THE INHERITANCE OF SHANK COLOR IN CHICKENS

C. W. KNOX
Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Received March 15, 1935

Several publications in recent years have given conflicting reports upon the inheritance of shank color. Yellow shank color has been reported to be inherited in a simple Mendelian fashion, the sex-linked gene for yellow shank color (Y) being dominant to its allele (y), the gene for blue shank color. In contrast to this, the gene for yellow shank color has been reported to be autosomal, the recessive gene (w) designating yellow shanks and the dominant gene (W) representing white shanks. Obviously, these two assumptions are in disagreement. Still another hypothesis is that yellow shanks carry a dominant sex-linked inhibiting gene for dark shank color (D), while dark shanks lack this inhibitor (d). In other words, the following situation prevails:

Dominant gene	Allele	Chromosome
W = white shanks	w = yellow shanks	autosomal
Y = yellow shanks	y = blue shanks	sex-linked
D = inhibitor of dark shank color	d = dark shanks non-inhibitor	sex-linked

WHITE AND YELLOW SKIN COLOR

PUNNETT (1923), in writing of white and yellow skin color, has stated that yellow and white appear to behave as a simple pair of characters, the latter being dominant to the former. A few years later, DUNN (1925) reported a similar situation in regard to the inheritance of yellow and white shank color. The factor for white shanks in DUNN'S (1925) report is assigned the symbol W, and he states that this gene is practically dominant to the gene for yellow designated as w. Two years later DUNN and JULL (1927) and LAMBERT and KNOX (1927) verified this theory. In addition to this, LAMBERT and KNOX (1927) have stated that this one autosomal gene affected the color of the skin, beak and shanks.

In contrast to this, WARREN (1928) has stated that the gene which determines shank color is very definite in its expression but is probably masked by certain plumage colors. The contrasted characters were yellow and blue shanks, which he designated as Y for yellow and y for blue shanks, and also stated that these factors were sex-linked.

From previous investigations it is apparently agreed that there are two kinds of pigments. HANAU (1881), JEFFRIES (1883), and GADOW (1891) state that the various colors of the shank are due to the presence or absence

of two kinds of pigment, orange-yellow and brownish-black. They agree that the orange-yellow is a lipochrome pigment which, in concentrated form, gives an orange-yellow color and in a more diluted form a light-yellow, even to an almost whitish color. The brownish-black pigment is in reality a melanic pigment and is responsible for the black, blue, and green shades.

The histological work of BARROWS (1914) verified this and showed, in addition, that the colors are varied according to the amount and kind of pigment and whether or not the pigment is deposited in the dermis or epidermis of the skin or both. It is evident from these investigations, therefore, that two kinds of pigments are concerned in shank color.

It is universally known that all breeds and varieties of chickens have yellow or white skin color, that is, the presence or absence of lipochrome pigment. Sometimes the melanic pigment in the shanks is so dark and so completely covers the shanks that it is difficult to ascertain the presence or absence of lipochrome pigment. In such cases, this can be done readily by observing the skin color of the bottom of the feet. For example, Jersey Black Giants have very black shanks but the bottom of the feet have some lipochrome pigment appearing through the black, whereas the Black Langshan has just as black shanks as the Jersey Black Giant but the bottoms of the feet have some white appearing through the black, showing the lack of lipochrome pigment.

In a study of the inheritance of lipochrome color, a backcross was made of an F_1 male (Buff Orpington×Barred Plymouth Rock) with Rhode Island Red females. The genotypes involved were ♂ *Ww*× ♀ ♀ *ww*. Half of the progeny would be expected to have a lack of lipochrome pigment, as indicated by the white shanks, and the other half would be expected to have yellow shanks indicating the presence of lipochrome pigment. The results obtained were 516 progeny without lipochrome pigment in the shanks and 448 with lipochrome. This was a deviation of 34 from the theoretical number expected, 482. Most of this deviation was due to the fact that the chicks were raised in batteries, which would tend to bleach the yellow shanks so that in some cases these would be classed as lacking lipochrome. It was further complicated by the range in shank color in both groups. The range in color was from an ebony black to a clear white or yellow.

It is evident from the previous investigations mentioned and this one that yellow pigment in the shanks is inherited in a simple monohybrid manner and is autosomal, being recessive to its allele, *WW*, the lack of lipochrome pigment in the shanks. Hence all breeds and varieties either have yellow pigment, *ww* (presence of lipochrome), or lack yellow pigment, *WW* (absence of lipochrome), in the shanks.

The only exception to this was the result reported by WARREN (1928), who stated that yellow (*YY*) was dominant to blue (*yy*) shanks. He probably arrived at this result due to the complication of blue shank color. If he had considered yellow and blue shank color in the same manner as DUNN and JULL (1927) he might have been in accord with the statement made in the preceding paragraph. Therefore, it is the writer's belief that the work of WARREN (1928) would more correctly fit in a study of the inheritance of dark rather than yellow shank color.

DARK AND LIGHT SHANK COLOR

Several conflicting theories in respect to the mode of inheritance of dark shank color have been advanced. However, as previously stated, dark shanks are caused by melanic pigment. The presence or absence of various amounts of melanic pigment and whether or not it was deposited in the dermis or epidermis or both, as well as the presence or absence of lipochrome pigment tend to make the whole problem complicated. The different combinations of these pigments or their absence are responsible for the various shades of shank color from white or yellow, blue or green, to the deepest color, the ebony black.

The best and most probable explanation of shank color is reported by DUNN and JULL (1927). In crosses involving the White Silkie, they reported that the genes involved in shank color were essentially as follows:

	MALES	FEMALES
White, pinkish white, pearl and light blue shanks, (may show spots of dermal pigment especially in the females and *Dd* males)	*WWDD* *WwDD* *WWDd* *WwDd*	*WWD–* *WwD–*
Orange yellow, light yellow, creamy shade (may show spots of dermal pigment in females and in *Dd* males)	*wwDD* *wwDd*	*wwD–*
Black, slate, blue and gray shanks	*WWdd* *Wwdd*	*WWd–* *Wwd–*
Black, dark green, light green or willow shanks	*wwdd*	*wwd–*

This classification is based upon two sets of factors, one autosomal for white and yellow color, and the other sex-linked and controlling dark shank color. Whether the genotypes given above cover all cases remains to be determined. PUNNETT (1923) suggested that there are indications that the presence of dermal pigment in certain instances is dominant to its absence. JULL (1932) states that "since it has been suggested that highly developed epidermal pigment is associated with black or dark brown plumage, it is possible that the gene *E*, for the extension of melanic pig-

ment throughout the plumage, may also affect the extension of melanic pigment to the shanks."

It has been shown that there are several ways in which the melanic pigment might be inherited. First, as stated by WARREN (1928) yellow is sex-linked and dominant to blue; second, the suggestion of PUNNETT (1923) that the presence of dermal pigment is dominant to its absence; third, as JULL (1932) suggests, the gene *E* for the extension of black in the plumage may be of a general nature and may affect the shank color; fourth, as classified by DUNN and JULL (1927).

RESULTS

With these assumptions in mind, data were compiled at the U. S. ANIMAL HUSBANDRY EXPERIMENT STATION, Beltsville, Maryland, in an attempt to clarify the inheritance of dark shank color. The problem appeared to be one involving the presence or absence of melanic pigment and the birds were so classified, omitting the yellow and white classification and making no attempt to differentiate dermal from epidermal melanic pigment. In this way complications were avoided that arose from the blue, green, and black shank colors.

THE EFFECT OF THE GENES *EE* AND *BB* UPON MELANIC SHANK COLOR

It has been observed in publications by BATESON and PUNNETT (1911), DUNN (1925), DUNN and JULL (1927), and WARREN (1928), that in crosses where the results secured were interpreted on a sex-linked basis such crosses involved a sex-linked plumage factor, usually that for barring. Therefore, in this experiment the first cross made was between Jersey Black Giants, which had black shanks, and Rhode Island Reds, which lacked black pigment in the shanks. Both breeds had the genes *ww* for yellow shank (skin) color so that this factor was eliminated in this cross. The plumage genes involved were mainly *EE* for black plumage and *ee* for the non-extension of black plumage, plumage genes that are not sex-linked.

The plumage of all of the 118 F_1 (*Ee*) progeny from reciprocal matings was black or predominantly black, showing no sex-linkage, and all showed varying amounts of melanic pigment in the shanks. There was more red in the plumage color of the F_1 males than in the plumage of the F_1 females, and about 40 percent of the plumage of 3 of the F_1 males was red; one of these males had yellow shanks with a tinge of melanic pigment, the other two males had shanks that were a dusky yellow. One of the F_1 males with black plumage had dark green shanks. All of the remainder of the F_1 progeny had predominantly black plumage and dark shanks. Thus it would appear that the melanic pigment in the shanks of this cross was dominant to yellow shanks, as suggested by PUNNETT (1923), and in

contrast to the results reported by WARREN (1928). This also agrees with the suggestion of JULL (1932) that the plumage gene *E* has some effect on the melanic color in the shanks. As a matter of fact, this cross shows that the gene *E* for the extension of melanic pigment is of a general nature and effects the deposition of melanic pigment in the shanks in about the same manner that it does the plumage color.

As further proof of this statement, three Jersey Black Giant females mated to a Rhode Island Red male proved to be heterozygous blacks, *Ee*, and produced 18 predominantly black offspring, (*Ee*), and 10 red (*ee*) offspring. Of the 18 black offspring, 17 had black shanks and one male with a considerable amount of red plumage had dusky yellow shanks. All of the red progeny, males and females, had yellow shanks devoid of melanic pigment. If there were separate genes for the deposition of melanic shank color in this cross, the red progeny, *ee*, would have had black shanks, the same as their black sisters. Of course, this does not obviate the possibility of a close linkage between black plumage color and dark shank color. Such a linkage would fix the dark shank color on the autosome carrying black rather than on the sex chromosome, as previously reported. However, it seems more likely and logical that the genes, *EE*, have a general effect upon the deposition of melanin, which would include the plumage, shanks, beak, and possibly dark eye color.

From a backcross mating of a Rhode Island Red male (*ee*) with F_1 (*Ee*) females (Jersey Black Giants×Rhode Island Reds), 260 red, 11 columbian, and 225 black progeny were produced. The 260 with red plumage color had yellow shanks, lacking any sign of melanic pigment in the shanks. The same was true of the 11 columbian-patterned progeny. There was considerable variation in the plumage and shank color of the 225 predominantly black progeny, most of which had dark shank color. It would appear, therefore, that while the gene *E* effects the deposition of the melanic pigment in the plumage and the shanks, there is considerable variation in the shank and plumage color in the heterozygous black (*Ee*) progeny. This variation is due, no doubt, to the effect of modifying factors and possibly different hormone activity, especially as between sexes. It was noted again that some of the F_1 males usually carried less black pigment in the plumage and less melanic pigment in the shanks than the F_1 females.

A similar condition was noted in 153 F_2 black and 49 F_2 red progeny from a Jersey Black Giant×Rhode Island Red parental cross. All of the birds with red plumage color had yellow shanks, whereas the progeny with black plumage had dark shank color. The amount of black pigment in the shank color of these birds closely approximated the amount of black observed in the plumage color.

In another backcross of Rhode Island Red females with an F_1 male (Buff Orpington male×Barred Plymouth Rock females), it was found that the barring factor affected not only the black plumage genes, *Ee*, restricting the black to alternate bars of black and white, but also practically eliminated the evidence of melanic pigment in the shanks. All of the progeny from this cross were classified with respect to plumage color as black and white barred, gold and white barred, black, gold, and columbian. Each group had the shank color classified as dark, medium and clear. Only black and almost black shanks were classified as dark, whereas the medium dark colored shanks included blue, green, dusky yellow, dusky white, light green and light blue, and the clear shanks included only the white and yellow shanks that lacked melanic pigment. The observed results are given in table 1.

TABLE 1

Plumage and shank color of backcross offspring, F_1 male (Buff Orpington male×Barred Plymouth Rock females)×Rhode Island Red females.

PLUMAGE COLOR OF OFFSPRING	SHANK COLOR								
	MALES			FEMALES			TOTALS		
	DARK	MEDIUM	CLEAR	DARK	MEDIUM	CLEAR	DARK	MEDIUM	CLEAR
Black and white barred *S-B-Ee* and *s-B-Ee*	2	9	105	1	15	69	3	24	174
Gold and white barred *s-B-ee*	0	0	71	0	0	49	0	0	120
Black *S-b-Ee* and *s-b-Ee*	44	29	23	48	28	15	92	57	38
Gold *s-b-ee*	0	3	95	0	4	59	0	7	161
Columbian (Silver) *S-B-ee* and *S-b-ee*	0	0	134	0	0	88	0	0	222

The data in table 1 show that only 7 of the 510 *ee* progeny from this backcross had medium dark shanks, the remaining 503 showed no melanic pigment. The 7 *ee* progeny were golds and showed a considerable amount of black, which might account for the medium dark shanks. However, the plumage of these birds were predominantly red. The data show that the 120 *ssBbee* and *s-B-ee* progeny (*B*– barring; *S*– silver; *b*– non-barred; *s*– non-silver; *E*– extension of black plumage; *e*– non-extension) had no melanic pigment deposited in the shanks. However, out of 210 black and white barred progeny 3 were classified with dark shanks and 24 with medium dark shanks. The 201 progeny were obviously of the genotype *BbSsEe*, *S-B-Ee*, *ssBbEe*, and *s-B-Ee*. The 187 predominantly black progeny, on the other hand, were mostly dark-shanked birds, 149 having dark or medium dark shanks. Only 38 of the black progeny had no melanin present in the shanks. These 38 individuals may have non-melanic shank color because they are heterozygous blacks and approximately half of them

would be heterozygous for such modifying factors as silver, *S-* or *Ss*, and the other half golds, *s-* or *ss*. These data demonstrate that the barring factor, *B*, affects the genes *EE* for the extension of black in the plumage and also has the effect of restricting the deposition of melanic pigment in the shanks.

The data in table 2 give the results obtained from an F_1 male (Buff Orpington male×Barred Plymouth Rock females) crossed with White Plymouth Rock females, and these data verify the statement made in the preceding paragraph. Of the 100 individuals from this mating 22 were non-barred, black birds (*Ee*) that had dark or medium dark shanks, whereas the 47 non-barred red and columbian progeny (*ee*) had clear white or yellow shanks. The 31 black and white barred group had 8 with medium dark shanks and 23 with clear white or yellow shanks. These results again show the effect of the gene *E* upon shank color and the restricting effect of the barring gene *B*.

TABLE 2

Plumage and shank color of backcross offspring, F_1 male (Buff Orpington male×Barred Plymouth Rock females)×White Plymouth Rock females.

PLUMAGE COLOR OF OFFSPRING	SHANK COLOR								
	MALES			FEMALES			TOTALS		
	DARK	MEDIUM	CLEAR	DARK	MEDIUM	CLEAR	DARK	MEDIUM	CLEAR
Black and White barred	0	4	14	0	4	9	0	8	23
Black	1	2	0	9	10	0	10	12	0
Gold	0	0	11	0	0	8	0	0	19
Columbian (Silver)	0	0	17	0	0	11	0	0	28

In another cross, Rhode Island Red males with Barred Plymouth Rock females, the F_1 male progeny were barred, all having yellow shanks, and the F_1 females were predominantly black with dark shanks. If the barring gene *B* acted as a restrictor of melanin in the plumage and in the shanks, as previously mentioned crosses show, this apparent sex-linkage of dark shank color would be expected. The cause, however, could not be attributed to sex-linkage of a dark shank color gene without considering the effect of the sex-linked barring gene, which has a restricting effect on the black color in the shanks as well as the black plumage color. Even in this cross, however, the data on 145 black female progeny show that the percentage of black plumage and the percentage of melanic pigment in the shanks are significantly correlated, being +0.72. It was also noted that the standard deviation from the mean of the plumage color was 22.1 percent and the standard deviation for shank color was 22.2 percent. In other words, there was as much variation in the plumage color of these F_1

females which were of the genotype *b-EeCC* as there was in the shank color. It has been found that in most plumage color crosses, the F_1 progeny vary considerably in plumage color and it is probable that the variation of plumage and shank color found in the F_1 black females merely expresses an expected normal variation.

It is evident from the results mentioned, that WARREN (1928), who used crosses involving barring, *BB*, (Barred Plymouth Rocks and White Leghorns) and black, *EE*, (Jersey Black Giants) in his study of shank color, did not have a critical cross in respect to shank color, that would separate the effect of the sex-linked barring genes *Bb* upon black color from a sex-linked gene for shank color. In addition to this, the number of progeny obtained was exceedingly small for a study involving linkage and crossing over percentages. Because of the small number of progeny involved, the percentage of crossing over found by WARREN between the rate of feathering and shank color cannot be differentiated statistically from the crossing over percentage between the rate of feathering and barring. This would be expected if the influence of barring on the general genes for melanic pigment, *EE*, is considered.

This would also apply to the work of SEREBROVSKY and WASSINA (1926), who stated that they found two questionable crossovers among 69 individuals.

HERTWIG (1933) also used crosses which were not critical enough for adequate analysis of shank color. She made the statement that the factor for slow feathering and inhibitor for negroid plumage color and dark mesodermal shank color in the crosses studied were absolutely coupled.

DUNN and JULL (1927) used a cross of White Silkie × White Leghorns in a study of the inheritance of dark shank color. This cross again is not critical enough as it does not differentiate between the effects of the barring gene brought in by the Leghorns (*BBEEII*, *B-EEII*) and the effect that might be due to a pair of sex-linked factors for melanic shank color.

In a more recent publication HAGEDOORN (1930) cites a cross of a White Leghorn male with two Barnevelder females. This would constitute another non-critical cross for shank color, since it is a cross of a *BBEEII* male with a non-barred, non-black female *b-eeii*. HAGEDOORN states that in crosses of Barnevelder male × dark-shanked females, it was shown that the yellow of the Barnevelder was not dominant, as in the case of the Leghorn, but recessive. A more fitting explanation, however, might well be that the yellow shank of the Barnevelder is recessive because it is non-barred (*bb*, *b-*) non-extended black (*ee*) bird, whereas the yellow shanks of the Leghorn are due to the barring gene *B*, which is dominant, thus making it appear that the yellow shanks of the Leghorn are dominant.

HAGEDOORN also states that all of the F_1 progeny obtained from the White Leghorn male × the Barnevelder females had yellow shanks, which was expected. In the F_2 generation, however, some of the females had dark shanks, but all of the males had yellow shanks. He explains this by assuming that the Barnevelder has yellow legs because it lacks a factor termed *BB*, and also lacks the sex-linked factor *AA*, the genetic formula for the color of the Barnevelder's shanks being given as *a-bb*, whereas the White Leghorn male would be *AABB*. It will be noted that this assumption is equivalent to stating that the Barnevelder females are non-barred, non-extended blacks, *b-ee*, whereas the White Leghorn males would be barred, extended blacks, *BBEE*. Both of these latter symbols are preferable to use in the light of the present data. Both formulae, of course, are identical in their action and result.

MACARTHUR (1933) stated, "That on the basis of published data, a great number of uncertainties and inconsistencies in the proposed arrangement of the genes on the sex chromosome map demonstrated the need for correction by further investigations." Such a situation is readily understood when it is realized that MACARTHUR and others in their investigations on crossing over used material involving certain characters the nature of whose inheritance has not been determined by parental crosses constituting a critical test. For instance, such genes as *Dd* (sex-linked dark shank color), *LiLi* (sex-linked down color), and *BrBr* (iris color) have not been satisfactorily explained as to their mode of inheritance so that they might be used in crossover investigations with impunity. To make matters more complicated some investigators have attempted crossover studies with too few progeny.

MACARTHUR (1933) also states that "the breed correlation between black feathers and dark shanks and dark eyes is also broken in many instances." This does not agree with the results obtained in this investigation. The difference is no doubt due to two things; first, the barring of the Silver Campine is not sex-linked as in the barring reported for the Barred Plymouth Rock; second, that the leaden blue shanks of the Silver Campine might indicate a separate gene for melanic shank color which the Barred Plymouth Rock does not have. If there is a separate gene for shank color, MACARTHUR'S statement might hold true for the material that he used but would not apply to the results as reported in this investigation where crosses were used that did not involve separate genes for melanic shank color.

It is evident from the data presented that the black plumage genes, *EE*, are of a general nature and that they cause the deposition of melanic pigment in the shanks as well as in the plumage. It has been shown also that there is as much variation in the effect of the black genes *EE* upon plumage

color as upon the shank color, which is indicated by the standard deviation. It has been demonstrated also that the barring genes *BB*, which are sex-linked, have a restricting effect upon the melanic pigment in the shanks as well as in the plumage. The restricting effect that these genes have upon shank color, however, vary to some extent although this variation is probably no greater than the variation in plumage color found in the F_1 heterozygous black females of the cross of Rhode Island Red males × Barred Plymouth Rock females.

THE EFFECT OF THE PLUMAGE COLOR INHIBITOR GENES *II* UPON MELANIC SHANK COLOR

In reciprocal crosses of Jersey Black Giants with White Leghorns, GODFREY and QUINN (unpublished data, BELTSVILLE RESEARCH CENTER) observed that all of the 134 F_1 progeny from the White Leghorn male × Jersey Black Giant females had yellow shanks free of any melanic pigment. The F_1 progeny from the reciprocal cross, however, consisted of females with slate-colored shanks and males all of which had yellow shanks.

In another cross, using a Black Minorca male × White Leghorn females, it was found that the 64 F_1 males had yellow shanks and of the 51 F_1 females 49 had blue shanks and 2 had yellow shanks. The 2 pullets with yellow shanks are probably the result of some error or mixing of the stock because even if these females lacked melanic pigment their shank color should be white and not yellow, due to the dominance of the *WW* genes for white skin color brought in the cross by the Black Minorca parental stock.

The results from these two crosses involving black plumage birds with dark shanks mated with White Leghorns with yellow shanks can be accounted for by the effect of the barring gene and the inhibiting genes, *II*, of plumage color. In the cross of the White Leghorn male, *BBEEII*, × Jersey Black Giant females, *b-EEii*, the resulting progeny would be of the formulae *BbEEIi* males and *B-EEIi* females. In this case all of the progeny would be expected to have yellow shanks because the barring factor would restrict the melanic pigment in the shank and plumage of both sexes. In the reciprocal cross and in the corresponding cross of the Black Minorca male × White Leghorn females the F_1 male progeny would be *BbEEIi* and should have yellow shank color, which proved to be the case. The female progeny, however, were of the genotype *b-EEIi* and although they were white in plumage color their shanks appeared as a dilute black (slaty or blue colored). This would be expected on the assumption that the melanic pigment gene *E* was present and the restrictor of melanin, *B*, absent in these F_1 females. As these F_1 females had a more dilute color in their shanks than the parental black breeds and the only difference between the genotype of black females with dark shanks, *b-EEii*, and the genotype of the

F_1 females *b-EEIi* is the presence of the inhibitor of plumage color, it is assumed that the difference (dilution of black in the shanks) is due to the inhibitor. Hence, in addition to the effect of barring on melanin in the plumage and the shanks, there appear to be other genes that affect the melanin in the plumage and the shanks, in this case a dilution effect of the melanic shank color by the plumage color inhibitor genes, *II*.

THE EFFECT OF THE CHROMOGEN GENES *cc* UPON DARK SHANK COLOR

There is also some evidence that the genes for lack of chromogen, *cc*, affect the melanic pigment in the shanks in a similar manner to that of the inhibiting genes, *II*. It is known that the White Langshan and the White Minorca have blue shanks. It is also known that these two breeds are reputed to be derived from the Black Langshan and the Black Minorca, respectively (PLATT 1925). The genotypes involved and the shank color in each breed is suggested to be as follows:

Black Langshan, *bbEEiiCC*, non-barred, black, non-inhibitor, and chromogen, black shanks.

Black Minorca, *bbEEiiCC*, same as above, black shanks.

White Langshan, *bbEEiicc*, same as above but lack chromogen, blue shanks.

White Minorca, *bbEEiicc*, same as above, blue shanks.

Thus it appears that the lack of chromogen in certain breeds has a dilution effect upon shank color, which is blue in the case of the *EEcc* birds and black in the *EECC* birds.

Previous investigations have shown that the genes, *WW*, are of a general nature and have the effect of producing white skin, beak and shank color and that the alleles, *ww*, produce yellow color. Crosses have been made and the results reported in this paper which show that the genes *EE* are of a general nature and affect not only the plumage color but also the deposition of melanic pigment in the shanks. In all probability they also affect the beak and eye color. Other crosses have been made which show the modifying effect of the barring genes *BB*, the inhibiting genes, *II*, and the genes, *cc*, for the lack of chromogen upon the deposition of melanic pigment in the shanks. There are probably other modifying genes for black, such as the presence of varying amounts of silver or red, and of hormones, especially sex hormones.

BATESON and PUNNETT (1911) crossed the White Silky × Brown Leghorns, the results indicating sex-linkage of shank color. Because the Brown Leghorns are a sex-linked, non-silver and a non-extended black, which the present data show does not influence melanic pigment in the shanks, it is possible that there is a pair of sex-linked genes for shank color. However, although the Brown Leghorn is reported to be an *ee* bird, it carries a con-

siderable amount of dark color. In the female, this color takes on the nature of a stippling effect of brown with a darker brown, and the male has solid black plumage in the breast and body and various amounts of black in the wings, tail, hackle, and other parts of the body. This extra amount of darker pigmented plumage in an *ee* bird may or may not have some effect aside from the sex-linked melanic shank color upon the presence or absence of melanin in the shanks. Hence, even such a cross as this one may not be critical enough to determine whether or not there is a sex-linked factor for dark shank color. However, until such observations are shown not to be well-founded, the fact that there is strong evidence, as reported by these two workers, for the existence of a sex-linked factor for dark shank color cannot be overlooked.

From all of the data gathered from various sources, including the data in this paper, the writer submits herewith nomenclature for a set of genes for shank color, including a sex-linked shank color gene. The various combinations of genes given in this list is an attempt to explain the inheritance of shank color with various combinations of the genes discussed in this paper. This list is by no means complete since it considers only the simpler effects upon shank color and omits all of the heterozygotes with their attending complications.

KEY TO GENES USED

Genes and Their Alleles

WW non-lipochrome (white color)	*ww* lipochrome (yellow color).
BB sex-linked barring plumage pattern restricts the black plumage to bars and practically a dominant restrictor of melanic pigment in the shanks.	*bb* non-barred plumage pattern, no restriction of melanic pigment in the shanks.
[1]*DD* inhibitor of melanic (dark) shank color. Sex-linked.	*dd* sex-linked gene for melanic pigment in the shanks.
EE extension of black plumage color, and of melanic pigment in the shanks.	*ee* non-extension of black plumage and shank color.
CC chromogen for plumage color non-restrictor of melanic pigment in the shanks.	*cc* lacks chromogen and has a diluting effect on the melanic pigment in the shanks.
II inhibitor of plumage color, dilutes the melanic pigment in the shanks.	*ii* non-inhibitor of plumage color and non-inhibitor of melanic pigment in the shanks.

[1] At present not critically differentiated from the barring gene, *BB*.

The Effect of Varying Combinations of the Above Genes upon Shank Color[2]

Males	Females	
	1. Barring—Black Plumage Series	
WWDDBBEE	*WWD-B-EE*	white shanks (neither lipochrome nor melanic pigment present).
wwDDBBEE	*wwD-B-EE*	yellow shanks (lipochrome, melanic pigment, *EE*, and restrictor, *BB*, all present).
[3]*WWddBBEE*	*WWd-B-EE*	possibly blue shanks (no lipochrome present but melanic pigment, *EE* and *dd*, and restrictor, *BB*, present).
[3]*wwddBBEE*	*wwd-B-EE*	possibly dark or green shanks (lipochrome, melanic pigment, *EE* and *dd*, and restrictor, *BB*, present).
	2. Barring—Non-black Plumage Series	
WWDDBBee	*WWD-B-ee*	white shanks (neither lipochrome nor melanic pigment present).
wwDDBBee	*wwD-B-ee*	yellow shanks (lipochrome pigment present but no melanic pigment present).
[3]*WWddBBee*	*WWd-B-ee*	possibly blue or light blue shanks (no lipochrome but melanic pigment, *dd*, and restrictor, *BB*, present).
[3]*wwddBBee*	*wwd-B-ee*	possibly green or light green shanks (lipochrome, melanic pigment, *dd*, and restrictor, *BB*, all present).
	3. Non-barred—Black Plumage Series	
WWDDbbEE	*WWD-b-EE*	dark shanks (neither lipochrome nor restrictor, *BB*, present but melanic pigment, *EE*, present).
wwDDbbEE	*wwD-b-EE*	dark shanks (lipochrome and melanic pigment, *EE*, present but restrictor, *BB*, absent).
[3]*WWddbbEE*	*WWd-b-EE*	dark shanks (neither lipochrome nor restrictor, *BB*, present but melanic pigment, *EE* and *dd*, present).

[2] All heterozygotes omitted.

[3] Genotypes at present not demonstrated.

[3]*wwddbbEE*	*wwd-b-EE*	dark shanks (lipochrome and melanic pigment, *EE* and *dd*, present and restrictor, *BB*, absent).

4. Non-barred—Non-black Series

WWDDbbee	*WWD-b-ee*	white shanks (no lipochrome, melanin, or restrictor present).
wwDDbbee	*wwD-b-ee*	yellow shanks (lipochrome present; melanic pigment and restrictor absent).
[3]*WWddbbee*	*WWd-b-ee*	dark shanks (blue) (melanic pigment, *dd*, present; lipochrome and restrictor absent).
[3]*wwddbbee*	*wwd-b-ee*	dark shanks (green) (lipochrome and melanic pigment, *dd*, present; restrictor absent).

5. Chromogen Series

A. Non-barred black

WWDDbbEECC	*WWD-b-EECC*	dark shanks (lacks lipochrome; melanic pigment, *EE*, and chromogen present).
wwDDbbEECC	*wwD-b-EECC*	dark shanks (lipochrome, melanic pigment, *EE*, and chromogen present).
WWDDbbEEcc	*WWD-b-EEcc*	blue or slaty blue shanks (lipochrome, restrictor, and chromogen absent; melanic pigment, *EE*, present).
wwDDbbEEcc	*wwD-b-EEcc*	green shanks (chromogen and restrictor absent; melanic pigment, *EE*, and lipochrome present).

B. Barred—Black

[3]*WWDDBBEECC*	*WWD-B-EECC*	white shanks (lacks lipochrome; melanic pigment, *EE*, restrictor, *BB*, and chromogen, *CC*, present).
wwDDBBEECC	*wwD-B-EECC*	yellow shanks (lipochrome, melanic pigment, *EE*, chromogen, *CC*, and restrictor, *BB*, all present).
[3]*WWDDBBEEcc*	*WWD-B-EEcc*	white shanks (as above, except chromogen genes lacking).

wwDDBBEEcc	*wwD-B-EEcc*	yellow shanks (as above, except chromogen genes lacking).

6. Inhibitor of Plumage Color Series

[3]*WWDDBBEEII*	*WWD-B-EEII*	white shanks (lacks lipochrome, with melanic pigment, *EE*, restrictor, *BB*, and inhibitor of plumage pigments *II* present).
wwDDBBEEII	*wwD-B-EEII*	yellow shanks (lipochrome, melanic pigment, *EE*, restrictor, *BB*, and inhibitor of plumage pigments, *II*, all present).
[3]*WWDDbbEEII*	*WWD-b-EEII*	blue shanks (lacks lipochrome and restrictor, *BB*, with melanic pigment *EE*, and inhibitor, *II*, present).
wwDDbbEEII	*wwD-b-EEII*	dark green shanks (restrictor, *BB*, absent; with lipochrome, melanic pigment, *EE*, and inhibitor of plumage pigments present).

SUMMARY

The genes *WW*, *Ww*, or *ww* are presumably present in all breeds and varieties of chickens, although evidence of their presence in the shanks is often masked by the deposition of melanic pigment in the shanks.

There is a single autosomal gene difference between white shank color (the absence of lipochrome pigment, *WW*) and yellow shank color (the presence of lipochrome color, *ww*) the former being dominant to the latter.

Dark blue and light blue shanks are caused by the deposition of melanic pigment in various cells of shanks that have a white skin for a ground color. Dark green and light green (willow) shanks are caused by the deposition of melanic pigment in the shanks which have a yellow skin for a ground color.

Black shanks are caused by the deposition of melanin in the shanks and appear the same in black plumage birds whether they have yellow or white skin color; however, one can be distinguished from the other in respect to skin color by examining the bottom of the feet. In the one case they will be white and in the other yellow.

The black plumage genes *EE* appear to have a general effect upon the deposition of melanic pigment and cause the deposition of melanic pigment in the shanks as well as in the plumage.

Any gene that affects the extension of black pigment in the plumage seems to have a restricting effect upon the melanic pigment in the shanks. This is especially true in the case of the barring genes.

Dark shank color caused by the black plumage genes, *EE*, is not sex-linked. However, it appears so when associated with modifying restrictive factors for the extension of black genes *EE* that are sex-linked, such as the barring factors.

There is still the possibility that there is a sex-linked dark shank color gene which has not thus far been adequately differentiated from the effect of black and barring plumage genes.

LITERATURE CITED

BARROWS, H. R., 1914 The histological basis of the different shank colors in the domestic fowl. Maine Agric. Expt. Sta. Bull. **232**: 237–252.

BATESON, W. and PUNNETT, R. C., 1911 The inheritance of the peculiar pigmentation of the Silky fowl. J. Genet. **1**: 185–203.

DUNN, L. C., 1925 The genetic relation of some shank colors of the domestic fowl. Anat. Rec. **31**: 343–344.

DUNN, L. C. and JULL, Morley A., 1927 On the inheritance of some characters of the Silky fowl. J. Genet. **19**: 27–63.

GADOW, HANS, 1891 Die Vögel, N. G. Bronn's Klassen und Ordnung des Tierreichs, Band 6, Abteilung 4. Integument oder System der äusseren Haut. p. 483.

HAGEDOORN, A. L., 1930 EEn Onderzoek Naar de Verschilpunten Tusschen het Barnvelderras en de Witte Leghorn, uit een Genetisch en Foktechnisch Oogpunt. Rapport aan het Bestur van de Nederlansche Genetische Vereen. Over de Fokproeven met Pluimvee in 1926, 1927, 1928–**29**: 1–38.

HANAU, A., 1881 Beiträge zur Histologie der Haut des Vögelfusses. Inaugural Dissertation, Bonn. Frankfort.

HERTWIG, PAULA, 1933 Geschlectsgebunde und autosomale Koppelungen bei Hühnern. Verhandl. der deuts. Zool. Ges. 112–118.

JEFFRIES, J. A., 1883 The epidermal system of birds. Proc. Boston Soc. Nat. Hist. **22**: 203.

JULL, MORLEY A., 1932 Poultry Breeding. New York: John Wiley & Sons, Inc. pp. 376.

LAMBERT, W. V. and KNOX, C. W., 1927 Genetic studies in poultry. II. The inheritance of skin color. Poul. Sci. **7**: 24–30.

MACARTHUR, JOHN W., 1933 Sex-linked genes in the fowl. Genetics **18**: 210–220.

PLATT, FRANK L., 1925 All breeds of poultry. Origin; History; Description, mating and characteristics. Amer. Poul. J. Chicago, Ill. pp. 226.

PUNNETT, R. C., 1923 Heredity in poultry. London: Macmillan and Co. pp. 204.

SEREBROVKSY, A. S. and WASSINA, E. T., 1926 On the topography of the sex chromosome in fowls. J. Genet. **17**: 211–216.

WARREN, D. C., 1928 Sex-linked characters in poultry. Genetics **13**: 421–433.