

Figure 1. Eumelanin and pheomelanin distribution in adult plumage for the known *E* locus alleles. *E* (extended black (*E*)), *R* (birchen black (*ER*)), *N* (wild-type (*e+*)), *B* and *BC* (brown (*eb*) and buttercup (*ebc*)) and *Y* (recessive wheaten (*ey*) and dominant wheaten (*eWh*)). Anderson *et al*., 2020.

**The Chicken *E* Locus Phenotypes**

1/17/2025

In the Anderson et al., 2020 chapter on feather color genetics we were limited on space that could be dedicated to the *E* locus, so I wasn’t able to fully describe the *E* locus (melanocortin 1 receptor, *MC1R*, gene) and the phenotypes associated with the various alleles. The *E* locus is very important for the understanding of the genetics of feather color, so I am expanding the explanation of this important locus before I get into how it fits in with what we have figured out about the genetics of feather color and patterns.

The *E* locus has been associated with the melanocortin 1 receptor (*MC1R*) gene. There is still uncertainty about the causative mutations for the various alleles. What I will try to describe in this article is what is known about the phenotypes associated with the *E* locus alleles, that had been characterized by 1990, when those phenotypes are expressed on a wild-type genetic background. Smyth (1990) described the *ebs* allele, but I do not know if I’ve ever had any experience with that allele. Murray McMurray Light Brown Leghorns did produce some chicks with speckled heads, but I do not know if it is what Smyth was describing. The Light Brown Leghorns did segregate for *eb* and it was the same *eb* allele (by coding sequence) as I found in the Smyth Brown tester line. I will likely have to explain how the basic *E* locus phenotypes are affected by some other feather color loci and bring in what we know about what *MC1R* genotypes are associated with those colors and patterns. I intend to write up something on the molecular biology and specific alleles in later posts. The uncertainty of allele association with phenotypes may be due to regulatory variants (only the coding sequence of the *MC1R* gene has been evaluated at this time) or it may be due to closely linked genes that modify the *E* locus phenotypes. It may even be due to modifying genes that are not closely linked to the *E* locus. Smyth (1990) speculated that the E locus could be a cluster of genes associated with the *E* locus phenotypes. We haven’t done the work that would associate flanking genes with some of the *MC1R* phenotypes.

Figure 1 has the different parts of the bird labeled. For illustrative purposes the distribution of eumelanin and pheomelanin is depicted on a basically wild-type genetic background. I had to use recessive yellow skin (*w*) and dominant sex-linked dermal melanin inhibitor (*Id*) when white skin (*W+*) and dermal pigmentation (*id+*) are the wild-type alleles. If I had not done this the epidermal pigmentation of the shank would not have been easily differentiated in the illustration. The *E* locus is also associated with pigmentation of the epidermal shank scales (scutes).

The phenotypes in Figure 1 come from the old literature and my personal experience. There will be other articles about what is known about how the *E* locus alleles interact with other feather color loci to create the various color patterns that exist in domestic chickens. I recommend Smyth’s 1990 chapter on feather color genetics in Poultry Breeding and Genetics. Smyth cites just about all the relevant research on the subject up until that time, and we really haven’t added much to it since then except the publications of W. Clive Carefoot (I recommend looking up Carefoot’s work). The paintings of the breeds in the American Poultry Association’s Standard of Perfection can be used to get an idea of what the breeds look like that I mention. You can also go to the Murray McMurray Hatchery web site (link provided in References).

There are two basic types of melanin. Eumelanin is the black (dark brown) pigment and pheomelanin is the yellow to red pigment. The *MC1R* gene somehow regulates the production of eumelanin and pheomelanin. *MC1R* is a G protein-coupled receptor, and sends a signal that induces eumelanin synthesis when the melanocortin ligand binds to the receptor. Somehow the regulation of this receptor can switch the melanosome from making pheomelanin to making eumelanin. When the MC1R receptor is inactivated only pheomelanin is produced. The original mouse recessive yellow *e* allele was eventually found to be a frameshift MC1R gene knockout mutation (Hauschka, et al., 1968 for initial phenotype description before the causative gene was known). Melanin has not been completely characterized in chickens as one might expect since it has been easier to look at feather melanin than skin melanin. What is disconcerting is that there is still disagreement about the shape of pheomelanin melanosomes (Galvan and Solano, 2016). By the time that we usually examine them the pheomelanosomes are mostly round structures, but the claim is that they do have a more elongated shape when they are first made, but that they are less stable and eventually round up. Trichochromes (another type of pigment molecule classified as pheomelanins) have been extracted from gold and red feathers. Smyth, 1990 cites some of his studies that extracted two types of trichochromes from brown body feathers, but only one type from the salmon breast feathers. Galvan and Solano, 2016 in their review lists 4 pheomelanin trichochromes found in red feathers, but their B and C types and their E and F types might be difficult to differentiate from each other, so Smyth may have only differentiated BC and EF types (Galvan and Solano, 2016, Figure 2). Smyth notes that after extraction of the trichochromes from the feathers with solvent that the feather color doesn’t change, but these are supposed to be pheomelanin pigments. There may be two types of pheomelanin produced in the chicken. The pheomelanin found in the hackle in males and females and wild-type body of the hens seems to be one type of pheomelanin. The salmon pheomelanin of the wild-type hen’s breast may be another type. Smyth found that one type of trichocrhome was missing from the salmon pigmented breast feathers.

The two pheomelanins are differentially affected by the sex-linked silver locus. We do not know what the difference is between the two types, and it could be a regulatory difference in how pheomelanin production is initiated. The dominant sex-linked silver (*S*) allele dilutes pheomelanin. You get white feathers in a sex-linked silver bird (*S*) where the feathers would be yellow to red in an (*s+*) sex-linked gold bird. The sex-linked silver allele does not effectively dilute the salmon color of the hen’s breast. The salmon breast may be diluted a little, but the breast retains the salmon color in breeds like the Silver Leghorns. There is some brownish pigment in the wings and body feathers of silver *e+* hens that does not seem to be diluted completely by silver. This residual brown may be a normal expression of the *e+* allele because it seems to take selective breeding, likely involving other modifiers to reduce it in silver breeds to get the clean gray wing and body feathers. The pheomelanin expressed in the male wingbow (sometimes called the shoulder, but it isn’t a forelimb shoulder) is often not diluted effectively by sex-linked silver and may be this same salmon pigment to some degree. This salmon pigment may also be associated with what was claimed to be autosomal red. I can’t find the references that referred to autosomal red, but it was pheomelanin that was resistant to sex-linked silver and did not seem to be dependent on the sex-linked gold allele for it’s expression. Autosomal red is the bane of Silver Penciled Rock breeders that have tried to improve their lines by crossing in Partridge Rock lines only to find a pheomelanin enhancer coming from the Partridge Rocks that isn’t diluted by sex-linked silver and seems to be associated with the cleaner penciled pattern of the Partridge Rocks. They get red pheomelanin expression messing up their silver pattern producing a rusty looking silver bird.

Wheaten hens may have the salmon pigment distributed over their whole bodies because sex-linked silver does not effectively dilute the wheaten hen body feather color. Salmon Faverolles are likely dominant wheaten and they are sex-linked silver on an otherwise wild-type feather color genetic background producing wheaten hens and black breasted silver males (Figure 1). Silver wheaten males may have the salmon pigment in their body feathers (as do the females) because the sex-linked silver Salmon Faverolle males retain some pheomelanin in their hackles, wingbows, secondary flights, back and saddle. It may be that the wheaten *E* locus allele may allow the distribution of the salmon pigment over the bodies of both males and females.

Sex-linked silver will dilute the pheomelanin in the body feathers other than the breast in wild-type (*e+*) hens. The pheomelanin in the body feathers of *eb* hens is also diluted by sex-linked silver. The *e+* and *eb* hen’s body feathers look brown due to pheomelanin with interspersed eumelanin “stippling”. Stippling is the pattern of tiny black spots distributed across the wing and body feathers of the hen. Sex-linked silver and otherwise wild-type hens retain the salmon breast, but the body and wing feathers become gray as the black stippling is now interspersed on a white background. The example given was Silver Leghorns. Silver Leghorns are *e+*/*e+* and may have additional modifiers that remove the pheomelanin from the males wingbow. Other silver breeds with the *e+* allele can retain some pheomelanin in the wingbow and saddle as does the Old English Game Gold Duckwing, but it may take additional modifiers to keep this color because the hackle and secondary flights can be white in the Gold Duckwing birds.

What is not noted in Figure 1 about the adult feather color is the color of the fluff at the base of the body or contour feathers. This feather undercolor is not visible unless you part the feathers and look at the color of the fluff. *E*, *ER*, *e+*, *eb*, and *ebc* alleles produce a gray undercolor, but in wheaten birds (both *ey* and *eWh*) the undercolor is cream or white. The undercolor can be darker red in Rhode Island Reds that might have recessive wheaten or dominant wheaten, so the color can be influenced by pheomelanin intensifiers, but wheaten alleles appear to reduce eumelanin expression in the feather undercolor.

**Dominant white:**

Dominant white (the *I* locus) may tell us something about feather pigmentation in terms of eumelanin and pheomelanin regulation. Hutt (1949) and Smyth (1990) both note that the dominant white allele (*I*) effectively dilutes black eumelanin, but is not effective in diluting pheomelanin. Red Pyle Games are an example of dominant white on an *e+* black breasted red genetic background. The normally black feathers are white, but the gold feathers remain gold. This also tells us that feathers that were destined to be black exclude the synthesis of pheomelanin even after the exclusion of eumelanin by dominant white. The decision to make eumelanin is probably initiated before pheomelanin, and the dominant white allele block of the distribution of eumelanin comes after black pigment synthesis has initiated. Pheomelanin synthesis is not initiated even though eumelanin distribution is blocked in feathers that were destined to be black.

We found that the dominant white locus was the *PMEL17* gene (Kerje et al., 2004). Somehow eumelanin melansome production and transport is inhibited even in *I*/*i+* heterozygous melanocytes thou the inhibition shows incomplete dominance. Pheomelanin melanosome production and transport is not as severely restricted. Hamilton (1940) found that White Leghorn (*I*/*I*) embryo cultures formed melanophores, but only small melanin granules, while *I*/*i+* heterozygotes (White Leghorn X Barred Rocks) cultured melanophores produced melanin granules about as large as Barred Rock cells, but the pigmented melanophores survived only a few days longer than the White Leghorn cultured melanophores, while the Barred Rock melanophores were viable for more than 10 days. Hamilton concluded that compromised viability of the melanophore resulted in the lack of pigmented feathers in the *I*/*I* homozygote and *I*/*i+* heterozygote. We found that a 9 base-pair insertion in exon 10 of the *PMEL17* gene was associated with the dominant white phenotype (Kerje et al., 2004). PMEL17 is required for eumelanin polymerization and melanosome formation, but the altered PMEL17 protein function may result in the death of the melanocyte so that the melanin that is produced in the heterozygote is not used in the feather follicle. A review by Raposo and Marks (2007) cites Hamilton (1940) and reviews more recent work on PMEL17 function. PMEL17 is involved in the formation of the premelanosome by creating the microfibrils onto which melanin is polymerized to produce the mature melanosome. So PMEL17 is required to initiate eumelanin melanosome formation, polymerization of the eumelanin, and is needed to keep the cell alive so that the melanosomes can be excreted and absorbed by the keratinocytes (keratinocytes are skin cells that also form the feathers).

In a review by D’Alba and Shawkey (2019) the claim is made that PMEL17 is suppressed in the development of pheomelanin melanosomes, and that PMEL17 is not needed to form the microfibrils in pheomelanin melanosomes that is required for the development of eumelanin melanosomes. Pheomelanin melanosomes develop without developing the PMEL17 microfibrils, and melanocytes making pheomelanin melanosomes do not seem to have the viability issues of eumelanosome forming melanocytes. Eumelanin production is initiated involving the *E* locus, but the eumelanin melanosomes do not mature in *I*/*I* homozygotes and the cell dies. In *I*/*i+* heterozygotes eumelanin melanosomes can develop, but the cells die and do not transfer the melanosomes to the keratinocytes. Melanocytes producing pheomelanin containing melanosomes avoid this fate (pheomelanin is produced in the absence of MC1R signaling). PMEL17 is not as important in the production of pheomelanin melanosomes and the viability of pheomelanin melanocytes. Dominant white does dilute pheomelanin in the feathers, but not to the extent that eumelanin is inhibited. It may be that production of defective PMEL17 is required to induce early melanocyte death. Enough PMEL17 is produced in the *I*/*i+* heterozygote to prevent the transfer of melanosomes to the keratinocytes and cause the early cell death of the melanocyte, but somehow pheomelanosome containing melanocytes avoid this fate.

**Extended black alleles *E* and *ER*:**

The *E* locus was named the extension locus for chickens and other animals exhibiting similar extended black melanin phenotypes. As the name implies the dominant extended black *E* allele extends black feathers into normally nonblack feather tracks. There is current uncertainty about what the difference between the *E* and *ER* (birchen) alleles could be in terms of the coding sequence of the gene. Davilia et al., 2014 found the two most common Lys92 (the MC1R protein has a lysine amino acid at the 92nd position of the protein) alleles associated with both birchen and *E* allele black breeds (Thr71-Lys92 and Met71-Lys92). Campo and Alvarez, 1993 did find evidence that separate *E* and *ER* alleles existed, and they found that the *ER* allele could produce the extended black phenotype when combined with melanotic (*Ml*). If the Met71Thr polymorphism is the difference between alleles the Thr71 variant (possible *E* allele) needs to somehow be attenuated in birchen breeds. I will discuss this when I get around to the molecular biology of MC1R. I think that Thr71-Lys92 may be the *E* allele because it has the highest frequency in black breeds that I have looked at while the Met71-Lys92 allele is found at the highest frequency in breeds like Laced Polish that need the birchen allele to produce their laced feather pattern on a black feathered background. Thr71 is just one amino acid away from Leu69 (melanic mouse variant that results in constitutive activation of the of MC1R as does the Lys92 variant). The Thr71 may enhance the constitutive activity associated with the Lys92 variant in some way. The Gln133 Fayoumi birchen allele described by Smyth (1990) was also found to segregate in birchen and *E* breeds in the flocks Davila et al., 2014 tested, but the Gln133 polymorphism was more common in birchen breeds.

The *E* allele is not fully dominant. When columbian (*Co*) and wheaten are present in the cross you can get nonblack feathers in both male and female *E*/*eWh* hybrids, but females can have more gold color in their hackles. The commercial black sex-linked cross of a Rhode Island Red male X Barred Rock female is such a *Co* cross (*E*/*eWh*). In some crosses that I made that may not have had *Co* present (I do not know if Australorps have *Co* like Barred Rocks and Rhode Island Reds) the heterozygous (*E*/*e+*) females were mostly black with a few nonblack feathers, while males had nonblack in their hackles saddles and wingbows. There are likely eumelanin modifiers that increase eumelanin in the feathers to create the show quality black breeds.

The birchen *ER* allele is known to segregate in black feathered breeds and eumelanin intensifiers would be needed to produce a fully black *ER/ER* homozygote. Without the intensifiers you produce the Old English Game birchen phenotype depicted in Figure 1. The *ER* allele does extend black into the secondary flight feathers and they become all black producing an all black wing depicted in Figure 1. Half (one side) of the secondary flight feathers are exposed when the wing is folded and the pheomelanin creates a color patch of the wing for mating display of the *e+* Red Junglefowl. This wing patch becomes black in birchen breeds. The *ER* allele leaves the male hackle, wingbow, back and saddle to express pheomelanin. Some of the body feathers and wing feathers may not be saturated with black pigment, and Smyth calls it “finely Stippled”. This is important to note because it is the birchen black allele that is used to create the autosomal barred and lacing of breeds like autosomal barred Campines and laced Polish and Sebrights that exhibit the laced tailed phenotype. *Pg* seems to be involved in rearranging the stippling on the feathers of partridge, penciled and laced breeds, and the *ER* and *eb* alleles are susceptible to this pattern manipulation in the presence of other genes like *Co* and *Db*. Smyth, 1990 notes that *ER*/*eWh* heterozygotes can have the *eb*-like (brown) body feathers in females, but *ER*/*ey* females are black feathered. I once crossed a White Crested Black Polish male to my “free” chick Salmon Faverolle female when I was raising birds in my backyard and was doing color crosses. I got poorly crested birds with muffs and beards and feathered shanks. The females had brown feathers with coarse stippling. This is probably evidenced that Salmon Faverolles do have dominant wheaten. Polish are usually claimed to have the birchen allele for White crested, Silver and Gold laced varieties.

The birchen black breeds usually have breast lacing as a breed characteristic, but this breast lacing has not been determined to be due to the *ER* allele. You can see this lacing in birchen Modern Games and Old English in the upper breast feathers of both males and females. It may be that the *ER* allele only allows breast lacing to manifest, and that breast lacing is due to other feather color loci. Campo and Alvarez, 1993 found Columbian locus *Co* allele in their birchen breed with breast lacing.

Both *E* and *ER* are incompletely dominant for the shank color. Heterozygotes with nonblack *E* locus alleles usually do not have fully black shanks in the crosses that I’ve made with *e+* and *eb* alleles. There are some nonblack shank scales on the shanks of the hybrids (some can have less black on their shanks than nonblack). Homozygotes can have fully black shanks, but there seem to be modifiers that can decrease the black in homozygous birds, or it could be that modifiers are needed to get clean black shanks. Something allows white or yellow to show in the shank and toes of homozygotes. Recessive mottling (*mo*) is known to remove eumelanin from the shanks of extended black birds. An example is the Ancona with it’s black mottled (about 1 in 5 feathers have a white tip) plumage and yellow shanks.

**Wild-type *e+*:**

For the nonblack alleles the males all have the black breasted red phenotype of the Red Junglefowl. The *e+* female has the Red Junglefowl pattern with pheomelanin in the hackle, body, and a salmon colored breast. The wings and body feathers have pheomelanin expressed with black eumelanin stippling (brown body feathers). The main tail feathers are darker brown with both coarser and finer stippling than the wings, but are surrounded by brown feathers exposing only the ends of the darker tail feathers. In Figure 1 I depict the main tail feathers of the female as black, but they are only darker brown than wheaten tail feathers (wheaten tail feathers have reduced eumelanin expression compared to *e+* and *eb*). The tail feathers can look black but the color isn’t saturated like the black you get in the tails of Columbian restricted hens. Light Brown Leghorns have the wild-type feather color pattern. The *e+* allele is recessive to the black alleles, but is dominant over *eb* and *ey*. Smyth, 1990 describes the *e+*/*eWh* female heterozygotes as having intermediate wheaten-brown body feathers. This likely means that there is less black stippling in the feathers of the heterozygote, and the pheomelanin pigment may have more salmon color than wild-type.

**The brown or partridge, and buttercup alleles, *eb* and *ebc*:**

The *eb* allele has been traditionally called the brown or partridge *E* locus allele. It is the allele found in Partridge Rocks and Dark Brown Leghorns. When the *MC1R* gene from the Buttercup breed was sequenced it was found to have the same coding sequence as the *eb* allele from other breeds including the Smyth Brown tester line (Kerje et al., 2003, Ellett, 2000). Smyth indicates that Brumbaugh and Hollander identified *ebc* as being different from *eb* allele, and noted his own work indicating that it was a distinct *E* locus allele in it’s effect on down pattern (Smyth, 1990). The Buttercup allele could still be a unique *E* locus allele, but the difference would have to be regulatory or due to a closely linked gene because the protein has the same coding sequence as the *eb* allele. The *eb* female lacks a salmon breast. The entire female body has the brown stippled feathers. I worked with the Smyth Brown tester line (fixed for the *eb* allele) that were essentially the Light Brown Leghorn color pattern with the *eb* allele and the brown female feather pattern. This line did have more eumelanin expression in the shanks that made them look a little brownish, and may have been a show defect. This is not the red shank color that runs down the shanks of some yellow and white skinned breeds that is not supposed to be counted as a show defect. The darker shank in Figure 1 is an exaggeration. The brown is not that noticeable, and show breeders have selected for a cleaner yellow shank in breeds like Partridge Rocks that are based on the *eb* allele. There is likely always a little leakage of eumelanin into the shank scales of *e+* birds, but there seems to be a little more in *eb*/*eb* individuals. It is not that noticeable and from a distance they look like typical yellow shanked brown leghorns.

The *eb* allele is recessive to the black alleles and the *e+* allele. The *e+*/*eb* heterozygous females have the wild-type feather pattern that includes the salmon breast in females. I found the *eb* allele segregating in the Murray McMurray Light Brown Leghorns. This was consistent with their claim that their line had been improved using the Danish Brown Leghorn layer line that had been known to segregate for the *eb* allele.

**The wheaten alleles *eWh* and** ***ey*:**

The existence of dominant and recessive wheaten alleles is controversial. Smyth (1990) notes that Carefoot (1981) found that the difference between dominant and recessive wheaten could be due to residual modifiers (modifiers are just other genes that affect the trait). There may only be one recessive allele, and dominance may be associated with other feather color loci influencing expression. There is the possibility that dominance is associated with only certain phenotypes that are not always scored for. Morejohn (1953) identified a recessive wheaten allele segregating in the Red Junglefowl line that he had been working with. The publication has a picture of wheaten yellow down chicks appearing among sibs with wild-type striped down. 2/3 of those striped down sibs would be expected to be heterozygous, but they showed no evidence of wheaten. This seemed to be a recessive wheaten. Dark Cornish and Rhode Island Reds were supposed to have recessive wheaten, but New Hampshire Reds (derived from Rhode Island Reds) were supposed to have dominant wheaten. Commercial layers rely on dominant wheaten in their sex-linked Silver and gold down color sexing lines.

What I found was that commercial layer Rhode Island Reds (bred for sex-linked down color sexing) and Murray McMurray New Hampshire Reds both had the same *MC1R* coding sequence with the Thr143Ala amino acid substitution. Commercial layers rely on dominant wheaten to remove the eumelanin usually expressed in the down of Columbian restricted chicks. Removing the eumelanin makes down color sexing (sex-linked silver and gold) more accurate. The dominant *Co* allele is needed to evenly distribute gold pheomelanin around the backs and heads of chicks, but *Co* is also associated with increased eumelanin expression on the backs and heads of the chicks. Wheaten reduces the eumelanin and allows the pheomelanin to be more easily quantified. You get full expression of pheomelanin on chicks with sex-linked gold, and the dominant sex-linked silver allele dilutes the pheomelanin. The point is that this wheaten allele shows some dominance on chick down because the commercially sexed product has reduced black in the down and is *eWh*/*eb*. It is known that *eb*/*eb* chicks show the black down on their backs in the presence of *Co* while wheaten chicks do not show the black on their backs in the presence of *Co* (See Figure 2). So there seem to be recessive wheaten alleles and wheaten alleles that show some dominance in the chick down.

I found the same Ala143 allele in Buff Minorcas (acquired from Murray McMurray) that are supposed to be mainly *ey*, but can have a mix of alleles. I only had half a dozen females to test, but they were all homozygous. These chicks have a buff down with no striping.

Salmon Faverolles are sex-linked silver instead of sex-linked gold, but they exhibit the wheaten pattern depicted in Figure 1. Wheaten birds have weak to no black hackle striping but there are modifiers such as *Ml* that will darken the hackle and body feathers. In the APA Standard the Cubalaya and Malay Black Breasted Red males and females are wheaten along with the wheaten Game fowl. Eumelanin is reduced in the hen and the wheaten salmon pigment is distributed over the entire body of the hen. The main tail feathers have eumelanin reduced from nearly black to a wheaten brown. As noted before this pattern includes a cream undercolor for the body feathers. The other alleles have a gray undercolor, but wheaten removes the eumelanin from the undercolor. New Hampshire Reds and commercial Rhode Island Reds have the cream and reddish undercolor, respectively. When I crossed New Hamps to Light Brown Leghorns the *eWh*/*e+* adult hybrids were poorly Columbian restricted, but the chicks had reduced eumelanin on the backs of the down (Figure 2). Like the commercial sex-linked cross with *eb* the wheaten allele reduces the eumelanin from the *Co* restricted back down. Figure 2C has an example of *Co* related eumelanin on the head and back of a chick. Some of the *eWh*/*e+* *Co*/*co+* chicks retained a pretty good wild-type stripe down pattern (Figure 2B) but eumelanin expression was reduced. The black eumelanin stripes flanking the main middle dark brown stripe were missing or diluted and not continuous. When these hybrids feathered out they were partially Columbian restricted with a light gray undercolor, so the removal of eumelanin from the undercolor was not completely dominant in this cross. I should note that the male hybrids were more Columbian restricted than the females (they had less black in their body feathers). We seem to have incomplete dominance, or the undercolor could be influenced by *Co* and remains gray (*Co* is a black restrictor, but it also has eumelanin enhancing properties). Wheaten reduction of eumelanin in both the chick down and the feather undercolor seems to show incomplete dominance.

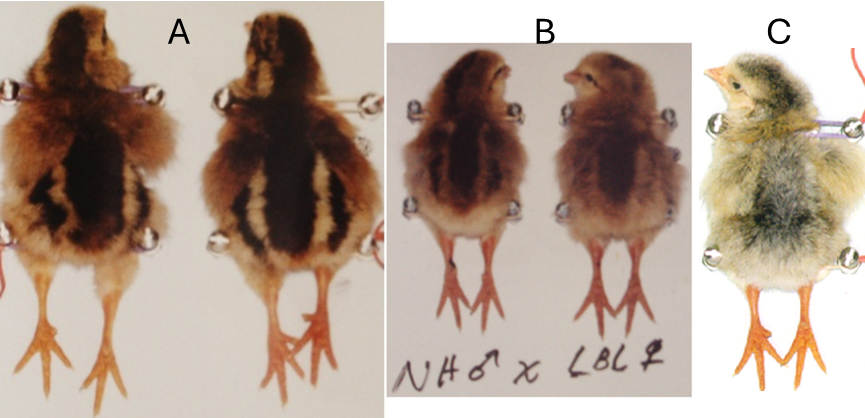


Figure 2. A. is two Murray McMurray Light Brown Leghorns (one with speckling on it’s head). B. has two chicks from a cross of a Murray McMurray New Hampshire Red male to the Light Brown Leghorns (*eWh*/*e+* *Co*/*co+*). C. is a sex-linked silver chick that shows the black down on the head and back that you get if dominant wheaten is not involved. *Co* restricted chicks can range from a light dusting of black down on the back and head to essentially black down. The Silver Columbian Wyandotte sire of this chick had darker down. You can go to the Murray McMurray web page and look up Buff Brahma and Columbian Wyandotte to see chick pictures and see how dark the down can get with *eb*/*eb* *Co*/*Co* chicks (Link provided in References).

Salmon Faverolles are supposed to have dominant wheaten and they have the cream undercolor. The dominance could be tested by crossing Salmon Faverolles to Light Brown Leghorns and observing the hybrids at hatch and in their adult plumage to determine the dominance of the allele. They might be like Morejohn’s chicks, or they may show wheaten down types and wheaten in the hybrid females adult plumage including the undercolor, as indicated in some of the old literature about dominant wheaten. Morejohn’s recessive wheaten obviously had heterozygous (*e+*/*ey*) females that were wild-type in both chick down and adult plumage, but for dominant wheaten the heterozygous (*eWh*/*e+*) females would be expected to have intermediate down type, intermediate adult plumage and lighter feather undercolor.

As noted dominant and recessive wheaten may be associated with other factors that influence the phenotype. It might be due to regulatory differences or the influences of a closely linked gene. The Speckled Sussex is supposed to be recessive wheaten, but one hen had the same *MC1R* coding sequence (Cys213) as a Red Junglefowl and Light Brown Leghorns that I had also sequenced (I had only sequenced one Speckled Sussex hen). It should be noted that the UCD001 reference Red Junglefowl genome sequence has the Cys213 MC1R sequence and has the *e+* allele and hen salmon breast with brown body phenotype (personal observation of the reference genome sequence). I never published the result, but another group of researchers found the same Sussex *ey* Cys213 sequence in their *ey* tester line (Davila et al., 2014). Both of the *MC1R* sequences that my lab had associated with Red Junglefowl and Light Brown Leghorns (Ellett, 2000) were found in this recessive wheaten tester line (Davila et al., 2014, Table 5, haplotypes H0 and H11 ). Davila et al., 2014 may have been dealing with a regulatory variant altering the wild-type alleles to wheaten or they may have selected for a closely linked gene that modified the wild-type *E* locus phenotype to wheaten. This “wheaten” variant might also be in the Sussex breeds. Both White Sussex and Speckled Sussex are supposed to be recessive wheaten, and our Speckled Sussex coding sequence was the same as the most common H11 haplotype sequence in the Davila et al., 2014 *ey* tester line. This same coding sequence is associated with the *e+* phenotype in Red Junglefowl and Light Brown Leghorns indicating that the difference may be regulatory or due to linked modifiers.

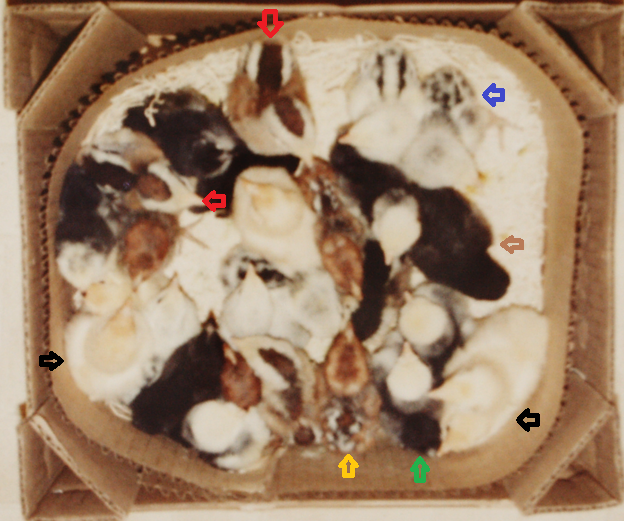


Figure 3. A box of chicks from Murray McMurray. There are 3 to 6 of each breed. **Red** arrows indicate two Silver Pheonix chicks. The **blue** arrow indicates a Silver Spangled Hamburg. The **brown** arrow indicates a Black Minorca. Another Black Minorca is between the two Silver Pheonix chicks and it shows the white down that can invade the face and head of black downed chicks. The two **black** arrows indicate Buff Minorca. The **green** arrow indicates one of the White Crested Black Polish chicks. The **yellow** arrow indicates a Silver Fayoumi chick. Of note is that the Spangled Hamburg and Fayoumi display the marbled chick down pattern that is thought to be due to the combination of *ER*, *Db* and *Pg*. Smyth (1990) has Fayoumi with *ER*, *Db*, *Pg* and *Co*, and the Spangled Hamburg with *ER*, *Db*, *Pg* and *Ml*, but Carefoot (2002) did not find evidence for *Co* in Fayoumi bantams.

**Down color:**

Each *E* locus allele has an effect on down color, and they can combine with other feather color loci to produce distinct down color patterns. Smyth (1990) doesn’t go into much detail about down phenotypes, but has pictures in Figure 5.2. Smyth refers to the pictures, but you can only see the backs of the chicks. One thing that I messed up on was recording the ventral (chest, belly and thigh), and I have to go by memory. I really should have taken some relevant pictures. I intend to write a description of the *MC1R* sequences that are associated with the known *E* locus alleles, and in this section I will only note the relevant variant amino acid positions. I also recommend going to the Murray McMurray website (link in the references) to view the pictures of the chicks of each breed mentioned. There seems to be two types of pheomelanin expressed in the chick down. One is subject to dilution by sex-linked silver and the other mostly associated with the reddish brown color is not diluted effectively by sex-linked silver, and could be related to the salmon pigment of the wild-type hen’s breast and the wheaten body feather color. You can see the difference in the Silver Pheonix chick (Figure 3). The usually gold stripes flanking the main brown dorsal stripe are diluted, but the main brown dorsal stripe is only diluted to a lighter shade of dark brown, and the thin flanking black stripes can be seen.

**Wild-type *e+* Down:**

Figure 2 has two Murray McMurray Light Brown Leghorn chicks. This is the down pattern of wild Red Junglefowl (*e+*). I used to raise Bobwhite Quail as a kid and the quail had a similar down pattern that included the bandit eye stripe that can be used to sex the Brown Leghorns with some degree of accuracy. The pullets often have a wider continuous unbroken eye stripe, while the cockerals have a thinner and often broken eye stripe. Females are also supposed to have a wider darker mid dorsal stripe (Hutt, 1949, page 218). There is a wide dark brown mid dorsal stripe enclosed by two thin black stripes. You cannot see these black edges very well in Figure 2, but the Silver phoenix (sex-linked silver *e+* down) in Figure 3, that has a lighter brown mid dorsal stripe, may show more clearly that there is a black edge to the mid brown stripe. The mid dorsal stripe is flanked by two gold (silver in the Silver Pheonix, Figure 3) stripes and then two dark brown stripes. It should be noted that the back pattern is repeated on the head with the bandit eye stripe representing the flanking dark brown back stripes, but the eumelanin seems to be more concentrated in the shorter facial down making the eye stripe nearly black. The ventral side is mostly cream colored with some gold pheomelanin along the flanks. There is some eumelanin dispersed along the sides and thigh area, but it may not be noticed until it is gone in some chicks. Speckled Sussex chicks (Figure 4) have the striped down pattern but reduced eumelanin on the backs (they may be recessive wheaten). Their ventral side is much lighter with darker gold color along the flanks. The absence of the black pigment is noticeable, and you get a clear cream and gold color without the grayish background of the normal wild-type on the ventral side of the chick. This can be clearly seen in Figure 4. I wish that I had an example of the ventral side of an *e+* down chicks. The loss of the interspersed eumelanin containing down plumules from the ventral side of the chick is clearly evident if you have a side by side comparison. Speckled Sussex are supposed to be recessive wheaten and this may remove the eumelanin from the chest and abdominal region of the chick, but Speckled Sussex obviously have pheomelanin and eumelanin intensifiers that darken the plumage to a rich dark brown in the adult, and these modifiers may be responsible for the amount of eumelanin that the chicks retain in their down. Wheaten down, even with some striping is usually wheaten pale down like you find in Salmon Faverolles. Wheaten down can have quite a bit of eumelanin expressed with the addition of eumelanin intensifiers like *Ml* (melanotic). The quail pattern is supposed to be dominant wheaten with eumelanin intensifiers (see Murray McMurray’s Quail Antwerp Bantam chick pictures).

Speckled Sussex and white Sussex are supposed to have recessive wheaten (*ey*), but in the single Speckled Sussex that I sequenced had the Cys213 amino acid polymorphism that I had found to be among *e+* Red Junglefowl accessions and in the Light Brown Leghorns. The *e+* breeds seem to have either Arg 213 or Cys213 polymorphism. It may be that recessive wheaten is a regulatory variant or due to a closely linked modifying gene. I intend to discuss this in the molecular biology discussion. In Figure 4 you can see that the Speckled Sussex chick has pheomelanin intensifiers (the adult plumage is dark brown in the hens) but it retains the wild-type striped down pattern down to the bandit eye stripe. There is less eumelanin in the dorsal and ventral down and eye stripe that might be associated with the wheaten allele.



Figure 4. Speckled Sussex (*ey*?) chick with less gray down in the chest and abdomen than wild-type. The Speckled Sussex does have pheomelanin intensifiers (very dark brown plumage), but the chick has reduced black in the striped down pattern

**Black down *E* and *ER*:**

The black down is shown in Figure 3 of the Black Minorca. The black down chick above the left facing red arrow shows the cream or white in the face and chest that is common in black down chicks. There can be extensive amounts of white or cream in the head and ventral down. Hutt (1949) notes that *E* black down is associated with shorter down. This down can be so short that it is associated with decreased hatch rate for some black breeds. The down is not always noticeably shorter. The Black Minorcas in Figure 3 had about medium length down. The shortest black down that I have observed was among Black Jersey Giants from Murray McMurray. I once got 300 chicks from McMurray hatchery and just specified Black Australorps and Black Jersey Giants. I needed them as a dominant white tester line. You could tell the breeds apart because the Australorps had white skin showing on the bottoms of their feet, while the Jersey Giants had yellow skin on the bottoms of their feet. You could have also told the chicks apart by the length of the down. The Australorps, on average, had noticeably longer down than the Jersey Giants. Both lines had the same *E* (a possible designation) allele (Thr71-Lys92). I found it to be the most common allele in black feathered breeds. The Black Australorp chicks had a lot of white down in their faces and ventral region, while the Black Jersey Giants had less nonblack down with some of the chicks having nearly totally black down. It might be that there are modifiers that darken or decrease eumelanin expression in the down and shorten or lengthen the down length.

I found what is likely the birchen allele in White Leghorns and it segregates in black feathered breeds but was the minor allele (Met71-Lys92). It may be the difference in alleles is due to linked modifying genes or may be regulatory. The Fayoumi birchen allele phenotype described by Smyth (1990) has a different sequence. It does not have the Lys92 polymorphism associated with increased eumelanin production in mammals and birds, and instead has the Gln133 polymorphism (Ellett, 2000). The allele was designated *ER* by Smyth, but it will likely get designated something like *MC1R\*RF* for Fayoumi birchen.

Smyth (1990) described the birchen allele being more susceptible to *Db* (Dark Brown). *Db* was first identified as turning black birchen down brown, and when I crossed a White Leghorn line segregating the likely *ER* allele (Met71-Lys92) to the Red Junglefowl (UCD001 from UC Davis). When the F2 segregated away from dominant white (*I*) some matings produced a dark rich brown color down instead of black down. It was about the same color as the Fayoumi down, but was not striped and marbled. The White Leghorn line was ADOL Line 0 and was not an inbred line, but it had been selected out of mixed commercial source for lack of endogenous *ALVE* retroviral sequences. Line 0 may segregate *Db* because UCD001 is the reference chicken genome sequence and does not have the *Db* allele (Gunnarsson, et al., 2011). I may be wrong and there may be some other modifying gene that causes the black down to turn brown in this cross. Smyth (1990) describes the Fayoumi as having the birchen allele and *Db*, and you can see dark brown marbled down in Figure 3.

***eb* and *ebc* down:**

The *eb* and *ebc* alleles are supposed to have distinct down patterns, but similar adult phenotypes. Both alleles have the same *E* locus coding sequence (Thr71-Lys92-Cys213-Pro215). It has the same amino acid sequence as the suspected *E* allele with the addition of Pro215. When I first obtained the sequence information it had been found that the Lys92 polymorphism created a constitutively active MC1R receptor that sent signal in the absence of ligand binding. It looked like the Pro215 might attenuate this constitutive activity that was associated with increased eumelanization in mammals, and *MC1R* gene knockouts were associated with decreased eumelanin expression, and production of pheomelanin. I got involved with a group of researchers that wanted to study the function of the MC1R receptor (Ling et al., 2003). I gave them the plasmid clones that I had of the *MC1R* gene coding sequences, and they recloned them into expression vectors that would produce intact MC1R receptors. The *eb* allele had higher constitutive activity than the black alleles, and the Fayoumi birchen allele did not have constitutive activity, but appeared to respond to alpha MSH stimulation. Things were not as simple as I had thought. I will discuss this when I discuss the molecular biology of the system.

The *eb* allele exhibits a range of down colors and patterns, likely due to genes that modify the expression of the *E* locus allele. The down can show the dorsal stripe pattern on the back of the chick, but the black eumelanin stripes may be missing on the back. What is left are the dark brown stripes on the back. The stripe pattern of the head is replaced by a brown helmet of dark down on the head. The pattern can range from a noticeable stripe pattern on the back to a uniform dark brown on both the head and back.

The *ebs* allele is depicted by Smyth, 1990 in the black and white photograph (Figure 5.2). It looks like a light (wheaten?) down with a broken wild-type striped pattern and you can see the thin eumelanin black stripes flanking the main dorsal dark brown strip. There is a broken striped pattern on the head that looks like it has the same pattern as the back.

***eWh* and *ey* Down:**

Smyth (1990) goes into detail about the differences that have been identified for the two wheaten alleles. I found the same allele (Ala143) in both New Hampshire Reds and commercial Rhode Island Reds (used for down color sexing). This allele does seem to be dominant in removing the black from Columbian restricted chick down, but is not completely dominant in the hybrid feather undercolor (the undercolor is light gray instead of cream) of New Hampshire Red crossed to Light Brown Leghorn hybrids (*eWh*/*e+*, *Co*/*co+*). The quail pattern is supposed to be dominant wheaten (*eWh*) with melanotic (*Ml*) but Davila et al., 2014 found their quail color line to have the *e+* Arg213 sequence with *Co*. I found the Ala143 wheaten allele in Buff Minorcas that were supposed to be mainly recessive wheaten (*ey*) with a mix of other alleles. Davila et al., 2014 found the same Ala143 allele in their dominant wheaten lines, but found the two wild-type allele sequences in their recessive wheaten tester line (Arg213 and Cys213). Amino acid position 213 is a conserved position in melanocortin receptor protein sequences of other animals, but the site can vary between Arg213 and Cys213 in MC1R protein sequences. Arg213Cys polymorphism seems to be allowed and retains function. It may be that the difference between alleles is due to regulatory differences or closely linked flanking genes for the two wild-type sequences to produce the recessive wheaten phenotype. Morejohn (1953) found his recessive wheaten to have clear wheaten down with no back and head striping. This type of clear wheaten down is found in Salmon Faverolles that are supposed to be dominant wheaten. Wheaten down can have stripes, but eumelanin is reduced in the chick down. A cross between Light brown Leghorns and Salmon Faverolles might be informative for the difference between recessive and dominant wheaten, but such a cross has not been published. Speckled Sussex are supposed to be recessive wheaten, but the chicks have a darker brown wild-type stripe pattern (Figure 4) with reduced black in the down. They obviously have pheomelanin and eumelanin intensifiers because the adult hen plumage is a deep rich brown with mottling, but the chicks have reduced black in both dorsal and ventral down. Wheaten seems to show a range of down types. Murray McMurray has a picture of Salmon Faverolle chicks that have the black head stripe that Smyth (1990) associated with recessive wheaten, and some chicks with almost no eumelanin on a wheaten down. The Salmon Faverolle chick that I obtained from Murray McMurray in the 1980’s had a clear wheaten down with no down stripes. I do not recall if it might have had some black down plumules on the head. Smyth, 1990 notes that recessive wheaten can show down striping. Dark Cornish are supposed to be recessive wheaten and sex-linked gold (*s+* *ey*/*ey* *Ml*/*Ml* *Pg*/*Pg*) the chicks have dark striped down. Murray McMurray has pictures of the chicks (link given in references).

Wheaten Table 1

|  |  |  |  |
| --- | --- | --- | --- |
| **Breed** | **Proposed allele** | **Polymorphism** | **Down** |
| New Hamp | *eWh* | Ala143 | Mostly clear with some striping on some chicks |
| Prod. RIR | *eWh* | Ala143 | Mostly clear with some striping on some chicks |
| Red-Barred Vasca | *eWh* | Ala143 | ? |
| Buff Prat | *eWh* | Ala143 | ? |
| Buff Minorca | *ey* | Ala143 | Clear Buff Wheaten |
| Speckled Sussex | *ey* | Cys213 | Darker brown striped down with reduced black |
| *ey* tester line | *ey* | Arg213Cys | ? |
| Salmon Faverolle | *eWh* | ? | Clear wheaten no striping |

My own data, and from and most frequent allele from Davila et al., 2014. Cys213 was the most frequent allele found in the Davila et al., 2014 *ey* tester line.

**Down differences due to sex-linked silver and gold:**

I do not think that silver and gold can be differentiated on *E* or *ER* black down chicks. The nonblack down of the facial and dorsal down does not seem to reliably differentiate silver and gold. I have only worked with segregating populations and never with specific crosses to make predictable genotypes. This down can be white or yellow, but it doesn’t seem to be related to sex-linked gold. Where the down is black there doesn’t seem to be a noticeable difference between silver and gold chicks. Smyth, 1990 notes that silver and gold is not differentiated on black down chicks.

Silver and gold can be distinguished on an *e+* striped down. The down of the gold back stripes and the facial down is subject to silver dilution. The dark brown stripes are not effectively diluted and the pheomelanin around the neck and sides of the chick do not seem to be as differentially diluted. You can look at the difference between Silver Leghorn chicks and Light Brown Leghorn chicks on the McMurray web site.

For the *eb* allele there are some crosses where you can tell silver and gold down apart, but silver does not dilute the brown down pigment very well until you add *Co*. It may be for the same reason that the dark brown back and head stripes are not diluted very well in silver breeds like Silver phoenix and Silver Leghorns. With *Co* present where the chicks are gold the down is subject to silver dilution, but *eb*, *Co* chicks have black down on their backs that reduce the area that can be typed for silver or gold down. By comparing Partridge Rock and Silver Penciled Rock chicks you can see a down difference between silver and gold with the *eb* allele, but the Partridge Rocks have pheomelanin intensifiers that darken the brown down. Silver Penciled Rocks do not have these pheomelanin intensifiers, and likely have pheomelanin diluters. I once raised a Partridge Rock line that had a uniform rich mink brown down with hardly any striping visible.

For the wheaten downs there doesn’t seem to be a difference in dilution by sex-linked silver. This may be due to the lack of pheomelanin pigment or what pigment there is may be the salmon pigment that is not subject to silver dilution. If you add *Co* the silver and gold wheaten down can be easily differentiated. Smyth, 1990 suggested that this was due to *Co* making the salmon pigment susceptible to silver dilution, but it may be that *Co* allows the normal gold pigment to replace the salmon pigment usually produced by the wheaten allele and distributes the pheomelanin gold pigment throughout the head and back of the chick as it does for the adult body feathers. It may be that the salmon pigment is also produced in the down of *eb* chicks making silver and gold differentiation difficult. I will try to get back to this notion when discussing the molecular biology of the *E* locus.

**Concluding Remarks:**

There are alleles associated with basic feather color differences on an otherwise wild-type genetic background, but we have not yet done the genetic evaluations that need to be done to identify these alleles and conclusively relate their gene sequences and flanking regulatory sequences to *E* locus phenotypes. Three black alleles have been, so far, identified by their different coding sequences, but all segregate in black and birchen breeds when different alleles were identified as being *E* or *ER*. The old literature notes that there may be a dominant wheaten allele and a recessive wheaten allele that produce the same adult phenotypes, but Carefoot (1981) found evidence that one allele could be affected by modifying genes to produce the dominance effects. Two coding sequence haplotypes have been associated with the wheaten haplotype with the one haplotype associated with recessive wheaten having the same coding sequence as the *e+* allele found in a Red Junglefowl, and the Murray McMurray Light Brown Leghorns. Another sequence (Ala143) is associated with the wheaten allele that shows some dominance in crosses such as the commercial layer silver-gold sexlinked down color sexing cross. The same (Ala143) coding sequence was found in Buff Minorca that are claimed to be recessive wheaten. It looks like either regulatory variation (only the coding sequences have been evaluated at this time) or modifying genes may be responsible for some of the wheaten phenotypes.

What needs to be done in order to clear the matter up and determine the exact effects of the various alleles is to produce congenic lines that differ only in their *E* locus alleles. There currently are no highly inbred lines that do not have dominant white (all the surviving inbred lines are derived from White Leghorns). Lines such as the New Hampshire Red and Australorp lines that were brought to over 80% inbred have been lost due to line reductions at research institutions. I’ve lost track of Briles’ mottled Ancona line, but it isn’t the genetic background to evaluate the *E* locus alleles on. UCD001 is a wild-type genetic background (Red Junglefowl the original reference chicken genome) and is inbred to a significant degree due to the low population size that had been maintained. UCD001 is also a decent laying bird (rescued once by crossing in White Leghorns). UCD001 has the same *e+* (Cys213) coding sequence that was also found in the recessive wheaten tester line by Davila et al., 2014. Introgressing the Davila et al., *ey* gene region into UCD001 should tell us if recessive wheaten is due to regulatory variation or flanking modifying genes. Ideally, all the alleles should be introgressed into UCD001, so that flanking regulatory sequence variation can be evaluated, but current gene editing technology can alter the coding sequences to what has been found in order to determine the effect of these coding sequences on phenotype. Crosses between the congenic lines would determine dominance between alleles, and provide phenotypes of the heterozygotes. The existing standard breed color varieties indicate that *E* locus alleles producing various phenotypes exist, but just looking at the color varieties shows segregating polymorphism and uncertainty of the genetic cause for the phenotypic differences.

**References**:

Andersson, L., Bed’hom, B., Chuong, C.-M., Inaba, M., Okimoto, R., and Tixier-Boichard, M.. 2020. Chapter 3, The genetic basis for pigmentation phenotypes. In. Advances in poultry genetics and genomics. Edited by Aggrey, S.E., Zhou, H. Tixier-Boichard, M., and Rhoads, D.D.. Burleigh Dodds publishing.

Campo, J.L. and Avarez, C.. 1993. Genetics of the Birchen and Blue Plumage Patterns in Leonesa Chickens. Poultry Science. 72: 1218-1223.

<https://www.sciencedirect.com/science/article/pii/S0032579119451183?via%3Dihub>

Carefoot, W.C.. 1981. Notes on the “wheaten” plumage phenotype in domestic fowl. British Poultry Science. 22: 499-502.

Article paywalled : <https://www.tandfonline.com/doi/abs/10.1080/00071688108447916>

Carefoot, W.C.. 2002. Hen-feathering mutation HF\*H may act as a eumelanizing factor and modify the expression of autosomal barring. British Poultry Science. 43: 391-394.

Article paywalled: <https://www.tandfonline.com/doi/abs/10.1080/00071660120103666>

D’Alba, L. and Shawkey, M.D.. 2019. Melanosomes: Biogenesis, properties, and evolution of an ancient organelle. Physiol. Rev. 99: 1-19

<https://pubmed.ncbi.nlm.nih.gov/30255724/>

Davila, S.G., Gil, M.G., Resino-Talavan, P., and Campo, J.L.. 2014. Association between polymorphism in the melanocortin 1 receptor gene and E locus plumage color phenotypes. Poultry Science. 93:1089-1096.

<https://www.sciencedirect.com/science/article/pii/S0032579119361024?via%3Dihub>

Ellett, A.. 2000. Melanocortin-1-Receptor (MCR-1) Gene Polymorphisms Associated with the Chicken E Locus Alleles. Inquiry: The University of Arkansas Undergraduate Research Journal. 1: 37-41.

<https://scholarworks.uark.edu/cgi/viewcontent.cgi?article=1270&context=inquiry>

Galvan, I. and Solano, F.. 2016. Bird Integumentary Melanins: Biosynthesis, forms, function and Evolution. Int. J. Mol. Sci.. 17: 520.

<https://www.mdpi.com/1422-0067/17/4/520>

Gunnarsson, U., Kerje, S., Bed’hom, B., Sahlquvist, A.-S., Ekwall, O., Tixier-Boichard, M., Kampe, O., and Andersson, L.. 2011. The Dark brown plumage color in chickens is caused by an 8.3 kb deletion upstream of the *SOX10*. Pigment Cell Melanoma Res. 24: 268-274.

<https://onlinelibrary.wiley.com/doi/10.1111/j.1755-148X.2011.00825.x>

Hamilton, H.. 1940. A study of the physiological properties of melanophores with special reference to their role in feather coloration. Anat. Rec.. 78: 525-548.

Article paywalled: <https://onlinelibrary.wiley.com/doi/10.1002/ar.1090780407>

Hauschka, T.S., Jacobs, B.B., Holdridge, B.A.. 1968. Recessive yellow and its interaction with belted in the mouse. J. Heredity. 59 : 339-341.

<https://academic.oup.com/jhered/article-abstract/59/6/339/826585?redirectedFrom=fulltext>

Hutt, F.B.. 1949. Genetics of the Fowl. McGraw-Hill Book Company. New York

<https://digital.library.cornell.edu/catalog/chla2837819>

Kerje, S., Lind, J., Schutz, K., Jensen, P., and Andersson, L.. 2003. Melanocortin 1-receptor (MC1R) mutations are associated with plumage colour in chicken. Animal Genetics. 34: 241-248.

Article paywalled: <https://pubmed.ncbi.nlm.nih.gov/12873211/>

Kerje, S., Sharma, P., Gunnarsson, U., Kim, H., Bagchi, S., Fredriksson, R., Schutz, K., Jensen, P., Heijne, G.V., Okimoto, R., and Andersson, L.. 2004. The Dominant white, Dun and Smoky color Variants in Chicken are Associated with Insertion/Deletion Polymorphism in the PMEL17 gene. Genetics. 168: 1507-1518.

<https://academic.oup.com/genetics/article/168/3/1507/6059563>

Ling, M.K., Lagerstrom, M.C., Fredriksson, R., Okimoto, R., Mundy, N.I., Takeuchi, S., and Schioth, H.B.. 2003. Association of feather color with constitutively active melanocortin 1 receptors in chicken. Eur. J. Biochem. 270: 1441-1449.

<https://pubmed.ncbi.nlm.nih.gov/12653999/>

Morejohn, G.V.. 1953. A gene for yellowish-white down in the Red Junglefowl. J. Heredity. 44: 46-52.

Article paywalled:

<https://academic.oup.com/jhered/article-abstract/44/2/47/820052?redirectedFrom=PDF>

You get to read the first page that describes the results, but you do not get to see the picture of the chicks.

Raposo, G. and Marks, M.S.. 2007. Melanosomes – dark organelles enlighten endosomal membrane transport. Nat. Rev. Mol. Cell Biol. 8: 786-797

<https://pubmed.ncbi.nlm.nih.gov/17878918/>

Smyth Jr., J.R.. 1990. Genetics of plumage, skin, and pigmentation in chickens. In Poultry Breeding and Genetics. Ed. Crawford, R.D.. Elsevier Science. Pages 109-167.

W. Clive Carefoot: Publications listed in PubMed. For some reason PubMed doesn’t come up with all Carefoot’s publications, but this is a place to start.

<https://pubmed.ncbi.nlm.nih.gov/?term=Carefoot+WC&cauthor_id=1393683>

American Standard of Perfection. Published by the American Poultry Association. Various copyright dates.

<https://amerpoultryassn.com/>

Murray McMurray web site with chick pictures. I am a big fan of McMurray hatchery. My wife still reminisces about when we’d get our yearly Spring shipment of chicks, with the surprise “free” chick from 40, years ago. Go to the web page and then click on the “details” tab above the breed of interest. You can search breed on the “All Baby Chicks” page. Chick picture tabs are on the lower left of the window that opens.

<https://www.mcmurrayhatchery.com/chicks.html>