolor.
Silver Laced Wyandotte	e^b/e^b, Co/Co, db^+/db^+, Ml/Ml, Pg/Pg	S,Id	Yellow legged blacks are usually based on the e^b E-locus allele.
New Hampshire	e^{Wh}/e^{Wh}, Co/Co, Mh/Mh, w/w	s^+, Id	With polygenes for red ear lobe and brown eggshell color. A primary difference between New Hampshire and Rhode Island Red is the wheaten allele at the E locus in the New Hamps.
Rhode Island Red	e^y/e^y, Co/Co, Mh/Mh, w/w, Db?	s^+, Id	With polygenes for red ear lobe and brown eggshell color.
Barred Plymouth Rock	E/E, Co/Co, w/w	K, S, B, Id	With polygenes for red ear lobe and brown eggshell color. The slow feathering gene, K, is believed to aid in obtaining a cleaner barring. Barred Rocks have yellow shanks because of the dermal melanin inhibiting property of Cuckoo barring. Without this, the breed would have near black shanks with yellow soles.
White Leghorn	Homozygous for either E, E^R or e^+, I/I, w/w, o/o	B, S, Id	Lines of Leghorns have been found with different alleles at the E locus.

Delaware	e^b/e^b, Co/Co, w/w	B, S, Id	With polygenes for red ear lobe and brown eggshell color. Delawares have Barred Rock and New Hampshire genetics.

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III. Chicken genes and comments

Below is a table of genes and gene symbols. Allelic genes (genes that have the same location or locus on a chromosome, which are also genes that substitute for one another) are grouped together. I have tried to incorporate relevant and interesting comments about these genes in the comment section. I continue to update this and correct it....

Chicken Genes of Common Interest

Sex-Linked Genes (alleles)	Symbol	Comments
Sex-linked barring	B	Barring, cuckoo barring. Dominant. Causes white barring pattern in red and black, sometimes used as a black inhibitor, most notably in Leghorns. Cuckoo barring is also an inhibitor of tissue pigmentation and is responsible for the yellow shanks of Barred Rocks. Shanks of females can be darker. Barring shows a distinct dosage effect. B/B gives wider bars than heterozygotes have. Incorporation of the slow feathering gene results in a cleaner, more sharply defined barring.
	b^+	Recessive wild-type gene. An allele of the sex-linked barring locus. Lack of barring.
Sex-linked dilution	B^{Sd}	Females that are hemizygous for B^{Sd} (having one B^{Sd} gene) have light blue and barred plumage as do the heterozygous males, however, homozygous males show a dosage effect and are essentially white. These homozygous males resemble dominant whites but differ in that they are epistatic to pheomelanin while dominant white is not.
comments		Sex-linked barring, B, sex-linked dilution, B^{Sd} and the wild-type, b^+ are alleles of the same locus. The order of dominance is $B^{Sd} > B > b^+$.
Brown eye	br	Not much is known about this gene and there may be a dominant inhibitor of brown eye. Many of the melanin-influencing genes have an effect on eye color.
Dwarf	dw	Recessive. Males are reduced in size by about 43%, females by 26-32%. Multiple alleles have been proposed. dw is responsible for some beneficial effects. dw homozygotes are more resistant to Marek's Disease and spirochetosis, fewer laying accidents, more aggressive immune response. Abnormal eggs are suppressed (soft-shelled, double yolks). Dwarfism, dw, does not effect mortality but does postpone the onset of lay in pullets up to two weeks. Although egg number and mass are slightly decreased by dw, feed efficiency (feed consumption per egg layed) in laying stocks is usually increased 13-25%.

	dw^B	Recessive but shows a dose effect; 'bantam' gene. Females reduced in size by 5-11% and males by about 5% in heterozygotes and 14% in homozygotes. Allelic with dw.
	dw^M	MacDonald dwarf. Reduces body weight by 13.5% and shank length by 9%. Allelic with dw.
	Dw^+	Wild-type gene. Lack of dwarfing alleles. Allows 'normal' size to develop.
Silver and Red-Gold	S	This gene is called 'silver'. Inhibits red pigment, pheomelanin. The expression of silver is sometimes affected by hormonal levels and is considered to be incompletely dominant and highly influenced by modifying genes.
	s^+	This gene is sometimes called 'gold'. Wild-type, recessive. Invokes red pigment.
Foot Color	Id	Light foot color. Dominant. Inhibits dermal melanin. Reported to have little influence on shank/foot color in birds with dark shanks due to E/E
	id^c	Recessive. This gene allows beak and sometimes plumage pigmentation in dominant white homozygotes.
	id^a	Allows green spots on shanks - this gene is not widely accepted and the effect of this gene may be due to the interaction of modifiers not allelic to this locus.
	id^M	Massachusetts mutation. Recessive. Unlike other alleles that belong to this locus, dermal melanin is present in shanks of day-old chicks. Other alleles take more time to express. The darkest shanks are produced in conjunction with E and i^+. The combination of id^M, E and I produces a pale blue or green color by about three months of age.
	id^+	Wild-type dermal melanin. Lack of dermal melanin inhibitors.
Sex-linked white skin	y	Recessive, causes white skin. Recessive sex-linked white skin causes yolks to be lighter in color and reduces xanthophyll levels in blood plasma. This is generally considered to be an inferior trait particularly since the autosomal white skin does not have these side effects on yolk color.
	Y^+	Wild-type gene. Lack of recessive white skin mutation.
Feathering Rate Genes	k^+	Sometimes called rapid feathering. Recessive.
	K	Late feathering gene
	K^s	Slow feathering gene
	K^n	Very slow feathering or 'delayed' feathering gene. The order of dominance among the genes allelic to this locus is $K^n>K^s>K>k^+$. The slow feathering gene is believed to be associated with a bald patch on the back of the adolescent bird. The feathers do come in given enough time. Since this is likely due to a dose effect of the slow feathering gene, the homozygous males should be the most likely to exhibit the trait. In my personal flocks, I have both males and females exhibiting this. Many novice poultry keepers wrongly attribute the bald back phenotype with a picking problem.
Brown eggshell color inhibitor	pr	This recessive gene results in a lack of protoporphyrin pigment (the brown eggshell pigment) even in hens with polygenic brown eggshell color. It can be employed to remove undesirable tints from eggs of white shelled strains.

Autosomal Linkage Group 1 Genes

Autosomal Genes	Symbol	Comments

Creeper	Cp	Short legged condition. Lethal in homozygous state. Dominant.
	cp^+	Recessive, wild-type gene. Lack of creeper trait.
Rose comb	R	Associated with poor fertility in some homozygous breeds. Dominant.
	r^+	Wild-type gene. Recessive. Lack of rose comb trait.
Lavender	lav	Recessive lavender has been associated with poor feather quality and even lack of feathers in some breeds. Lavender dilutes both black and red; changes black to grey and red to cream. Blue fowls termed "self blue" are normally lavender homozygotes. A mating of two lav homozygotes (blue fowls) will produce blue offspring. Lavender causes dilution by inhibiting the transfer of pigment granules from melanocytes, which produce them, to the feather structure. Lavender expression in homozygotes is present in chicks and adults.
	Lav^+	Dominant, wild-type gene. Lack of lavender trait.

Autosomal Linkage Group 2 Genes

Autosomal Genes	Symbol	Comments
Crest	Cr	Crest feathers are similar in shape and texture to hackle feathers. There may be more than one allele. Incompletely dominant.
	cr^+	Wild-type gene. Lack of crest.
Pied / Mottle	mo (pi)	The pied pattern is recessive black and white as in Exchequer Leghorn. Research has shown that the pied and mottle patterns are due to the mottle gene. It is no longer accepted that 'pied' is a distinct gene from mottle, however it is not known why the mottle gene causes the pied pattern in some birds and the typical mottle pattern in others. Mottle causes a white tip at the distal end (end farthest from the skin) of the feather. Chicks with extended black and mottle (E/E mo/mo) as in the Exchequer Leghorn will often have black restricted from the belly and sometimes the head.
	Mo^+	Wild-type gene. Dominant. Lack of mottling.
Dominant white	I	Incompletely dominant. Influences eye pigment. Inhibits black pigment, eumalanin. This gene is 'leaky' and will allow black specks through. Generally not as efficient at producing a solid white bird as are two copies of recessive white. Heterozygotes of dominant white, I/i^+ are often grey with the grey color visible in the chick down. Dominant white dilutes, but does not eliminate, epidermal melanin.
Smoky	I^S	The smoky gene is an allele belonging to the dominant white locus. Smoky is dominant to dominant white in both chick down and adult plumage in that extended black with I/I^S (E/E I/I^S)results in grey chick down and adult plumage. Research to date indicates that $i+/I^S$ heterozygotes express more the wild-type phenotype with respect to this gene indicating a recessive character with respect to the wild-type. Smoky is dominant on the chick down of $I^S/i+$ heterozygotes in that down that should be black is grey. The melanosomes resulting from the expression of smoky resemble those resulting from Andalusian Blue. Smoky dilutes black much more than red/gold. An important difference between Smoky and Andalusian Blue is that Smoky in the homozygote state produces a grey/blue bird while Andalusian Blue homozygotes are splash. Therefore, Smoky fowl will breed true.
	I^D	This gene is often called 'Dun'. Incompletely dominant, off-white. Allelic with dominant white.
	i^+	Wild-type gene. Lack of dominant white.
Frizzle	F	Incompletely dominant. The action of the frizzle gene is localized in the feather follicle. It causes a structure abnormality in the feather and abnormalities of internal organs (enlarged heart, spleen, gizzard and alimentary canal) are common.

	f^+	Recessive, wild-type gene. Lack of frizzle.

Autosomal Linkage Group 3 Genes

Autosomal Genes	Symbol	Comments
Skin Color	w	Yellow skin color. Recessive.
	W^+	Dominant wild-type gene. Autosomal white skin gene. Prevents the transfer of xanthophyll into the skin, beak and shanks but does not effect the eye iris, egg yolk or blood serum. This gene is considered to be the wild-type because it is present in the Jungle Fowl.
Blue eggshell	O	The action of the blue eggshell gene is dominant to the action of the white eggshell gene, o. Blue and brown egghell genes present simultaneously give a shade of green on the exterior of the egg. The blue eggshell color permeates the shell while brown is primarily an exterior coating.
	o	Recessive wild-type gene. Lack of blue eggshell color gene. Causes white eggshells in the absence of brown eggshell color genes.
Pea comb	P	Dominant. Sometimes referred to as triple comb. Heterozygotes often display a prominant central ridge with much smaller lateral points.
	p^+	Wild-type gene. Recessive. Lack of pea comb.
Naked neck	Na	Incompletely dominant. Turkens. Causes bare skin on the neck which becomes reddish toward sexual maturity. Heterozygotes show a small tuft of feathers on the neck above the crop, which is almost missing in the homozygote. The Na allele is associated with increased tolerance for heat, which is probably due to the 30% reduction in overall plumage for heterozygotes and 40% for homozygotes. Na is also associated with a small increase in meat yield and lower body fat content. An increase in embryonic mortality of up to 10% is attributed to Na.
	na^+	Recessive, wild-type gene. Lack of naked neck. Allows full feathering.
Silkie	h	Recessive. The barbs of the feathers are highly modified giving the silkie a 'woolly appearance.
	H^+	Dominant, wild-type gene. Lack of silkie trait. Allows normal feather structure.
Melanotic	Ml	Dominant. Black intensifyer, one of the genes which, in concert with Pg and other genes, is responsible for plumage patterns. There is speculation that there may be more than one eumelanin intensifying gene similar to Ml and non-allelic.
	ml^+	Recessive, wild-type gene. Lack of melanotic eumelanin enhancing gene.
Pattern gene	Pg	Dominant. This is the pattern gene which, together with other genes is responsible for the patterns of plumage. The pattern gene doesn’t seem to express in the absence of Ml in combination with some of the E locus alleles. See text. The pattern gene with the Db and Co Columbian-like restrictors is believed to be responsible for autosomal barring.
	pg^+	Recessive. Wild-type gene. Lack of pattern gene.
Dark brown	Db	Incompletely dominant. Changes black down of E, E^R to reddish-brown. Adults males exhibit a Columbian-type pattern of black, modifies red to orange-tan. Db is a better restrictor of black in males than females.
	db^+	Wild-type gene. Recessive. Lack of dark brown-type Columbian restriction.

Autosomal Linkage Group 4 Genes

Autosomal Genes	Symbol	Comments
Duplex comb	D^v, D^c	Dominant alleles. The superscripts 'v' and 'c' indicate the 'V' and 'cup' shaped phenotypes and are considered to be separate genes.
	d^+	Recessive, wild-type gene. Lack of duplex trait.
Multiple spurs	M	Dominant. Causes more than one spur per shank on males.
	m^+	Wild-type gene. Recessive. Lack of multiple spur trait.
Polydactyly	Po	Dominant. Having too many toes. The fifth toe develops on top of the first toe and is longer than the first toe. There are several degrees of expression of this gene.
	Po^d	Duplicate polydactyly. Dominant to the wild-type allele. An extra toe is present as well as an elogation and splitting of the original first toe. Extreme expression can accompany this gene in which the most extreme cases the entire foot is duplicated.
	po^+	Wild-type, recessive. Allows normal foot.

Other Autosomal Genes

Autosomal Genes	Symbol	Comments
Autosomal barring	Ab	Non-sex-linked barring. Sometimes called 'parallel pencilling'. This is not a real gene, rather autosomal barring is due to combinations of Pg, Co, Db with e^b, E^R, and e^{bc}. See text.
Breda combless	bd	Recessive. Birds with this gene are almost completely lacking comb and wattles. Females are considered to be completely combles and males have a tiny comb.
	Bd^+	Dominant, wild-type gene. Lack of breda combless trait. It is believed that this gene is necessary for chickens to produce a comb.
Blue	Bl	Incompletely dominant. Andalusian blue-dilutes black: blue pigment is a modified black. Two nigrum genes, E, and one Bl gives a blue chicken; two Bl genes gives splash.
	bl^+	Wild-type gene. Recessive. Lack of blue eumelanin dilution gene, Bl.
Brachydactyl	By	Dominant. Abnormally short digits (toes).
Recessive white genes	c	Thought to give a cleaner white than dominant white. Varieties of White Plymouth Rock, Wyandotte, Minorca, Orpington, Jersey Giant, Dorking, Langshan, Silky and others often carry recessive white genes. Many varieties carry both dominant and recessive white. Allows dark eyes. Pigmentation in chick down varies.
	c^{re}	Recessive white allele that allows red eyes.
	c^a	Autosomal albinism. Alellic with the recessive white genes. Evident via lack of eye pigment. Some melanin present in chick down.
	C^+	Wild-type gene. Dominant. Lack of recessive white mutations.
	Comments about the C locus	The order of dominance among the recessive white alleles is: $C^+ > c > c^{re} > c^a$. The presence of other pigment inhibiting or enhancing genes will influence the chick down color. Some adults have a grey color.

Champagne blond	Cb	Dominant. Inhibits pheomelanin (red / gold). The presence of the gene is not observable on the wild-type down
	cb^+	Wild-type gene. Recessive. Lack of chanpagne blond dilution.
Columbian	Co	Incompletely dominant.Confines black to hackle and tail in both sexes (called Columbian restriction). Thought to cause a gradient in color from head to tail. Modifies Wheaten to Buff Columbian. Has no effect on extended black, E.
	co^+	Wild-type gene. Lack of Columbian restriction. Recessive.
Red diluter	Di	Dominant. Dilutes red, changes red to buff.
	di^+	Wild-type gene. Lack of red diluter. Recessive.
Dark	Dk	A proposed gene of an allelemorphic series that darkens the shade of red. Pheomelanin enhancer(s). Dk^l was proposed for the dark brown Leghorn and dk^+ for the wild-type allele.
Ginger	Gr	This gene may be Columbian, Co, or closely related. This may not be a distinct gene.
Grey	no symbol	Recessive, dilutes black to brown/grey.
The E-locus alleles	E	Often called 'extended black', 'nigrum' or 'self black'. Extends black, changes red to black, red inhibitor.
	E^R	Birchen. Resembles extended black, E, but with non-black breaks on head and hackle. Body is black with some stippling (flecks/dots) of other color. Used as red inhibitor in Leghorn.
	e^b	Partridge (brown). Sometimes represented as e^p, females have non-salmon breast with stippling. Males are wild-type.
	e^{Wh}	Dominant wheaten. Female body varies from light salmon to wheat color, some black may be present. Males are wild-type.
	e^+	Wild-type. Female: breast is salmon brown and devoid of stippling, body is black and brown in stippled pattern. Males: black breast and abdomen; non-black hackle, saddle and wings.
	e^s	Speckled. Resembles e^b but with less pronounced stippling. Males are wild-type.
	e^y	Recessive wheaten. Female: resembles dominant wheaten with more coarse black stippling on breast and back. Males are wild type.
	e^{bc}	Buttercup allele. Resembles the e^b phenotype.
	Comments on E-locus alleles	The order of dominance among the generally accepted E-locus alleles is: $E>E^R>e^+>e^b>e^s>e^{bc}>e^y$. The birchen allele is incompletely dominant to dominant wheaten and the wild-type alleles. Additional alleles have been proposed for the E-locus but research to verify these as separate alleles has not been done. As of this writing, the buttercup allele has been sequenced and has been found to be the same sequence as the e^b allele. The buttercup phenotype then is due to modifiers or interactions with other genes. Every E-locus allele influences adult female phenotype. However, all the adult male phenotypes are the same as wild-type except for extended black and birchen.
Ear tuft	Et	Dominant. Lethal in homozygous state. Thought to be associated with birth defects, particularly in the ear structures.
	et^+	Wild-type gene. Recessive. Lack of ear tufts.
Erminette	no symbol	Black spots and flecks, variable black and white feathers, similar to pied.

Fibromelanosis	Fm	Dominant. Sounds like a disease. The name was suggested by F. Hutt in the 1940s to emphasize the association with connective tissue pigmentation. This gene is responsible for the deep skin pigmentation of silkie. Fm is strongly influenced by dermal melanin inhibitors such as the sex-linked Id mutation.
	fm^+	Wild-type gene. Recessive. Lack of fibromelanosis.
Long tail	Gt, mt	The Gt gene (dominant) allows continual growth of tail and saddle feathers. The mt gene allows certain tail and saddle feathers to be nonmolting.
Henny plumage	Hf	Dominant. The term comes from 'hen feathering' in which male plumage is indistinguishable from female plumage.
	hf^+	Wild-type gene. Recessive. Lack of henny feathering.
Cream	ig	Dilutes red. Recessive. A major pheomelanin dilution gene. The gene symbol derives from "inhibitor of gold".
	Ig^+	Wild-type gene. Dominant. Lack of cream dilution.
Lace	Lg	This is not a real gene. See text. Partridge Rock, Silver Pencilled Rock.
Beard-Muff	Mb	Incompletely dominant. Characteristic of Ameraucana, Easter Egg Chickens (faux-Araucana)
	mb^+	Wild-type gene. Recessive. Lack of beard-muff.
frizzle modifyer	mf	Recessive. Reduces/modifies the effect or expression of the frizzle gene. This gene can modify frizzle heterozygote expression to the point that they are almost indistinguishable from the wild type. Modifies the extreme expression of the frizzle homozygote.
	Mf^+	Wild-type gene (uncertain). Dominant. Lack of frizzle modifyer.
Recessive melanotic	mi	Enhances black, (helps) change red to black. E + mi gives a black chicken.
	Mi^+	Wild-type gene. Dominant. Lack of recessive melanotic enhancing.
Mottle	mo	Recessive. Makes a white tip on end of feather. Changes a black bird to Mottled and a Buff Columbian to a Mille Fleur. Dilutes epidermal melanin. There may be several alleles corresponding to this locus or non-allelic modifying genes.
	Mo^+	Wild-type gene. Dominant. Lack of mottling.
Mahogany	Mh	Dominant. Mahogany restricts eumelanin and enhances the color of red. Rhode Island Red is a good example. Restricts black in the back and wing of both males and females. Down color seemes to be unaffected by mahogany.
	mh^+	Wild-type gene. Recessive. Lack of mahogany.
Pink-eye dilute	p^K	Dilutes both feathers and eye color. Recessive.
Recessive polydactyly	po-2	Recessive. A number of extra toes can be present even ascending the shank. Associated with leg deformities, significant decrease in hatchability and much higher post-natal mortality.
		Dominant. Two different feathered leg loci with perhaps four alleles for the Pti-1 locus (Pti-1, Pti-1B, Pti-1L and $pti\text{-}1^+$: one should always assume the wild-type allele although not always mentioned). Research has shown that the Pti-1 and Pti-2 genes are most likely not allelic (they belong to different loci of the chromosome). When both Pti-1 and Pti-2 alleles are present, heavy

Feathered legs	Pti-1, Pti-2, Pti-1B, Pti-1L	feathering as in Cochin, Sultan, Belgian d'Uccle results. If only one is present, the feathering is weaker as in Langshan, Faverolle, Breda. These genes demonstrate a dose effect. Regarding the Pti-1B and Pti-1L genes, the following is from Somes' 1992 paper in Poultry Science: "The Langshan and Brahma breeds were both shown to possess the same single shank-feathering locus, but because of their differences in phenotype and penetrance in the genetic crosses it was suggested that they possessed different alleles at this locus. This locus was designated as Pti-1, with Pti-1L being the Langshan allele and Pti-1B the Brahma allele. The Brahma allele was shown to be dominant over the Langshan allele. Both the Sultan and Cochin breeds were shown to possess two shank-feathering loci, and the data suggested that one of the loci in the Sultan contained the Pti-1L allele. It is hypothesized that the comparable allele in the Cochin breed was Pti-1B. It is proposed that the second locus in both of these breeds is similar, and the symbol Pti-2 is suggested."
Recessive feathered legs	pti-3	The recessive leg feathering gene was identified in a Russian breed referred to as the Pavlov breed. Test matings confirmed the recessive nature of this gene.
Dominant Rumplessness	Rp	No coccyx (tail vertebra), reduces hatchability.
	rp^+	Wild-type gene. Recessive. Lack of dominant rumplessness. Fowls usually have tails.
Recessive Rumplessness	rp-2	A skeletal mutation commonly called 'roachback'.
Red spash white	rs	Recessive. Two copies of this gene give a white bird with spashes of red and black. Chicks are white with a red head spot. This gene may be extinct now. It was first isolated in a line of Rhode Island Reds, but it was not maintained nor has it be re-identified.
Recessive black	sg	Not much is known about this gene. Eumelanin intensifier. There may be a number of genes that play this role.
Spurlessness	sl	Recessive. Fowls have no spurs.
Snow-white down	sw	Recessive. The chick down is white rather than yellow.
Vulture hocks	v	Recessive. Long and stiff feathers on the posterior area of the tibia. Characteristic of Belgian Bearded d'Uccle, Breda, Sultan.
Dorking white	wh	Recessive.
Woolly	wo	Recessive.

Glossary

Allele One of a number of possible alternative forms of genetic information at a gene locus [1]. *A member of a set of genes that all have the same location on a given chromosome. For example, extended black and birchen are allelic genes (to each other) because they are both found at the E locus or location.*

Epistasis (also epistacy) suppression of the effect of a gene by a nonallelic gene [3]. *A gene for trait A somehow having an effect on triat B is an example of epistasis.*

Gene The unit of heredity [1]. *A piece of DNA in a chromosome that contains the coded information for a trait.*

Generation notation: The original members of a mating are referred to as the parental (P) generation. The first generation of progeny from the parental cross is referred to as the first filial generation, F1. The progeny of a cross in which one or both of the parents are from the F1 generation is an F2 generation (F1 x F1 = F2) and so on [1].

Heterosis The deviation between the cross and midparent means [2]. *The difference in some property, for example rate of lay, between a cross bred line and the average for the parent lines. For example, "hybrid vigor" can be thought of as an effect of heterosis.*

1. J.J. Pasternak, *An Introduction to Human Molecular Genetics*, *Mechanisms of Inherited Diseases*, Fitzgerald Science Press, Bethesda, MD 1999.

2. R.W. Fairfull in *Poultry Breeding and Genetics*, R.D. Crawford, ed., Elsevier, 1990, page 913.

3. Webster's Seventh Collegiate Dictionary

Websites:

Poultry Genome Project http://poultry.mph.msu.edu/

Poultry Genetics Board http://www.insidetheweb.com/messageboard/mbs.cgi/mb55754

UCDavis http://animalscience.ucdavis.edu/extension/avian/pfs1b.htm

MSState http://www.msstate.edu/dept/poultry/avianemb.htm