The Pigeon Genetics Newsletter

news , views and comments .

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December Issue 2014

Due to requests from a number of you , I have added the (news , views and Comments) back to the Newsletter name ! I had several people say that I should charge a fee but I feel it is important to keep it available without charge ! Two people asked me not to use colours as printing is costly , but I feel the fact that it is FREE offsets that matter. I will attempt to have three levels of material : (1) beginners, (2) Intermediate , and (3) Advanced , so that everyone may find something of interest.

By now most of you will have reduced your flocks to the Breeding pairs that you want producing in early 2015. The Shows for 2014 are just about finished , and probably each of you have purchased a new bird or two to bring in that special colour trait you have been wanting to get . Here is the place to show what you plan to do ., and anything you have done to date . Everyone is waiting to see what the other fellow has accomplished . Even if we get to the shows ., we seldom get a chance to talk for long ., or see just what you are working on . Here , there are no interruptions. Whatever you are doing ., PLAY Safe ! Mask up when cleaning floors ., perches and nest boxes . Open the lofts and if possible let the birds outside to reduce the flying around and stirring the dust up more. It is not the heavy dust that causes problems ., it is the smoke-like fine dust that hangs in the air and enters your eyes, nose and mouth. It clings to the walls of your loft, your clothes and hair. You do not notice it is harming you internally until the damage is done.

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Now for Genetics ~ (1) Beginners .

Blue Series Bar Pattern

Blue series Checker pattern.

The Base Colour Black Pigment is located at the major Colour Locus on a strand of DNA.

The Rock Pigeon is referred to as a Blue Bar The overall appearance is referred to as "wild type" with the symbol (B) or (+), this indicates a Blue series bird with two black bars on each wing . The feet are free of feathering, with no ornamentation of feathering (crests , rosettes , etc.) elsewhere. There will be twelve tail feathers and no deviations from the normal wild type specimens.

The blue series black pigment is an Intermediate sex-linked gene. It is recessive to the Ash-red series mutation, and Dominant over the brown series mutation which share the same spot or locus on the DNA strand. They are called sex-linked Alleles as they are located on the sex chromosomes.

"Pattern" refers to the arrangement of colour "HUES". On the wing shields, the granules of black pigment are clumped together, leaving negative areas around each clump to cause us to see a gray/blue bird as opposed to a black bird. The black portions of the wing PATTERN (bars & Checks)are referred to as COARSE SPREAD with a symbol (C). Here the pigment is densely deposited so as to allow no negative areas around it. The tips of the wings and a band across the tail are also dark, but this is called smooth spread. In order for us to see the entire bird as "BLACK" we need to have another mutation in which the granules of black pigment have been arranged evenly over all portions of the bird's feathers throughout . This gene is called "SPREAD FACTOR ".The Blue Series can be modified in tone and Intensity by a host of other Modifying Mutations. This diagram shows how a typical pair sex-linked for colour works. The Females colour base must be Dominant over that of the male in order for the "criss-cross " to be readily seen.

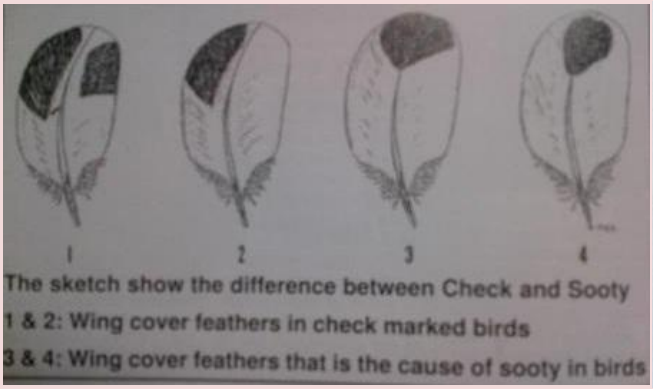
Blue Cock X Ash-red hen

= Ash-red males carrying blue. & blue females

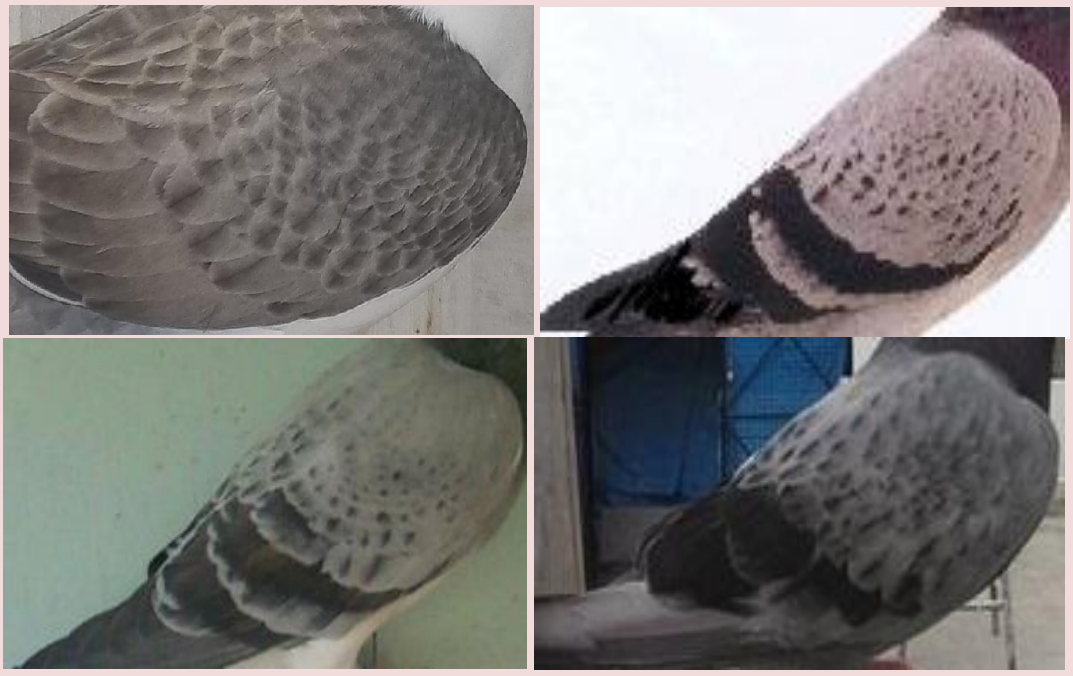
(2) Intermediate genetics :

Observing feathers Closely :

Here are a few pictures of feathers and the distribution of Pattern & Sooty Factor.



Sooty somewhat resembles checker pattern on the Blue barred Pattern. Sooty usually darkens with age. Not an actual genetic Mimic as it is a mutation , not monipulated by time and need .



Sooty is therefore , just a coinsidental look-a-like of some checker patterns

More on sooty at the end of this Newsletter. Here the photos were taken from the Facebook Groups.

Sooty on bar is called "Dapple"., on spread ash Sooty may appear as a lacing called "Strawberry".

Below are photos of youngsters that appear to be expressing Sooty

Last Issue we offered a number of subjects with the hope that we would have input from YOU the readers . Ironically ., as I opened the first several Issues back when Paul first took over the Newsletter in 1982 ., I realized that these same topics were being discussed and to this day have no resolve. The main reason of course is that we are not taking a scientific approach , but instead we are trying to prove or disprove theories by breeding exersises without actually understanding what is going on biologically speaking .

" Spread factor the Gene " , does it actually spread smooth spread pigment arrangement over the entire bird ., or does it rearrange just the Clumped pigment to become smooth spread ., or is there another explanation ? I am of the opinion that Spread factor permiates all of the feathers evenly with smooth pigment in addition to coarse pattern . Modifiers give a visual blend. Ash differs due to the granules being differently shaped. I am expecting several other articles on this subject in the near future.

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Toy Stencil and frill Stencil ~ When they appear together we simply refer to them as Frill Stencilling ., We have learned that the (Ts) trait is a combination of at least two genes that must work together to give us the white Toy Stencilling effect ., and that an unspecified third trait (ts3) almost certainly must be present as well. When the Dominant Ts1 trait is alone it gives a Bronze pattern. Ts2 is an oystershell colour. Toy Stencil affects only the wing pattern (C) areas and will express even with epistatic traits such as Spread factor and recessive red/yellow..

Frill Stencil gives us only a white expression that affects the feather ends that have darkening traits. Sooty factor is one of those traits that facilitates this stencil factor , (fs) will express on smooth spread areas and that may include body and neck feather if darkening traits are present. This trait has been designated as a recessive but it has proven to be variable and even unpredictable , based upon what modifiers accompany it !

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Bronze and grizzle : Few want to take on the Topic of "BRONZE" ., or "GRIZZLE" , and usually prove not to be working with the trait that they think they have . These two traits sometimes actually seem to work together in a variety of ways . We have a wide range of ideas as to just where one BRONZE gene stops and another begins . Generally it is felt that the difference , if there is any., lies in the region (s) where each one expresses on the birds or individual feathers . We will revisit all of these traits in future Issues .

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(3) - advanced genetics:

From Gary Young :

Hi Bob, I spoke with the U of U pigeon genetics researchers at the Salt Lake Premier show last Saturday. They were taking blood samples of almonds especially because that is what they are presently working on.

They have placed an interactive pigeon genetics game online at
http://learn.genetics.utah.edu/content/pigeons/

wish I had that back when I was teaching high school biology. ....Gary.

Photos of Columba guinea from the net .

{ I have asked the U of U to contribute to the Newsletter , but had no response to date . }
Editor

Anyone who has advanced information about Colour Genetics in Pigeons is welcome to write an article and send it in to me anytime ! B.R.

"Regulatory changes underlines recessive red colouration in Columba livia"

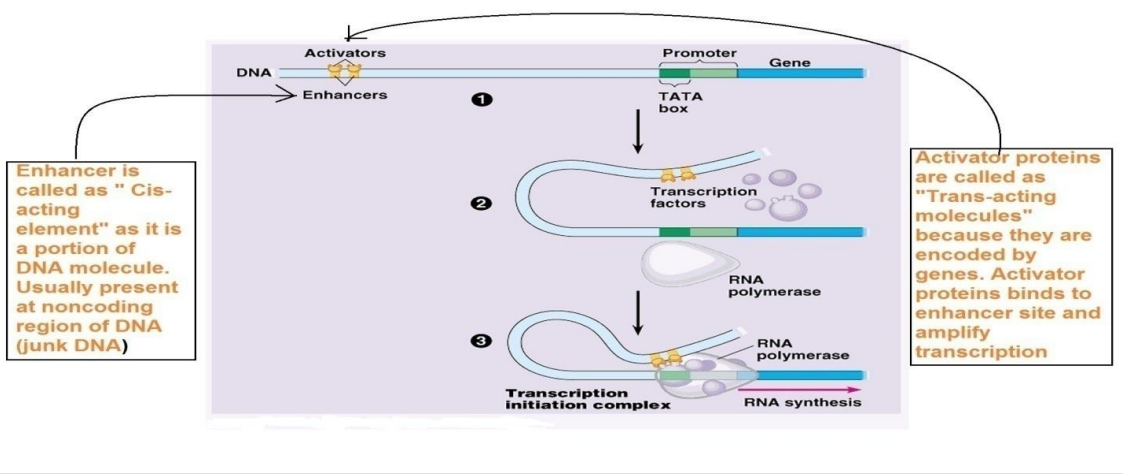
- **Presented by Jith Peter , Palakkad India.**

Before I talk about the research report about Recessive red mutation in Columba livia published by the University of Utah, I think it is important to give a brief idea about Regulation of expression of genes in Eucaryotcs (Mainly Transcriptional regulation), and some facts about the Sox10 and Tyrp1 genes and their relation. It will help to understand things properly.

There are many different types of cells present in multicellular organisms like different types of birds, Mammals etc, and in almost every type of cells of an organism , the exact same type of genome (DNA) is present. But not all genes are expressed in all cells all the time. Indeed, much of life depends on the ability of cells to express their genes in different combinations at different times and in different places. There are various gene regulatory networks present in the cells and these regulatory mechanisms control the expression of most of the genes (but not all) present in the nucleus of cells. Gene expression refers to the multistep process that ultimately results in the production of a functional gene product, either ribonucleic acid (RNA) or protein. The first step in gene expression—the use of deoxy-ribonucleic acid (DNA) for the synthesis of RNA (transcription)—is the primary site of regulation. However, gene expression also involves extensive post-transcriptional and post-translational processes, as well as actions that influence access to particular regions of the DNA. Each of these steps can be regulated to provide additional control over the kinds and amounts of functional products (either Protein or sometimes RNA) that are produced. But now all we need to know is just about the 'regulation of transcription' only.

Regulation of transcription

Transcription—the initial step in all gene expression—is controlled by regulatory sequences of DNA, usually embedded in the non-coding regions of the genome (junk DNA). The interaction between these DNA segments and regulatory molecules, such as transcription factors, can engage or repress the transcriptional machinery, influencing the kinds and amounts of products that are produced. These DNA sequences flanking a gene are called cis-acting because they influence expression of genes usually on the same chromosome, in otherwords cis-acting elements are part of DNA molecule. A trans-acting factor is a protein coded by a gene , which can diffuse through the cell from its site of synthesis to its DNA-binding site and can regulate transcription of other gene (or genes).So we can say that, Trans-acting protein molecules bind to cis-acting elements on DNA and regulate expression of a gene or genes .



.After lots of search in the google I found this picture, not a perfect one, but pretty enough to demonstrate the transcriptional regulation.

In the first step numbered (1), you can see that a portion of DNA consists of a gene, in the start of a gene there is a promoter region and at extreme left of the DNA (upstream to the gene) there is an enhancer region.

Promoter is a region of DNA that initiates transcription of a particular gene. Promoters are located near the transcription start sites of genes upstream on the DNA as you can see in the pics. Now 1000s of neucleotides upstream (opposite to the direction of transcription) or sometimes downstream (on the same side of the direction of transcription) , we have things called "Enhancers" also called as "distal control elements". These enhancers are just non-coding regions of DNA (junk DNA) and they don't code for any functional product (protein or RNA) but are conserved in the genome and necessary for the regulation of expression of a gene or genes associated with it. For the initiation of transcription of a gene, first a transcription factor, (a protein encoded by another gene) attaches with the promoter region of the gene which has to be transcribed. And then a mediator protein(encoded by another gene) carries RNA Polymerase (which is what transcribes the gene) and attaches with a transcription factor which is already attached at the promoter region. Followed by this, a number of general transcription factors and co-activators come and attach there and form a multi-protein complex bound to DNA called the transcription initiation complex. We have another protein called "Transcription activator" that attaches to the enhancer region. NOW transcription of the gene is ready to be activated. A protein called (DNA bending protein) , bends the DNA and allows the transcription activator protein to come in contact with the transcription initiation complex. Contact between the activator protein and the transcription initiation complex amplifies the transcription mechanisms. The amplified (activated) transcription of sox10 and Tyrp1 genes normally happens in the melanocytes of wildtype pigeons. Mutation to the enhancer region and/or to the gene which encodes transcription factor can results in either 1) increased level of expression of the gene in question and then we say expression of the gene is "up regulated". 2) Or it can results in decreased level of expression of the gene in question and then we say expression of the gene is "down regulated".

If you think you have not understood the mechanism of Transcriptional activation properly ,Then watch this video before you are going to continue any further !.
" http://m.youtube.com/watch?v=5MfSYnltYvg# "… copy the link and paste in youtube search.

Sox10 and Tyrp1 gene relation that is already known, revealed from the molecular research in other Species.

So, we know that protein encoded by one gene can control the expression of other gene (s) by binding to the enhancer site of the gene which has to be controlled.

According to the research report in Mice published by the ISREC (Swiss Institute for Experimental Cancer Research), National Center of Competence in Research (NCCR) Molecular Oncology, Switzerland- in 2006, "A conserved transcriptional enhancer that specifies Tyrp1 expression to melanocytes". The report also says The Tyrp1 enhancer is trans-activated by Sox10. Which means the protein encoded by the Sox10 gene needs to activate the enhancer region of Tyrp1 gene, for the normal level of transcription of the Tyrp1 gene in the melanocytes.

Just as the Tyrp1 gene has an enhancer site, the Sox10 gene also has a conserved enhancer site that specifies Sox10 expression to melanocytes, which is trans-activated by a protein encoded by some other gene. In melanocytic cells there is evidence that the Sox10 gene expression may be regulated by "Microphthalmia-associated transcription factor (MIFT)" . Perhaps that is the protein transactivating the Sox10 enhancer. Amazing how expression of a gene can affect many other genes!!. Deletion of the conserved Sox10 enhancer results in pigmentation defects in other vertebrates, including a lack of pigmentation in the mouse and increased pheomelanin production in chickens .

Other than Tyrp1 enhancer activation, SOX10 protein has many molecular functions including regulation of embryonic development, if you look at the wikipedia you can see the molecular functions of the Sox10 gene. So it is common sense that one may wonder why deletion at the Sox10 enhancer in Chickens and Mice does not result in a lethal mutation….The reason probably is that the enhancer specifies expression of Sox10 only in the melanocytes (pigment producing cell). In other cells the expression of the Sox10 gene is independent of the enhancer, so the deletion in the enhancer really is not affecting expression of the Sox10 gene in other cells.

Epistatic and Combinatorial Effects of Pigment Gene Mutations in the Domestic Pigeons.

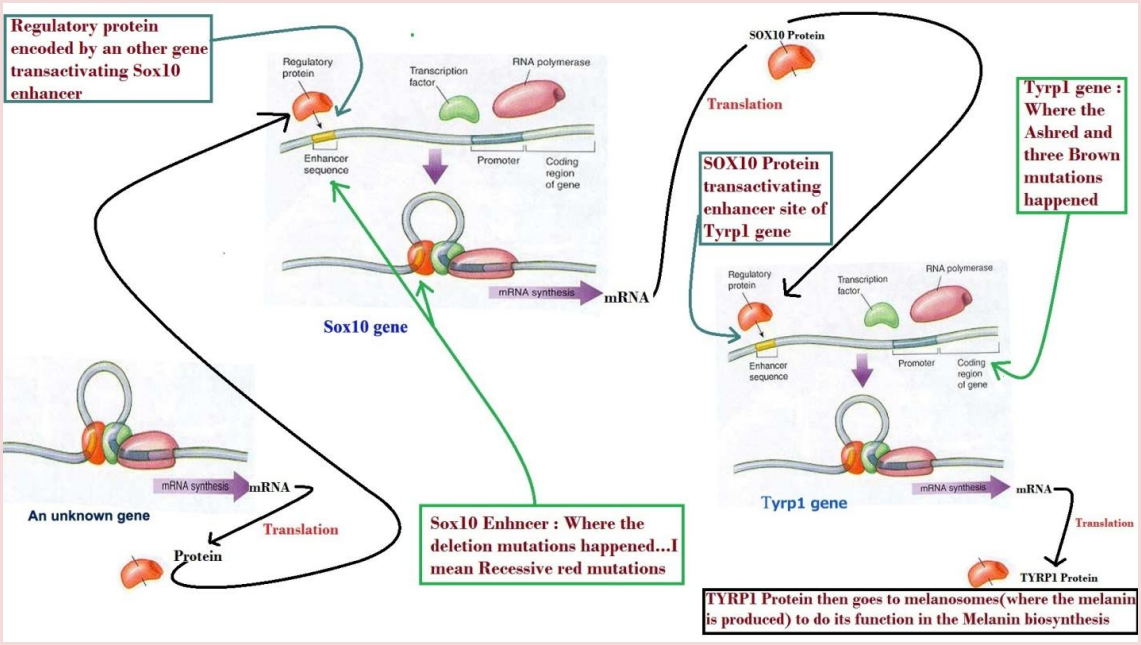
According to the research report on recessive red mutation published by the University of Utah ", repeated deletions of a Sox10 enhancer underline recessive red mutation". To identify candidates for recessive red mutation, they compared expression of several genes involved in melanin biosynthesis and found that the transcription factor Sox10 and one of its target genes, Tyrp1 (the B locus), were significantly down-regulated in feathers of recessive red birds. Whereas other melanin biosynthesis genes did not show any alteration in the level of expression, suggesting that a mutation directly or indirectly affecting the Sox10 expression might underlie the recessive red phenotype. Alignment of the pigeon reference genome assembly (a recessive red Danish tumbler) upstream of the Sox10 to the orthologous regions of the chicken and zebra finch genomes identified a 7.5 kb (7500 bases) deletion in the pigeon genome. Furthermore, four recessive red birds in their genome re-sequencing panel—but no wild-type birds—were homozygous for this deletion (named e1 allele). Importantly, the deletions in pigeon, chicken, and mouse all span a conserved enhancer element that drives the Sox10 expression in melanocytes.

To test for broader association between the pigeon Sox10 enhancer deletion and recessive red phenotype, they genotyped 41 recessive red pigeons from 19 breeds and 103 wild-type pigeons from 45 breeds. They found that 21 recessive red birds (but no wild-type birds) were homozygous for the deletion harboured by the reference genome (a recessive red Danish tumbler). An additional 17 of the recessive red birds (but no wild-type birds) were homozygous for a second, 2.5 kb (2500 bases) deletion (named e2 allele) that partially overlaps e1 allele, and the remaining three birds were heterozygous (e1//e2).

Since both pigeon deletions span the Sox10 melanocyte enhancer, they predicted that the reduction in Sox10 expression in recessive red birds was due to a cis-regulatory change,, from the related test they confirmed it. Saying that these results also demonstrate that the increased pheomelanin production due to mutations in Mc1r locus of mammals and the Sox10 locus in pigeons are not orthologous (two genes are said to be orthologus if they are diverged after a speciation event). It also says that , similar to the brown phenotype, recessive red appears to have evolved more than once in pigeons. While we do not observe obvious phenotypic distinctions between e1 and e2 homozygotes, it is possible that the different deletions generate subtly different effects on color by altering other unidentified regulatory elements. So, one of the identified alleles might be Ember??, Since that is not clear from the report, and also ember show considerable phenotypic difference to recred in adult plumage, we can assume that both are recessive reds.

The epistatic relationship of recessive red mutation to B locus is now easily reconciled in light of their molecular identities and mutations: Sox10 directly regulates Tyrp1 expression in melanocytes , which explains how loss of Sox10 expression abrogates phenotypic effects of Tyrp1 genotypes. **Interestingly, the recessive red phenotype caused by Sox10 down-regulation is distinct from the brown phenotype of Tyrp1 loss-of-function mutants, possibly owing to contributions of additional Sox10 regulatory targets or residual b allele activity.**

So that's what they gave in the report. From the diagram below you can see how Sox10 directly regulates Tyrp1 expression in melanocytes. ~~~~~~pg.31



Transcriptional activation of the Sox10 and Tyrp1 genes in the “melanocytes”.

Starting from the left-down-side you can see an unknown gene code for a protein, which then goes and binds to the enhancer site of the Sox10 gene and then Sox10 transcribes normally and produces SOX10 protein, which then goes and binds to the enhancer site of the Tyrp1 gene, then the Tyrp1 transcribe normally. But in the case of recessive red birds, the enhancer site of Sox10 is deleted, so the activator protein cannot bind there and activate Sox10 transcription,, so it results in down-regulation of SOX10 protein. The reduction in SOX10 protein affects Tyrp1 gene expression as the SOX10 protein needs to activate Tyrp1 gene transcription,, so the expression of Tyrp1 is also down-regulated. So that’s how the recessive red mutation is epistatic to the colour locus (Tyrp1).

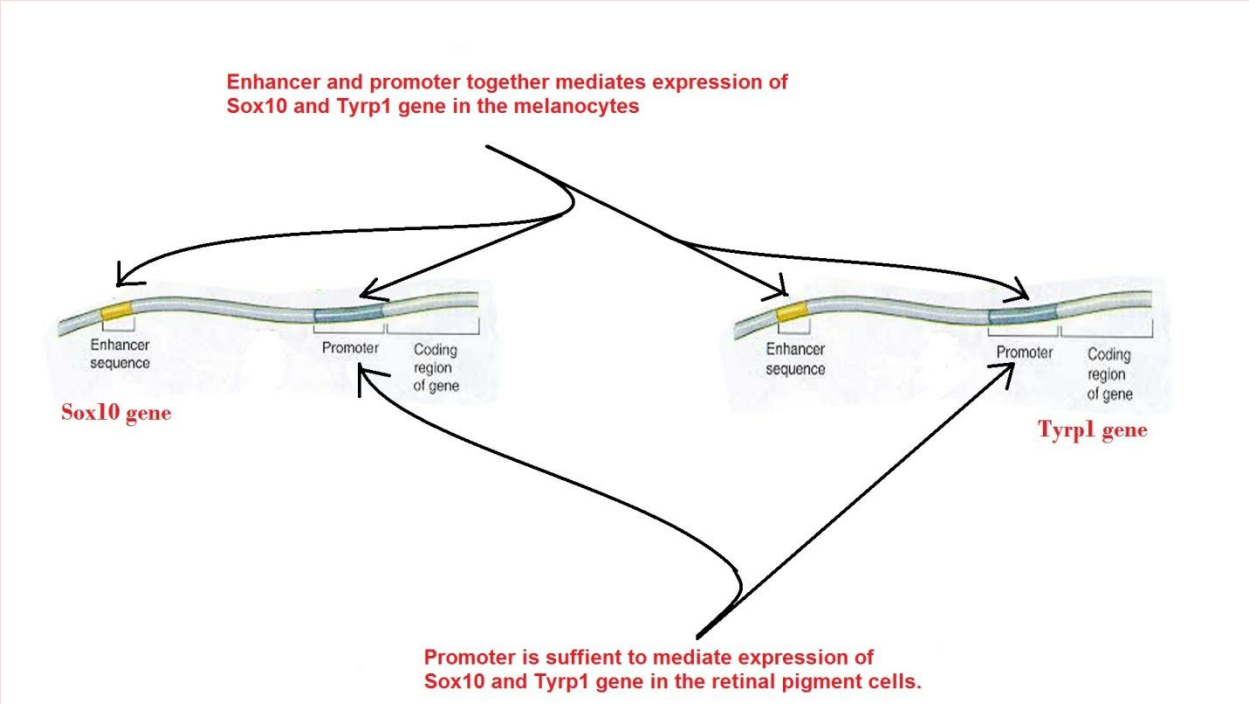
Here you can see values of relative expression of Tyrp1 gene (colour locus) in melanocytes of blue/black, brown and recessive red birds that I simply copied from the report of University of Utah.

Tyrp1 relative expression: blue/black = 1 +/- 0.556
Tyrp1 relative expression in Brown = 0.009 6 +/- 0.005
Tyrp1 relative expression : recessive red = 0.0001 6 +/- 0.00006

Expression in blue/black is the normal level of expression and we know that. In the case of brown we know that it happened in the Tyrp1 gene and Lack-of-function of the gene results in brown phenotype. But in case of Recessive red?? The mutation is indirectly affecting expression of theTyrp1 gene .Anyway incase of both brown and recessive red mutations the “change in the expression of Tyrp1 gene is almost the same”… in both cases expression of the gene is much less compared to the expression in blue/black birds. You can see that from the values I have given above. But then why do both mutations result in completely different phenotypes??.. That’s the reason the researchers think that **the protein coded by the Sox10 gene probably works on additional genes involved in the melanin production beyond Tyrp1 gene (colour locus) OR the recessive red phenotype is the result of residual (b) allele activity.**
This is something not given in the report of U of U, but from my own understanding after reading many such reports in human and other mammal genome research and of course a little bit of pigeon breeding experience. I suppose, it is not proven in birds yet . But when looking at some examples I think it may be ~~~pg.32

the same case in pigeons also. In future, It may be proven as not the case,, but I believe there is nothing wrong in sharing my thoughts with you guys……

We know that browns have pink eye in the nest and it is because of the lack of the melanin which is normally present in the inner wall of the retina at the back of the eyes. It is due to lack of expression of Tyrp1 gene. But in the report published by the U of U we saw that in the case of both brown and rec red mutations it affects Tyrp1 expression and in both cases it results in reduction in the gene expression., but then why doesn't recessive red show pink eyes in the nest?? In fact they have normal black eye in the nest unless they are brown based and/or have some other mutation which causes pink eyes…..Here is a possible reason behind it.....

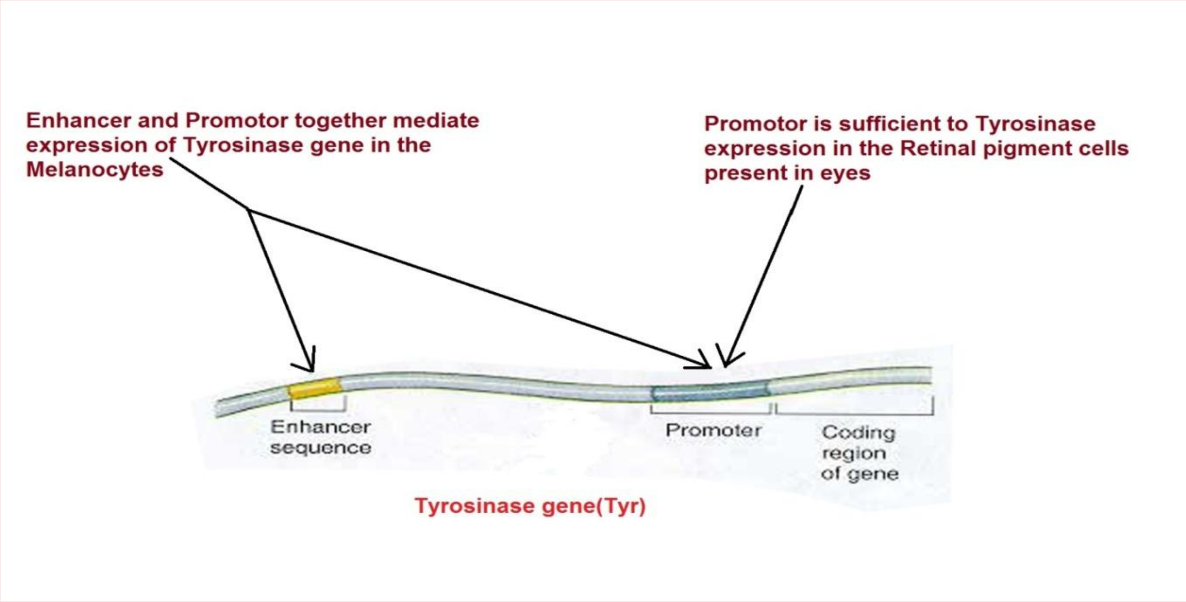


Different cis-acting elements are involved in RPE and melanocyte-specific gene expression of Sox10 and Tyrp1genes in mammals. I suppose it is not studied in birds, but might be the same in birds also.

Melanocytes arise from the neural crest whereas Retinal pigment cells (cells that produce melanin in the eyes) originate from the optic cup of the developing forebrain. pigmentation genes are controlled by different regulatory networks in melanocytes and retinal pigment cells. In melanocytes both enhancer and promoter needs to express the Sox10 and Tyrp1 gene normally, whereas in the retinal pigment cells present in the eyes the gene expressions are regulated by their promoters. So deletion in the Enhancer site of Sox10 gene does not affect expression of Sox10 and Tyrp1 gene in the retinal pigment cells. "

We know that there are similar examples present in pigeons like Recessive white (There are more similar examples). Recessive white cuts off melanin production from the feathers, skin, beak and toe nail, but they have black eyes in juvenile as well as in adult plumage, so the melanin production in the eyes of the recessive white bird is normal like the wild type specimen, unless the bird is brown based and/or it has some other mutation which causes pink eyes. The difference in the melanin pigment level in eyes and on feathers (skin and beak) might be because pigmentation genes are controlled by different regulatory networks in the melanocytes and retinal pigment cells.

Here is a possible reason for the case of recessive white. ~~~~~~~pg.33



Different cis-acting elements are involved in the RPE and the melanocyte-specific gene expression of the tyrosinase gene in mammals. I suppose it is not studied in birds, but might be the same in birds also.

Tyrosinase is the key gene in the melanin biosynthesis and is known to be involved in the first two steps of melanin production and a step downstream in the eumelanin production. So mutation in this gene can result in albinism. But what if a mutation happens somewhere in the regulatory region of the Tyrosinase gene, like the enhancer site of Tyrosinase or to a gene that encodes for the activator protein that transactivates the enhancer site of Tyrosinase?. It can result in down regulation of the Tyr gene in the melanocyte, but since the promoter is sufficient to control expression of Tyr gene in the retinal pigment cells, the mutation will not affect expression of the gene in the retinal pigment cells present in eyes. I think such a case is possible in the case of recessive white.

To those who think it is bit complicated to understand,,, yes it can be. But if you have understanding in Mendelian genetics of pigeon, read it two or three times, go through the diagrams and imagine it in your mind then probably you will get it.

“ The power of imagination makes us infinite.” – John Muir

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<u>Below a bit more on the topic of Sooty Factor !</u>
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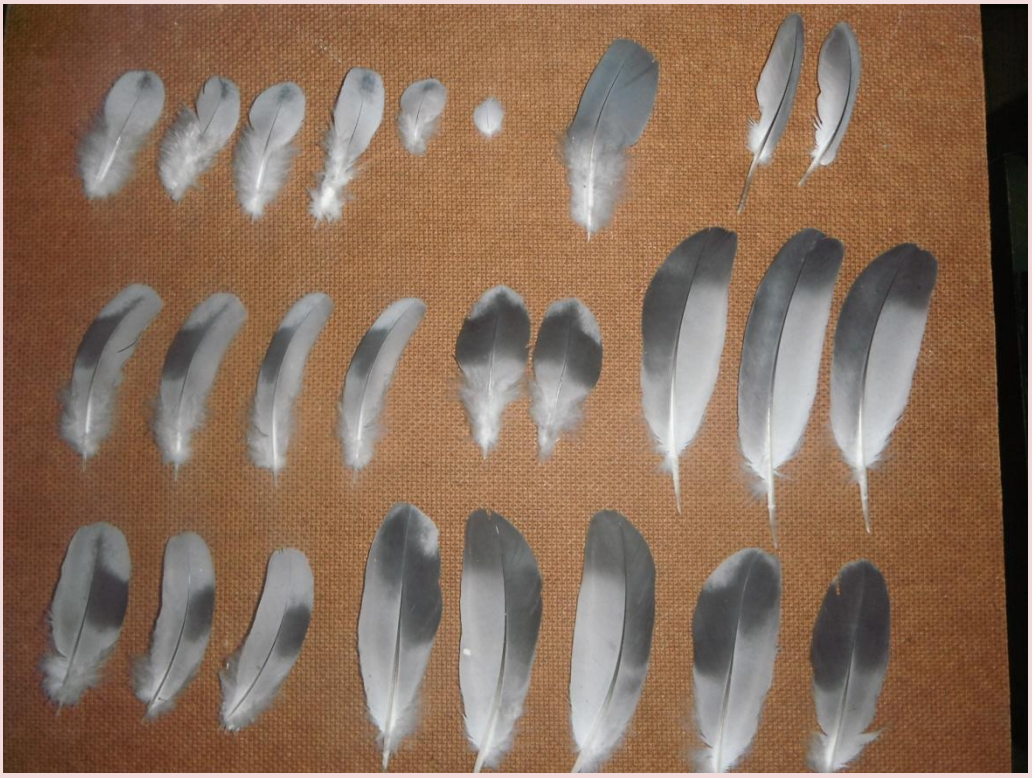
There may be more than one type of Sooty . We know that one early publication in the U.S.A stated that it had been considered to be a recessive , but was later proven to be Dominant with symbol (So). Then a recent publication out of Germany has it listed as a recessive gene symbol (so) .

There are those who have said that it does not show in the juvenile feather ., but increases with each successive moult. Others have reported a strong expression in the juvenile plumage that decreased after the first moult .

Below : Sooty factor (dirty/smoky) ? ash-red bar by Vince Van Royen , courtesy Layne Gardner .

pg.35

Moulted feathers of the above Sooty Factor Blue bar Feral . **{Photos by Editor }**



Below is a normal Coarse spread bar pattern feather , and dual sided Checker pattern marking. Note that the Sooty on the top bar feathers above , extends along the rachis , whereas here the bar is clean cut .

Sooty Blue bar American Show Baldhead Roller (Jay Turner) courtesy Layne Gardner .

Often Classical Grizzles and smoky factor birds are mistaken as Sooty Factor birds . Perhaps we can compile a few photos in the future to demonstrate how this can happen .

Sooty , Dirty Blue bronze Bar pied , with one unknown factor , post in Facebook by Pedro J.P. Bento .

Until the New Year , that is it from us here in the Pigeon Coop , Now lets hear from You ! ~~pg.38