TOP 10 GENETIC MYTHS IN AVIAN GENETICS

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Introduction

Not surprisingly, and despite innumerable benefits, aviculturists continue to find it daunting to explore the principles that explain heredity. Even the word, "genetics" may cause some to inwardly cringe and shudder. Yet, genetic principles are crucial to our understanding, because knowledge of genetics can empower aviculturists with the ability to produce healthier strains of viable breeding stock and future generations of robust species. When correctly bred, breeding lines pass on such set qualities as increased egg production, higher fertility, strong immune systems - and when properly applied - the ability to produce primary and rare color mutations to order. An understanding of simple Mendelian rules and other modes of inheritance need not be complicated, and many models, once understood, are surprisingly simplistic.

Although our first inclination is to apply genetic principles to outward color mutations and patterns in birds, the laws of genetics equally pertain to all the traits inherited in a bird's genotype. When applied correctly, the laws of genetics allow us to produce large, saleable baby birds raised by fertile parental generations who successfully incubate, feed, and wean their own babies, and also allows us to show the most exquisite bird to top perfected show standards. In other words, the principles of heredity affect every inherited trait - both visible and invisible - in a bird's genotype or full genetic makeup.

Many of us learn avian genetics from our own hands-on work "in the field" (i.e., the aviary), and from information gathered and contained in our own studbooks, rather than through formal study. Our budding knowledge and gradual absorption of facts over time contribute to the subtle and seemingly effortless process of learning through osmosis. Admirable. Except that even the most successful aviculturist may not possess the text book knowledge to separate scientific fact from popular misinformation. We pay attention to what is written on the internet, or passed on as good advice by the well meaning, to sometimes discover that what is stated from "experts" may not be as factual as hoped for. The following attempts to address some of the top ten myths that are still perpetuated today in avian genetics so that we can side-step these errors.

Genetic Myth #1: Mutations are synonymous with hybrids. False!

There is still much confusion around the differences between breeding color mutations within a species, and producing hybrid offspring between two different species.

For clarity, the term "mutation" refers to changes in the outward color or pattern of a bird. So, what are color mutations? Mutations may be caused by a variety of factors, but generally arise in aviculture as a sudden onset of a variation known as a spontaneous mutation which produces a new, anomalous color or pattern that differs from the nominant (wild form) of the species. Some spontaneous, partial mutations - if not fully developed - may require a planned, selective breeding program requiring several generations to achieve the full maturation of the final mutation.

Viable mutations are able to reproduce because the genes - when inherited in the progeny - are either passed on as visuals (homozygous), or carried as splits (heterozygous), depending upon the full genotype of the parental generation.

Producing hybrids, on the other hand, requires mating a bird of one species to a bird of a different species. For example, breeding Red-lored Amazon parrots to Green-cheeked Amazon parrots would result in offspring that are neither 100% genetically Red-loreds (Amazona autumnalis), or 100% Green-cheeked Amazons (Amazona viridigenalis). Instead, the two different species would produce hybrid offspring that would not be recognized as either species - autumnalis or viridigenalis.

On the other hand, breeding a Red-lored Amazon parrot to a Lutino mutation Red-lored Amazon parrot, would produce all Red-lored Amazon parrots. In other words, all resulting offspring - whether nominant (wild colored) Red-lored Amazons, or Lutino mutation Red-lored Amazons, would all be the same species - 100% Red-lored Amazon parrots (Amazona autumnalis).

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Therefore, generally speaking, when examining a new color or pattern mutation, we are looking at an example of a color mutation in the nominant wild species. We are not looking at the offspring of dissimilar species, but rather where both parents are the same species. An example of a color mutation would be a Blue Indian Ringneck Parakeet, bred from Indian Ringneck parents. An example of a hybrid would be offspring from dissimilar species, e.g., one Indian Ringneck Parakeet parent and one Alexandrine Ringneck parent.

Although color mutations are encouraged and sought out by many aviculturists, most avicultural organizations frown upon the production of hybrids for an important reason. Producing hybrids and breeding their successive young will dilute the limited gene pool of stock available for the species involved. To continue to do so may affect the future availability of species. Whereas, producing color mutations is simply changing the color and/or pattern

of the species outwardly and in no way affects their future establishment in captivity when bred responsibly.

Genetic Myth #2: Color mutations, compared to the nominant species, are inherently weak. False!

Mutations are a permanent change in the DNA sequence of a gene affecting the genetic information of a hereditary characteristic or trait. DNA in a gene can be caused to mutate by two major occurrences. A mutation can be acquired due to environmental effects such as radiation or other agents, or when errors occur when a cell copies its DNA prior to the cell dividing.

Mutations can be inherited randomly from one or both parents. If a parent has a mutation in its DNA, that mutation is then passed on to its offspring. Mutations occur more frequently in captivity than in the wild due to such factors as a more limited gene pool, the increased chance of survival, and possibly an increase in genetic drift. For example, this may be why we are beginning to find more color mutations occur with the increase of producing some of the larger Psittacine birds compared to decades ago.

Although established mutations can be purchased and bred to reproduce in the aviary, new mutations occur randomly and cannot be deliberately created, but rather, happen by chance.

A common misconception is that color mutations, when compared to the nominant (wild) species, are inherently weak. Although there are a few autosomal recessive mutations in aviculture that have had inherent difficulties, it is not a given that new color and pattern mutations will be associated with problem outcomes or lethal genes.

One example of positive intervention occurred with the supposed original emergence of the (autosomal) Recessive Silver Cockatiel in Europe during the early 1960s, when it was first reported to have produced chicks that were born blind. While first breeding cockatiels in the mid-1970s, the author became aware of Recessive Silvers imported from Europe, some few years later. These birds, however, did not have the inherent blindness or weakness of the earlier birds and were, in fact, quite healthy. It appears the lethal genes were either selectively bred out, or equally likely, as rumor had it, a new strain of Recessive Silvers were developed, one which did not carry any lethal factors.

Whenever another gene or allele becomes available, the one easiest to work with often finds favoritism. For example, in exhibition Budgerigars, once the Australian Dominant Grey color mutation became available, which appeared identical to the recessive Grey, interest waned in working with the English Recessive Grey that required a minimum of two generations to produce visuals. Aviculturists naturally favored the Australian Dominant Grey, which would produce visuals in half the nest in the first generation. These Dominant Greys, carrying large

size, good head qualities, and other coveted show characteristics often found their way to the top of the show bench. It was not unusual to find the (nominant wild) Normal Greens, Dominant Greys - and the cross – Grey-greens, to make up the top ten bench at many exhibition budgerigar shows.

There are instances of weakened ino (i.e., Lutino or Albino) mutations that have been labeled as defective in some way, e.g., weak, inferior, "poor doers," and who inherit a multitude of problems from eyesight difficulties to immune compromised defenses. Although some birds may be affected initially in some species, aviculturists have proven that line-breeding and affective outcrossing can breed out problems initially effecting a line of birds.

In fact, there are quite a number of robust, healthy inos frequently seen at bird shows, including many winning top bench ino mutations, and champion pedigreed inos who keep winning top awards, reproducing winning progeny. As an example, while it had been common to see only Normals win a best in show in many parrot species divisions, it is not unusual today to find mutations included among the top bench birds.

Unfortunately, new bird owners may not always select healthy birds from strong bloodlines, or be educated on how to provide adequate nutrition, exercise space and optimum management practices when beginning to keep and breed birds. Such practices can and do affect gene pools and future generations of stock. Any bird, whether of the nominant wild coloration, or a color mutation, will only be as hardy as its genes and the breeding program employed from which it originated. If a bird comes from healthy well-bred stock, produced from proven bloodlines (that are not necessarily limited to exhibition stock), then the result will be a more hardy bird.

Even show breeders know the old genetic adage, "like produces like." If you start off with problem birds (e.g., highly inbred, compromised stock, etc.), then that is what you will produce: weak, sickly birds. The laws of genetics apply to any bird, whether it is the nominant wild color or a variant color mutation.

Occasionally, some new mutations, especially autosomal recessives, can be linked to lethal factors. However, the responsible breeder will work with the line establishing a viable, healthy strain, before ever attempting to let go of such stock.

As aviculturists, producing healthy bloodlines is our responsibility. And this responsibility is not merely limited to producing color mutations, but includes any

TABLE 1

SEX-LINKED INHERITANCE

Cock	Hen	Progeny
1. Sex-linked	Non-linked	= 50% Non-linked/Sex-linked cock 50% Sex-linked hens
2. Sex-linked	Sex-linked	= 50% Sex-linked cocks 50% Sex-linked hens
3. Non-linked	Sex-linked	= 50% Non-linked/Sex-linked cocks 50% Non-linked hens
4. Non-linked/Sex-linked	Non-linked	= 25% Non-linked/Sex-linked cocks 25% Non-linked cocks 25% Sex-linked hens 25% Non-linked hens
5. Non-linked/Sex-linked	Sex-Linked	= 25% Non-linked/Sex-linked cocks 25% Sex-linked cocks 25% Sex-linked hens 25% Non-linked hens

Key: Where "Non-linked" is Normal (the nominant wild form), or any other color that is not sex-linked. This table may be used to check the results of individual sex-linked mutations, one at a time. While the charts demonstrate the graphing of several sex-linked mutations together (with other colors or modes of inheritance), each sex-linked trait may be double checked, one at a time, in the above table.

Example: Non-linked/Sex-linked cock x Non-linked hen

P1 XL X | X Y Normal/Lutino cock (XL X) | Normal hen (XY), where L = Lutino

XL X | XL Y 25% Normal/Lutino cocks | 25% Lutino hens

X X | X Y 25% Normal cocks | 25% Normal hens

Note: It is easier to understand why a hen can never be split (heterozygous) to a sex-linked trait when looking at a chart. As a hen's sex chromosomes (XY) have only one "X", the only possibility is to be visual for the trait (XL Y), or not (XY). This is because hens do not have a second "X" sex chromosome to cover or hide a recessive sex-linked trait on a companion "X" sex chromosome. Additionally, it is believed that the "Y" sex chromosome is too small to carry locus sights (designated addresses) for sex-linked color traits to reside.

TABLE 2

DOMINANT X RECESSIVE MATINGS

P1 Cock	x	Hen	=	F1 Progeny
1. Dominant	x	Dominant	=	100% Dominant
2. Dominant	x	Dominant/Recessive	=	50% Dominant 50% Dominant/Recessive
3. Dominant	x	Recessive	=	100% Dominant/Recessive
4. Dominant/Recessive	x	Dominant/Recessive	=	25% Dominant 50% Dominant/Recessive 25% Recessive
5. Dominant/Recessive	x	Recessive	=	50% Dominant/Recessive 50% Recessive
6. Recessive x Recessive			=	100% Recessive

Example Budgerigars::
Where GG = Light Green; Gb = Light Green/Sky Blue; and bb = Sky Blue

Model: 1,2 3,4 (High School Algebra "FOIL" METHOD = First, Outer, Inner,Last)

1,3
1,4
2,3
2,4

1. GG GG
GG
GG
GG
GG
100% Normal Green (GG)

2. GG Gb
GG
Gb
GG
Gb
50% Normal Green (GG)
50% Normal Green/Blue (Gb)

3. GG bb
Gb
Gb
Gb
Gb
100% Normal Green/Blue (Gb)

4. Gb Gb
GG
Gb
bG
bb
25% Normal Green (GG)
50% Normal Green/Blue (Gb, bG)
25% Blue (bb)

5. Gb bb
Gb
Gb
bb
bb
50% Normal Green/Blue (Gb)
50% Blue (bb)

6. bb bb
bb
bb
bb
bb
100% Blue (bb)

Key to above table
*White includes Whites of Suffusion (i.e., White Sky Blue, White Cobalt, and White Mauve) that act as double recessives.

**Unknown refers to not enough data has been reported by breeders, or colors do not yet exist.

stock we work with, be it a family line, strain, or stud of birds. The genetic modes of inheritance such as those employed with producing color mutations can theoretically be applied to the many valued traits in the establishment of all species, e.g., health, fertility, longevity, resistance to disease, and other important traits we wish to preserve.

Genetic Myth #3: All chromosomes are alike. False!

Chromosomes are found inside the cell and can be thought of as a single coil of DNA containing the genes or heredity of life. While chromosomes always travel in pairs, in most instances they are identical, with the exception of one pair of chromosomes called the sex chromosomes.

Because most of the avicultural literature assigns sex-linkage in terms of "X" and "Y" chromosomes, rather than the scientific notation written as "Z" and "W," this column will continue with the "X" and "Y" notation most aviculturists are accustomed to. While the sex chromosomes known function is to determine the gender of the offspring, and as we are reminded that chromosomes always travel in pairs, we find that the sex chromosomes of cock birds are identical (XX), but differ in the hen (XY).

When an egg is fertilized, each offspring will inherit one sex chromosome from each parent. The cock will always donate an "X" from his "XX" sex chromosomes; the hen will donate either an "X" chromosome, or a "Y" chromosome from her "XY" sex chromosomes (with an equal chance of donating either an "X" or a "Y" chromosome).

If the cock's "X" sex chromosome unites with the hen's "X" sex chromosome to form "XX," the offspring produced will be male. If the cock's "X" sex chromosome unites with the hen's "Y" sex chromosome, the offspring produced will be female. Therefore, unlike mammals, in avian species it is the female who determines the gender of the offspring by whichever sex chromosome she donates to the union.

Looking at the remainder of the chromosomes, we assign the term autosomes to all chromosomes, with the exception of the

one pair of sex chromosomes. Autosomes carry a multitude of genes, including genes for color mutations, which we refer to as autosomal. The difference between autosomes and sex chromosomes is important, because, as we will see, while hens can never be split (heterozygous) to sex-linked colors or traits, hens may be split (heterozygous) to autosomal recessive colors and traits.

Genetic Myth #4: Hens may be split (heterozygous) to sex-linked traits. False!

At present, it is theorized in sex-linked avian mutations that only the "X" chromosome is sufficiently large enough to carry the genes for sex-linked recessive traits. The "Y" chromosome, being significantly smaller, has no locus (e.g., "address") sites available for such color alleles to reside. It becomes increasingly clear when charting sex-linked inheritance why hens cannot carry sex-linked recessive colors in hidden form, as do their male counterparts **(Table 1)**.

Because the hen's "Y" chromosome is too small to carry locus sites for color alleles, and at the same time is unable to cover, or mask, the color alleles on the "X" sex chromosome, the recessive genes on the "X" sex chromosome are revealed and must show themselves. This is why hens (XY) show sex-linked recessive colors more frequently. They need only carry the mutation on their one "X" chromosome for the mutation to be visible.

Therefore, whenever working with sex-linked colors in hens, the old adage, "what you see is what you get," could never be more true. If you can't see it, it's just not there. However, this statement only applies to sex-linked recessive traits, because hens may certainly be split (heterozygous) to autosomal color mutations that work under different rules of inheritance.

Genetic Myth #5: A bird may be a "dominant recessive" color mutation. False!

Unfortunately, this myth has become a common misconception and does not need to be. To the best of the author's knowledge,

TABLE 3

HIERARCHY OF RECESSIVES

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Note: This table, composed by the author, demonstrates the hierarchy of autosomal recessive mutations and does not include sex-linked, co-dominant, or intermediate dominant mutations.

BUDGIES (and a general guide in parrots that produce similar psittacin pigments):

Color	Dominant to:	Recessive to:
Normal Green	All Colors	None
Dilute Green (e.g. Greywing)	Blue in all Suffusions	Normal Green
Pastel Green (Yellow)	Blue in all Suffusions	Normal Green Dilute Green
Normal Blue	All Blue Suffusions	All Green Suffusions
Dilute Blue (e.g. Grewing Blue)	Pastel Blue (White)*	All Green Suffusions Normal Blue
Pastel Blue (White)	None	All Green and Blue Suffusions

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Example using Normal Grey, Pastelface, Recessive Silver and Whiteface

Color	Dominant to:	Recessive to:
Normal Grey	All Colors	None
Pastelface	Whiteface	Normal Grey
Recessive Silver	Unknown**	Normal Grey
Whiteface	None	Normal Grey Pastelface

Key to above table

*White includes Whites of Suffusion (i.e., White Sky Blue, White Cobalt, and White Mauve that act as double recessives.
**Unknown refers to not enough data has been reported by breeders, or colors do not yet exist.

TABLE 4

SINGLE AND DOUBLE FACTOR DOMINANT INHERITANCE

1. Singe Factor	x	No Factor	=	50% Single Factor 50% No Factor
2. Singe Factor	x	Singe Factor	=	25% No Factor 50% Single Factor 25% Double Factor
3. Single Factor	x	Double Factor	=	50% Single Factor 50% Double Factor
4. Double Factor	x	No Factor	=	100% Single Factor
5. Double Factor	x	Double Factor	=	100% Double Factor

Key: "No Factor" represents a Normal, or any other mutation which is not governed by Single and Double Factor Dominance inheritance.

Example: Cockatiels: Single Factor x Single Factor = 1:2:1 ratio with equal chance of either gender.

P1 Single Factor Dominant Silver cock x Single Factor Dominant Silver hen

= F1 25% No Factor (e.g., unaffected)
50% Single Factor Dominant Silver
25% Double Factor Dominant Silver

a gene cannot exist in both a dominant mode of inheritance and a recessive mode of inheritance, concurrently. It is either one or the other **(Table 2)**. A gene may, however, interact with other genes or alleles in a variety of behaviors, depending upon their mode of inheritance. This has more to do with allelic forms of genes occupying a locus site, which is beyond the scope of the present article.

In this example, however, it may be helpful to think in terms such as single, simple recessives, and the less powerful double recessive. The confusion appears to stem from the lack of understanding that while an autosomal recessive color is recessive in its mode of inheritance, it may simply behave in a dominant (or commandeering manner) whenever interacting with a double recessive mutation.

For example, in Budgerigar genetics, while the autosomal recessive allele for body color Sky Blue is recessive to Normal Green, recessive Sky Blue will act or behave in a dominant manner to the double recessive White (i.e., Blue x White = all Blue split White progeny). This relationship among recessives merely indicates their hierarchy behaviorally. It does not justify labeling the allele Sky Blue as "dominant," because genetically Sky Blue is an autosomal recessive. Rather, Sky Blue behaves in a dominant manner in the hierarchy of relationships with double recessives (i.e., White). However, as an autosomal recessive, it would be genetically incorrect to label Sky Blue as a dominant gene **(Table 3** copyrighted by author**).**

Similarly, the Pastelface (Pastel) Cockatiel is our first autosomal recessive Cockatiel mutation to interact in a "dominant" relationship to the double recessive Whiteface. For example, one may say the autosomal recessive, Pastelface, is recessive to Normal Grey, but behaves in a dominant manner to Whiteface. To call the autosomal recessive Pastelface a "dominant" mutation would be genetically incorrect. As far as we know at this time, the recessive Pastelface is recessive to all cockatiel mutations, with the exception of the recessive Whiteface.

What Cockatiel, Ringneck, and other parrot breeders must learn to understand (as do Budgerigar and Lovebird breeders who routinely work with such colors), is that such recessive genes merely demonstrate a hierarchy of relationships among autosomal recessive color mutations, in particular, how such recessives are inherited when working with double recessive mutations.

Genetic Myth #6: "Cross Over" (Crossing Over) occurrences can be predetermined or predicted. False!

Crossing over refers to a phenomenon in genetics where chromosome pairs swap segments of their chromosomes (i.e., chromatids) during meiosis, resulting in a recombination of linked genes. Such "crossovers" as termed in the fancy, happen by chance occurrence, and there is no way to predict when or where it will happen in avian color genetics. However, once crossing over does occur, it will result in some of the genes linking together to produce a new combination such as a cross, or double mutation. Without the original act of crossing over (e.g., coupling, or recombinant forms), many double mutations would not exist today.

For example, when this author presented a genetics workshop to color breeders in Boston during 1989, reference was made to a biologist who formally observed a near 30 percent occurrence of crossing over between the Lutino and Pearl genes in a very small population of cockatiels during the late 1970's. Such a crossing over from XL XP to XLP XN (where L stands for Lutino; P stands for Pearl; and N stands for Normal Grey) in a male cockatiel - known as coupling or cis linkage - was originally responsible for producing a small percentage of the first Lutino Pearl hen genotypes (XLP Y). Unfortunately, the information has been taken completely out of context and reprinted by subsequent authors as, "A crossover occurs about 30% of the time in male cockatiel chromosomes," which is simply untrue and without basis. Always know your source.

Genetic Myth #7: The Albino Mutation in Cockatiels is a single mutation, therefore a hen may be split (heterozygous) to Albino. False!

The "Albino" mutation in Cockatiels, unlike the majority of inos in other species, is not a singular, spontaneous color mutation. Rather, the "Albino" Cockatiel is actually a cross mutation between the sex-linked recessive Lutino mutation, and

the autosomal recessive Whiteface mutation. When birds of the correct genotype are paired they produce the cross mutation, Whiteface Lutino that is an all white bird with red eyes, depigmented beak and feet, that is void of all melanin (grey) and psittacin (yellow and orange) pigments. Rather than call the cross mutation by its genotypic name, Whiteface Lutino, the phenotypic label "Albino" has caught on as the avicultural lingo simply because it looks like the inos of other species.

Because the "Albino" Cockatiel is the result of two separate color mutations, one of which is a sex-linked color, it is incorrect to state that a hen can be split to "Albino." Hens, like cocks, can be split to autosomal recessives such as Whiteface, however unlike cock birds, hens can never be split to sex-linked recessives such as Lutino (refer to Myth #4). Therefore, whenever speaking of "Albino" Cockatiels, a hen could only be split (heterozygous) to Whiteface. This is also true of many other species where ino (i.e., Lutino or Albino) is sex-linked, although inos do exist in other modes of inheritance.

Beware of purchasing a Cockatiel hen represented as "guaranteed split to Lutino or 'Albino'," or you will be disappointed. The rule when working with sex-linked hens is "what you see is what you get." Hens simply cannot be split to Lutino or "Albino." Therefore, when working on the production of the "Albino" mutation in Cockatiels, a Cockatiel hen may only be visual for these mutations: "Albino," Lutino, or Whiteface; she may also be visual for Lutino and split to Whiteface; or Normal Grey split to Whiteface.

TABLE 5

COMBINING AUTOSOMAL RECESSIVES WITH CO-DOMINANT TRAITS

Example: P1 Whiteface Single Factor Dominant Silver cock
x Whiteface Single Factor Dominant Silver hen

= F1 wSwS = 25% Whiteface Double Factor Dominant Silvers
wSw = 25% Whiteface Single Factor Dominant Silvers
wwS = 25% Whiteface Single Factor Dominant Silvers
ww = 25% Whiteface

Note: The above has equal opportunity of producing either gender. These gametes can be checked with Table 4: Single and Double Factor Inheritance (i.e., #2 Single Factor x Single Factor = 1:2:1 ratio); and Table 2: Dominant and Recessive Matings (i.e., recessive x recessive = 100% recessive).

Genetic Myth #8: Single and double quantities are synonymous with single and double factor inheritance. False!

Autosomal recessive color mutations are sometimes referred to as having single or double quantities. If a color mutation is carried on both autosomes, the bird is said to carry a double quantity and will therefore display the (homozygous) phenotype for the autosomal recessive trait. Put another way, the color mutation, or any recessive trait, must be carried on both chromosomes in order for a bird to appear visual for the mutation.

However, if the recessive mutation, or trait, is only carried in single quantity on one autosome, the bird is then split (heterozygous) for the trait, carrying the trait in hidden form. Therefore, if only one autosome is affected by a single quantity of the trait, the remaining autosome serves to mask the autosome that carries the recessive trait. In avicultural lingo, a bird with a single quantity is called "split," or heterozygous, denoted by writing the slant sign before the name of the mutation (e.g., Normal/Pied is a normal appearing bird carrying a gene for the Pied mutation).

On the other hand, single and double factor color mutations refer mainly to autosomal mutations in one of the dominant modes of inheritance. Single and double factor birds may appear in two distinct color forms, expressed visually (phenotypically) in their outward appearance **(Table 4)**. Although such dominant mutations may theoretically be charted alongside sex-linked, or autosomal recessive color mutations (within the same individual or when charting a pair of birds), it functions as a co-dominant expression since the allelic gene present in a heterozygous state is expressed as a single factor phenotypic color form of the mutation. In other words, single factor birds will visually appear as one color;

TABLE 6

DARK FACTOR INHERITANCE

1. No Dark Factor	x	No Dark Factor	=	100% No Dark Factor
2. No Dark Factor	x	One Dark Factor	=	50% No Dark Factor 50% One Dark Factor
3. No Dark Factor	x	Two Dark Factors	=	100% One Dark Factor
4. One Dark Factor	x	One Dark Factor	=	25% No Dark Factor 50% One Dark Factor 25% Two Dark Factors
5. One Dark Factor	x	Two Dark Factors	=	50% One Dark Factor 50% Two Dark Factors
6. Two Dark Factors	x	Two Dark Factors	=	100% Two Dark Factors

Key: "No Factor" represents a Normal, or any other mutation which is not governed by Single and Double Factor Dominance inheritance.

Example: Budgerigars (applicable to psittacine species with equivalent psittacin pigments).

Dark Factor	Green Series	Blue Series
None	Light Green	Sky Blue
One Dark Factor	Dark Green	Cobalt Blue
Two Dark Factors	Olive Green	Mauve
None	Light Yellow	Light Grey
One Dark Factor	Mustard Yellow	Medium Grey
Two Dark Factors	Olive Yellow	Dark Grey

Note: Families of Yellow and Grey correspond with Light, Medium and Dark shades

Example 2: Cobalt Blue Lineolated Parakeet x Normal Green Lineolated Parakeet:
P1: One Dark Factor (Cobalt) x No Dark Factor (Normal Green)
= F1: 50% No Dark Factor: Normal Green/Blue
50% One Dark Factor: Normal Green/Cobalt
Note: Test-breeding is required to determine which F1 offspring carries the dark factor Cobalt.

double factor birds will usually appear as a uniquely different and alternate color form of that mutation.

Common examples of co-dominant and intermediate dominant mutations include the Dominant Silver Cockatiel, the Yellowface Budgerigar, and the Grey-green Indian Ring-necked Parakeet, respectively. When such colors are bred with others, new and interesting cross mutations may result, e.g., Whiteface Dominant Silver Cockatiels, Yellow-face Opaline Clearwing ("Rainbow") Budgerigars; Yellow-head Gray-green Indian Ring-necked Parakeets, etc. Such color combinations can require a great deal more work to chart compared to simple sex-linked recessive or autosomal recessive color mutations, especially when combined with additional existing mutations or modes of inheritance **(Table 5)**.

The terms "single and double quantity," may appear deceptively similar to the terms "single and double factor." However, the aviculturist might find it easier to associate "quantity" differences with homozygous and heterozygous genotypes (i.e., visuals and splits) in recessive traits; while "factor" differences indicate fully expressed, distinctly different colored phenotypes (i.e., two distinct color forms) appearing in dominant mutations.

Genetic Myth #9: Single and double factor inheritance is synonymous with dark factor inheritance. False!

In many of the popular species in aviculture that have been bred for numerous years in captivity, mutations have further differentiated themselves by being affected by color modifiers. Some modifiers, such as dark factors, can change the appearance of a standard color mutation.

For example, dark factors are well known in the Budgerigar Fancy. In the nominant wild green Budgie - referred to as the Light Green - the appearance of dark factors work to modify the Light Green into the Dark Green (one dark factor), and Olive Green (two dark factor) color shades. In the blue series, dark factors appearing in the Sky Blue Budgerigar result in Cobalt (one dark factor), and Mauve (two dark factor) color shades. Dark factors also affect the Yellow series, Grey series, and other mutations in Budgerigars **(Table 6)**.

Similarly, lovebirds and several Psittacula species such as Indian Ringnecks have followed suit with dark factor modifiers, which have permeated their green and blue series and other various colors. Many Cockatiel breeders already argue that Normal Grey Cockatiels show dark factors, ranging from Light, to Dark Grey, somewhat paralleling the example of the Grey Budgerigar mentioned above. We also find dark factors in other parrots from the smaller species such as Parrotlets, Lineolateds, and the Australian Parakeets to more rarely, the larger species of Psittacines.

While single and double factor mutations (as discussed under Myth #8), generally operate by a dominant mode of inheritance affecting specific mutations (e.g., Yellowface Budgerigars, Dominant Silver Cockatiels, etc.), dark factor modifiers appear to exist in both dominant and recessive autosomes affecting a number of color forms (e.g., Dark Green, Olive, Cobalt, Mauve, Dark Yellow, Olive Yellow, Medium Grey, Dark Grey, etc.).

Genetic Myth #10: The avicultural community does not affect the future gene pool. False!

To understand the big picture and the avian gene pool, an unlikely yet insightful comparison would be to discuss the use of antibiotics in humans. The medical

community is distressed by the casual use, or overuse, of antibiotics in human beings because each time any of us use these drugs it enables today's "super bugs" to grow immune to the antibiotics used. Once it becomes resistant to others, such an antibiotic would become ineffective for you or me.

So what does this have to do with the avicultural community contaminating the avian gene pool? Just as each of us make our own medical choices, similarly, as aviculturists working with breeding birds we each contribute our breeding lines - healthy or ill - to the overall gene pool.

When we fail to cull offspring from parents who carry disease, or unwittingly set traits for disease in bloodlines such as liver disease, diabetes, weakened immune systems, etc., we perpetuate these genetic traits in others' future breeding stock. Similarly, when we fail to monitor top exhibition lines that become weakened by producing birds with low fertility, smaller clutch sizes, or lack of parental skills, we contribute these problems to the gene pool and perpetuate them in others' aviaries. By continuing to cull birds (that can and do make excellent pets in the right homes), we all help to strengthen each others' stock.

Aviculturists must be ever vigilant in culling their lines for healthy, productive offspring whenever possible and encourage customers to do likewise. Whether we need an unrelated outcross for our own aviary, or a companion pet for a precious child, we can educate ourselves to produce robust birds that have an excellent chance for a long, healthy life, whether for our own aviary, or for future aviculturists and the future of birds.

Reference:

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