The Pigeon Genetics Newsletter

Editor R.J . Rodgers Nova Scotia Canada .

November 2014 Issue

Frank of Wilmington MA writes :

Bob, read your Newsletter with great interest. And thanks for taking over a genetic newsletter that Paul has so freely given of his time and efforts over many years. Before I begin giving my views on your article, realize that I am a Modena fancier and thus my comments are given in regards to my genetic knowledge as it applies to this breed.

First let me explain my version of smooth and coarse spread areas of a pigeon. This comment has nothing to do with the genetic Spread Factor. Smooth spread is that area of a pigeon dealing with the bar area of the tail feathers and the heavily ends of the major flight feathers, sometimes referred to as the remiges. Coarse spread deals with dark colored areas in the bars and checked (tri and T-pattern shield patterns) areas in the wing shield and includes the secondaries and coverts. If one has the genetic book written by Quinn please read pages 43 and 44 as he presents a great understanding of these terms and what they apply to what particular area of a pigeon. **{Editor's note : Quinn shows a diagram and care is needed to see just where the arrows point . he later clarifies by saying that coarse spread is on the portion known as the shield }**

Quinn also comments on different examples of certain other genetic factors and what areas will be effected by these factors. Example: Modena bronze effects primarily the coarse spread area of the wing shield. Grizzle effects the same coarse wing shield area but also other areas of the pigeon.

The genetic Spread (S) factor, in most cases, has the ability to create a complete overall intense Black pigeon in the Blue family and Dun being the dilute version. A intense Brown (chocolate) and Khaki being the dilute version. In the Ash-Red family another matter of expression appears in the outward phenotype expressions. Yes, they are other combinations of genetic factors that can create a Black or Brown pigeon, but these creations have nothing to do with the Spread (S) factor.

Why in most cases? Because in the correct combination of the genetic Toy Stencil Factors (TS1; Ts2; ts3) which only effect the wing shield area, we find a white colored shielded area depending upon the shield pattern in question. Sooty factor doesn't have any control in the phenotype of this ability to punch through the effects of Spread (S) factor. It is the Toy Stencil factors. Likewise in the recessive Red factored pigeons, this combination of Toy Stencil genes also expresses the wing shield pattern. What Toy Stencil gene allows this to happen, I have no idea. Maybe some others with a greater knowledge than I have can answer this matter.

Spread (S) factor does mask the effects of Sooty in the Black or Brown family, so if Sooty was present in these colors, how would we be able to know it was in the pigeon?

The bronze wing shield pattern color (TS1) found in Modenas is masked by the Spread (S) factor.

I believe there is still much to be learned about the Toy Stencil factors, and someday someone will uncover this mystery.

{ Editor's reply } I have sent a PDF file of Frank's comments back to him with my personal comments and told him I would print his letter here for others to respond .   BR.

Gene Hochlan writes :

Hello Bob,

Congratulations on becoming the editor of Pigeon Genetics Newsletter. Paul Gibson did a wonderful job for many years in this role and will be missed but if he will still contribute then he is not out of the picture. I have been a subscriber since David Rinehart was the editor and back then it was still called Pigeon Genetics; News, Views and Comments and I still have all copies. Dr. Willard F. Hollander was a good friend of mine and gave me all of the issues he was responsible for editing.

Spread Factor : Don't wish to make conflicting remarks about it but covering the entire bird with Smooth Spread still holds water and there are several facts that illustrate this with Frill Stencil being one of the best examples. Many years ago I also wondered why one could see the pattern (Course Spread) on some black pigeons and after raising a few out of certain matings I came to a conclusion. It is not due to lack of certain modifiers but rather because of ONE in particular. Lebanon Bronze of the Shikli Ahmar can be considered the namesake for this form of bronze but there are others that can mimic these markings on Ash Red. If you check Ash Red Indian Fantails, Lahores, Old Dutch Capuchines, Jacobins and several others you will find the parallels; perhaps not with the same intensity depth but recognizable nonetheless. If you try to transfer this bronze to Blue it will virtually disappear phenotypically but of course it is still there genetically. Combine this bronze with Blue Spread and presto washed out Black with pattern showing through. I have related this breeding result a number of times but I don't believe anyone ever bothered to take me seriously. My Lebanon Bronze mimic came from Jacobins. Instead of point blank rejection perhaps someone should try repeating this project with one of the above mentioned breeds.

{ Editor's response} Gene has a few different ideas to offer ., I hope everyone will read it over and offer their views . I have placed picture here with the permission of Anwarul kabir who is the Breeder of the black . Several Breeders of the (richly coloured T-pattern Ash-red lahores have reported Red pairs producing shiny pure black offspring , indicating that these are in fact SPREAD factor Ash-reds in some cases with exceptional Bronzing ! Note that the black has Bar pattern . This red was bred by and belongs to Nilesh Rajput .

Gary Young writes :
Congratulations on your first edition of the Pigeon Genetics News. Following up on your discussion on spread. There are very black birds in certain breeds that do not have the spread gene (you mentioned blackwing Archangels as a case in fact). I added the spread factor to copper and gold blackwing Archangels back in the 80's and again recently when Jim Oldham asked me about some that he had. They are black selfs with no gimpel bronze. Some other darkening factor or factors besides spread make them appear black.

I was also quite surprised to discover that my shiny black whitetail Catalonians related to John Rossner's recent imports did not have spread in them. When mated to blackwing Archangels they produced only dark check offspring -- no spread blacks. The white tail completely disappeared with the exception of a couple of youngsters that had a few white tail feathers.

In my experience with breeding Archangels, the presence of a spread gene completely inhibits the expression of gimpel bronze so that the birds have black bodies even when they are homozygous for gimpel.

I forgot to mention that besides being dark checks, the black whitetail Catalonian X Archangel crosses all had some degree of gimpel bronze on the bodies and a high degree of iridescence from both parents as would be expected.

A prominent Oriental Frill breeder once told me that his birds were both frill and toy stencilled and that frill stencil combined with toy stencil makes a better colored bird. I found that my "Zuau" (stencilled) Catalonian exports had frill stencil in addition to toy stencil but their white tails hid any spottail expression so it was not obvious until I put them with non-whitetailed mates. Beautiful bronze stencilled Catalonians called "Firebacks" are made by crossing black whitetail Catalonians with the whitetail stencilled Zuau. **{ Text in Blue added later in another email }**

This summer, I mated a couple of show quality Starlings to wild type blue and found they did not have the spread factor in them either. This was not completely unexpected however because Starling breeders I have talked to in the past say they have to cull closely to keep the dark black color. One youngster I produced had a beautiful spotted tail indicating the presence of frill stencil in both parents, although it does not normally appear in stenciled Starlings.

Gary Young

{ Editor's response} Gary offers a few more interesting points . We welcome your responses to him !

~~~~~~

Garry Glissmeyer writes :

Of interest (?) to you and perhaps others who work with tail breeds, small or large, there seems to be a difference in genes which affect Count (number of feathers in the tail) and Wrap (how far around the tail "wraps", the circumference. "Seems" to be. Not scientifically proven.

We, in the Indian Fantail Club have not done a controlled study of it, but experiential observation would indicate that it is true. Read on...

Why is it even important? For us in the tail breeds, we often have desired shapes and qualities outlined in our Standards describing the "ideal" tail. In Indians, it is supposed to wrap around at least 3/4 of the way "plus one or two additional feathers" for the ideal. (It used to be 7/8 of a wrap, but we lessened the wrap percentage to allow the wing tips not to be pushed down to the floor by the tail.).

Our long-held belief was that the more feathers in the tail, the greater the wrap? So we all were hoping and praying for those tail counts of 34-40 feathers. But some of us (Lynn Kral, myself, Dan Skiles, et al...each Master Breeders) started noticing it wasn't just count which created wrap.

To wit: in 2006, I raised a White Indian Fantail hen which had one of the largest tails in my loft, and had nearly 7/8 wrap. But! But...it was not a show quality tail... it had slight "gaps" in between most of her tail feathers, She
~~~~~~

only had 27 feathers, yet a 7/8 wrap. Not enough feathers to snugly fit side-by-side, or layered one-under-the-other (like shingles on a roof) for show. I did show her in Des Moines that year, and the first thing the judge observed was, "She stations beautifully, has the tail size and shape we are after, but there is air between most of her tail feathers...gappy. A fault."

Conversely, I had a "Cream" (ash red dilute) Saddle hen with 37 tail feathers, who's tail only came down barely past halfway! And the highest cluster of her tail feathers were massed at each end of the tail, making it almost impossible to arrange neatly for show. Too many in too small an area.

Lynn Kral, one of our winningest breeders, experienced similar birds in her loft. Nelson Smith, another Indian breeder, has also noted this: count and wrap seem to be controlled by different genetic genes.

We are now believing, and experiencing these two genes (Wrap, and Count...our names) act as simple, recessive genes. And are accordingly breeding toward them using the same recessive gene processes: Keeping records of tail count, and percentage of wrap; thereby, we know which bird has (or is) from parents with wrap, or count, or both -- and mating those birds together which show (or carry) these traits. We can thereby improve Count. Or Wrap. Or both.

The results are quite consistent with recessive genetics. If there are any Fantail, or other tail-breed breeders reading this, have you noticed, or experienced the same?

Photos provided by Editor from Facebook edited :





{ Editor's Response } I have made many crosses involving other Breeds with Indian Fantails ., also with the Classic American Fantail ., and Between the two Fantail Breeds . The tail structures even in the f1's varied greatly , and seemed to develop differently as they were back crossed . While I did not follow that closely ., I believe that it gives credence to what Garry postulates above .

~~~~~~

Colour genetics research at three American Universities :

Work at the University of Utah ... Prof. Shapiro's team at University of Utah published research last year in the journal *Science* that revealed results from the first large-scale sequencing of the pigeon genome. That collection of more than 100 billion DNA bases from 40 pigeons, provided the basis for the new work.

The following written by Jith Peter , Palakkad India .

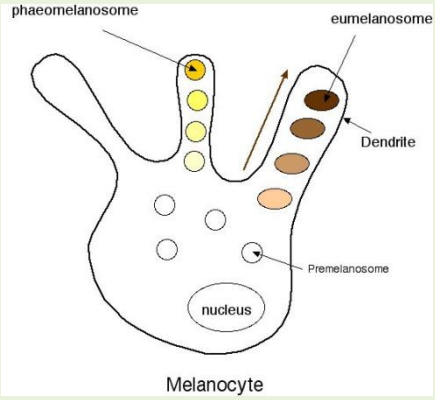
Before I talk about the research report published by the University of Utah, I think it is important to give a brief idea about gene expression (protein synthesis) in the molecular level and Biosynthesis of Melanin in the cellular level, it would help to understand things properly.

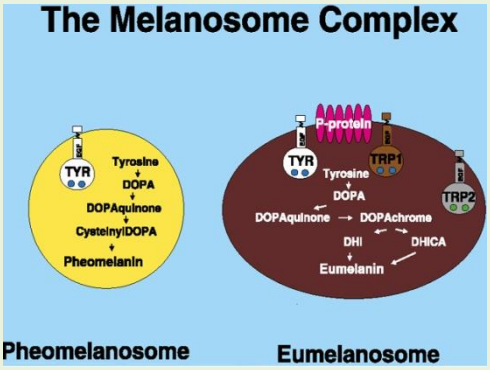
Genetic information, stored in the DNA (Genes) is expressed through transcription to RNA and, in the case of messenger RNA, (mRNA), subsequent translation into proteins. The pathway of protein synthesis is called translation because the "language" of the nucleotide sequence on the mRNA is translated into the "language" of an amino acid sequence (Protein). The process of translation requires a genetic code, through which the information contained in the nucleic acid sequence is expressed to produce a specific sequence of amino acids. Any alteration in the nucleic acid sequence may result in an incorrect amino acid being inserted into the polypeptide chain or deletion of some amino acids from the chain, and result in production of altered protein, which may cause one of these changes in the molecular level 1) loss of function of the gene, 2) gain of new function, 3) increased level of gene activity, 4) reduced level of gene activity.

Usually Newly made proteins undergo a number of processes to achieve their functional form. They must fold properly, and mis-folding can result in degradation of the protein. Many proteins are covalently modified to activate them or alter their activities. Finally, "proteins are targeted to their final intra- or extracellular destinations by a signal peptide present in the proteins themselves. Once the protein reaches the target, an enzyme known as a Signal peptidase may cleave
~~~~~~

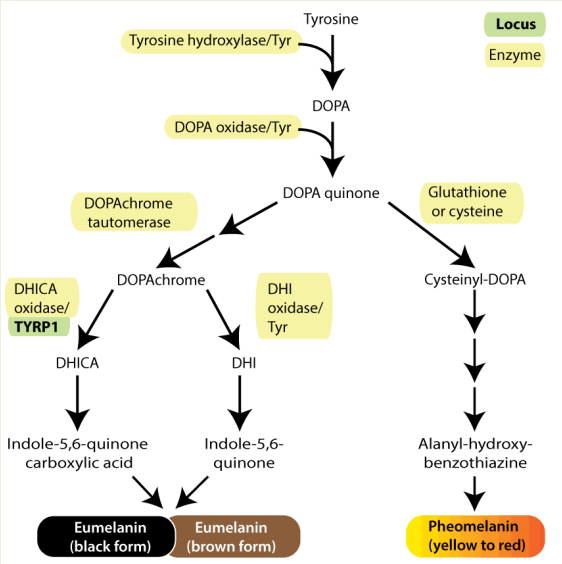
the protein and generate a free signal peptide and a mature protein. The free signal peptides are then digested by specific proteases, and the mature protein take part in the biochemical process.

Biosynthesis of Melanin





Melanin is synthesized in a multistep biochemical pathway that operates within a specialized intracellular organelle known as the melanosome. Melanosomes are produced inside the Melanocytes and they pass through several developmental stages , starting in the middle of the melanocyte cell, and migrating to the outer edges of the cell through the dendrites . . Melanocytes can produce eumelanosomes or pheomelanosomes at different times, switching from one to the other. The Melanocytes migrate into the dermal pulp of the developing feather germ, where the melanin is packed into Melanosomes and then those Melanosomes are transferred to keratinocytes for deposition into developing feathers. The type, amount and size of the Melanosomes and melanin particle production is controlled by genes.



Actually we don't need to go detailed through the Biosynthetic pathway of Melanin production, that is not why I attached the diagram from the Internet, instead it will give us brief Idea about how Melanin is

produced from one of the 22 Amino acids present in the cells called Tyrosine, AND how and where the gene products (proteins) interact with chemical intermediates at various stages of biosynthesis of Melanin.

Feather color is a polygenic trait, Many genes contribute toward producing these different color shades by taking part in the synthesis of different amounts or kinds of substances that give rise to the visible color differences.

Tyrosinase gene (Tyr) is the first gene identified in the tyrosinase gene family, code for a key enzyme in the biosynthesis of melanin called tyrosinase. In many species mutation in the Tyr gene results in Albinism, the albino mutation present in pigeons may also be in the tyrosinase gene. Tyrosinase is known to be involved in the first two steps of melanin production (hydroxilation of tyrosine to DOPA and oxidation of DOPA to DOPA quinone), these two steps are common for both eumelanin (black pigment) and pheomelanin (red pigment) and you can see that , in the diagram. A third enzymatic role has been proposed for tyrosinase: the oxidation of DHI to indole-5,6-quinone,you can see that step in the downstream of "eumelanin pathway" in the given diagram, however it has also been proposed that this oxidation is performed through a peroxidase. So the first two tyrosinase-catalyzed reactions are common to eumelanogenesis and pheomelanogenesis. It is known that they diverge after the formation of dopaquinone, but the mechanisms responsible for this divergence are poorly understood. Dopaquinone is the key intermediate in the formation of pheo vs eumelanin. The eumelanins are derived from the metabolites of dopachrome. An enzyme known as dopachrome tautomerase catalyses the conversion of dopaquinoe to DHICA(5,6-dyhydroxyindole-2-carboxylic acid), the enzyme is coded by a third known gene in the tyrosinase gene family called Tyrp2 gene. Whereas in the case of pheomelanogenesis, an aminoacid called Cysteine non-enzymatically react with Dopaquinone and form Cysteinyl-DOPA, it then undergoes metabolism and results in pheomelanin production. It is known that concentration of Cysteine is a key factor for the production of Cysteinyl-DOPA, but the mechanism behind the control of concentration of the cysteine within the melanosome and the remaining steps of pheomelanogenesis is poorly understood. One more gene in the Tyrosinase gene family (secondly discovered gene in the family) called Tyrp1, is also involved in the melanin production. The function of Tyrp1 is subject of much controversy. Experiments on the mouse Tyrp1 provided evidence that the protein functions as DHICAoxidase, a reaction downstream in the eumelanin synthetic pathway (you can see that in the diagram,, conversion of dopachrome to DHICA). In addition to the DHICAoxidase activity, Tyrp1 gene is known to modulate the Tyrosinase gene. Moreover, the pheomelanogenic pathway is largely unknown and the structure of the final melanin polymer is not resolved and might, thus, contain both eumelanin and pheomelanin……In addition to the tyrosinase gene family, The MSH cell surface receptor and the melanosomal P-protein (A mutation called pink eyed dilute in mice present in this locus which is similar to the pink eyed dilute present in pigeons) , are the two most obvious candidate genes influencing variation in pigmentation phenotype in humans and mice, we don't need to know in detailed about them now as the research paper is not talking about them.

According to the latest research report "Epistatic and Combinatorial Effects of Pigmentary Gene Mutations in the Domestic Pigeon'' published by University of Utah, Multiple Mutations in Tyrp1 Underlie Base Color Variation in Pigeons.Tyrp1 gene is the first cloned pigmentation gene and later mapped to the mouse brown locus. The gene is located on the Mouse chromosome 4 and Human chromosome 9, in both Human and Mouse the gene is located on autosomes whereas in pigeons we know that it is in the Sexlinked Z-chromosome. The gene is pretty large, encompasses 8 exons (coding region) separated by 7 introns (non-coding region).

To investigate the molecular identity of the B color locus, they compared the genomes of 6 ash-red pigeons to 26 blue/black pigeons and found that All blue/black pigeons were homozygous G

(Glycine)-one of the four bases present in DNA) on the Tyrp1, whereas ash-red pigeons were hetero- or homozygous for C (Cytosine), consistent with the dominant mode of inheritance of ash-red. The BA mutation causes an alanine-to-proline substitution at codon 23 (A23P), corresponding to the cleavage site of the signal peptide. This type of mutation is called as missense mutation (change in a single aminoacid in the protein level).In addition, they found a perfect association between the dominant BA mutation and the ash-red phenotype in an additional 49 ash-red birds from 20 breeds, and 105 blue/black or brown birds from 36 breeds. These results suggest that the ash-red mutation occurred only once and spread species wide through selective breeding. Tyrp1 mRNA levels from developing feathers of B+ and BA pigeons were indistinguishable indicates normal level of transcription (gene expression) of BA allele. however, the location of the BA mutation at the highly conserved cleavage site of the signal peptide suggested that cleavage efficiency might be affected. From the related tests they found that cleavage efficiency was dramatically reduced by the BA mutation. Furthermore, spatial organization of pigment synthesis differed between B genotypes. Pre-melanosomes in regenerating blue/black (B+) feathers had a well-organized, lamellar matrix, and melanosomes were darkly pigmented, whereas ash-red (BA and B+/BA) feathers had a disorganized matrix and only lightly pigmented melanosomes. After incubation with the melanin precursor L-DOPA, melanosomes from both wild-type and ash-red birds became darkly pigmented, indicating normal catalytic activity of the melanogenic enzyme tyrosinase (Tyr) in ash-red birds. However, pigment synthesis in B+ feathers showed strongest staining localized to the limiting membrane of the melanosome, whereas staining was diffuse in melanosomes from BA and B+/BA feathers. Thus, the striking reduction in TYRP1 cleavage efficiency may disrupt the spatial organization of pigment synthesis activity, providing insight into the molecular basis of dominance of the BA allele. A similar missense mutation near the same cleavage site present in mice causes melanocyte death probably through the accumulation of cytotoxic pigment intermediates; however, unlike the mutation in mice , the pigeon BA mutation results in a different kind and localization of melanin production rather than abrogation of melanogenesis.

In contrast to the single ash-red mutation, Tyrp1 sequences from 51 brown pigeons from 30 breeds revealed a nonsense mutation (R72X) symbolized as b1 and two different frame-shift mutations (symbolized as b2 and b3), predicted to be null alleles. The nonsense mutation is due to a single base pair change. The codon containing the changed base may become a termination codon. so the new codon causes termination of translation at that point, and the production of a shortened (truncated) protein. Whereas in the case of frame-shift mutations , some nucleotides are deleted from the coding region of a message sequence, frame-shift mutation occurs and the reading frame is altered. This can result in a protein with a radically different amino acid sequence, or a truncated product due to the creation of a termination codon. Tyrp1 mRNA abundance in b3 pigeons—the most common b allele in our sample—is greatly reduced or absent and indicates brown mutations result in null alleles. Mutations are called null mutation (lack-of-function mutation) when the mutation results in lack of molecular function of the wild type allele. More than one allele for brown suggests that unlike ash-red phenotype the colour has evolved multiple times in pigeons. Several brown pigeons did not have any of the identified b alleles, raising the possibility that additional mutations might also cause brown feather color (possibly they are dilute blue series birds or any other brown mimic).

Based on these results their analyses suggest a model in which BA is a neomorphic allele that alters processing of the mutant TYRP1 protein within the cell. A mutation is said to be neo-morphic when the resultant protein possesses a novel function in the molecular level, neo-morphic alleles are always dominant or partial dominant. Since TYRP1 can modulate Tyrosinase(Tyr) activity, they postulate that the BA version of TYRP1 protein alters normal Tyr functionality, resulting in an increased ratio of pheomelanin to eumelanin production. In contrast, Tyrp1 loss-of- function alleles b1,b2,b3 cause brown pigment production, consistent with findings in other vertebrates like mice.

Sylvie Eglin writes :

Hello Bob,

I am a breeder of Triganini reduced colour. I would ask other breeders of reduced if they could explain to me the difference between black reduced and andalusian reduced . If some breeder could send some pictures of reduced pigeons, it would be interesting . Sincerely , Mr EGLIN .

These six birds bred by Mr Eglin:

One rather consistant effect of Indigo is to cause this half light , half dark expression on the closed flights of the wing minus the laced edges in non-reduced birds . Owner Franz Metakohy from Facebook.

\~~~~~~

Sylvie Eglin asks : I would like to know the genetic formula of my pigeons in pictures. It would be nice if I could have some contacts with people who have Triganini of this colour.

\~~~~~~

University of Utah Pigeon Research, by Gary Young

Reviewed and approved by Mike Shapiro, associate Professor, Dept of Biology

DNA researchers at the University of Utah have constructed a genetics lab to help them understand the molecular basis of phenotypic diversity in pigeons. The birds are kept in cages contained in a carefully controlled environment where they can be examined. A visit to a pigeon show at the county fair or other place will convince anyone that they are spectacularly diverse and exhibit variation in more traits than perhaps any other animal. Artificial selection since Neolithic times by pigeon breeders has resulted in a massive selection experiment. Striking differences in behavior, vocalizations, skeletal morphology, feather ornaments, colors, color

patterns, and many other traits has resulted in over 350 breeds. DNA researchers have found that they are easily bred in the lab and make excellent models for research without harming the animals. One of their more recent discoveries is that the molecular basis for plumage color in pigeons is closely related to skin & hair pigmentation and even skin disease in humans.

To get started, the researchers identified and described the genetic material (genome) that encodes the DNA of domestic and feral populations of pigeons. Their conclusions illustrated the genetic relationships among 70 different domestic breeds of pigeons and also illuminated the geographic origins of breed groups in India and the Middle East (Divergence, Convergence, and the Ancestry of Feral Populations in the Domestic Rock Pigeon; Current Biology Vol. 22, pp 302-308, February 21, 2012).

They went on to identify and describe the chromosomal location "EphB2" for the feather crest (cr) mutation which causes localized molecular reversal of feather bud polarity during early embryonic development. They found evidence that the crest (cr) mutation evolved just once and was afterward carefully selected and modified by pigeon breeders to form peak, shell, mane, or hood ornaments. This research was published in Science magazine (Genetic Diversity and Evolution of the Head Crest in the Rock Pigeon; Science, Vol. 339, pp 1063-1067, March 1, 2013, <sciencemag.org>).

The same group of scientists recently identified the chromosomal locations for several basic plumage colors (Epistatic and Combinatorial Effects of Pigmentary Gene Mutations in the Domestic Pigeon; Current Biology 24, 459-484, February 17, 2014). Their molecular evidence confirmed the findings of applied pigeon genetic researchers that, "The classical major color locus (B) in domestic pigeons is a sex-linked gene that confers one of three "base" colors: wild-type blue/black (B+), ash-red (BA), and brown (b). The BA allele is dominant to B+ and b, and b is recessive to the others. Blue/black and brown phenotypes result from high amounts of eumelanin and low amounts of pheomelanin; melanin ratios are reversed in ash-red birds. In addition, the autosomal recessive red (e) gene acts epistatically to the blue-black (B) locus to elevate pheomelanin production, generating red plumage color irrespective of B locus genotype. Mutant alleles of a third locus, the sex-linked recessive dilute (d), interact additively with B and e to lighten plumage color and further enrich pigmentation diversity."

A locus on a chromosome may be compared to the GPS location for a particular address or geographic object. It is exact and precise. The University of Utah researchers found that the locus for the blue/black, ash-red, and brown gene is "Tyrp1"; the locus for the recessive red gene is "Sox10", and the locus for the dilute gene is "Sic45a2". They are currently studying other color and color pattern traits, as well as anatomical traits that vary among breeds.

~~~~~~

Finally ., here is a fun phenotype for you to ponder . Post of one of Mohammed Shoaib's Sherazi at Multan Pigeon Club Face book Group..

Take a stab at the genotype :
~~~~~~

~~~~~~

So ends the second Issue of our talks here in the Pigeon Coop ! I hope you found something of interest to you and that you will feel free to email me with your photos and questions and answers ! The December Issue is being prepared now . If you are new to Genetics ., please do not be shy about asking anything that you have on your mind about Colours , Ornaments ., etc. Some one here will likely know the answer , or at least have a suspicion as to what the answer might be . Until next Issue ~ Editor Bob Rodgers. bob_rodgers556@hotmail.com
~~~~~~