marker map which covers the chromosome adequately.

The results from this study confirm those from simulation studies (Hospital et al. 1992), which showed that markers can be used efficiently in backcross introgression breeding programs where the aim is to introgress a small part of the donor breed and simultaneously recover the recipient genome as quickly as possible. Using 2 to 11 markers per chromosome to select against the donor genome sped up the genome recovery by approximately two generations relative to random selection of individuals with the introgressed gene (Hospital et al. 1992).

The results shown here have implications for quantitative trait loci (QTL) mapping studies. In such experiments two breeds are usually crossed that are very different for a quantitative trait of interest, and marker and phenotypic data from a backcross or F_2 population are analyzed to find evidence for QTL. However, the standard null hypothesis usually is that there are no QTL segregating in a particular region, whereas we know that there must be genes somewhere in the genome which can explain the (large) breed difference. Alternatively, we may wish to test a genetic model of many linked loci which are fixed for alternative alleles in two breeds. Such a model would predict the relative weights of regression coefficients for individual markers if we would perform a multiple regression of phenotypes on all markers on a chromosome. The relative weights (regression coefficients) follow from the results presented in this study. These weights can be tested against the observed regression coefficients, and such a test was found to work well in simulation studies in that it could discriminate between genetic models based on a single QTL and models based on many linked QTLs (Visscher and Haley, in press).

Appendix

Relative Index Weights for Equally Spaced Markers

For the first backcross generation, ignoring subscripts for $t = 1$, let $X = \mathbf{b}'\mathbf{X}$ be an index of individual marker scores, with $\mathbf{b}$ an m × 1 vector of weights for marker scores X_i ($X_i = 0$ or 1/2), and $\mathbf{X}$ an m × 1 vector with observed values X_i. Markers are assumed to be evenly spaced along a chromosome with length L (Morgans). The index weights $\mathbf{b}$ are calculated so as to maximize the correlation between X and N. Index weights are calculated as $\mathbf{b} = (\mathbf{var}(\mathbf{X}))^{-1}\mathbf{y} = \mathbf{V}^{-1}\mathbf{y}$, with $\mathbf{y}$ a vector of covariances with $\mathbf{y}_i = \mathrm{cov}(X_i, N)$.

To show that the weights of b_1 and b_m are 1/2 relative to the weights for the other markers, we first show that $\mathbf{V}^{-1}$ is tridiagonal for the first backcross generation. Because we assume evenly spaced marker loci, all elements of $\mathbf{V}$ are functions of r, the recombination rate between adjacent marker loci. The matrix $\mathbf{V}$ has a special form in that for a particular value of $|i - j|$ all elements are identical. For example, for $|i - j| = 0$, $V_{ij} = 1/16$, for $|i - j| = 1$, $V_{ij} = (1/8)(1/2 - r)$, and for $|i - j| = 2$, $V_{ij} = (1/8)(1/2 - 2r(1 - r))$. In general, $V_{ij} = (1/16)[(e^{-2d})^{|i-j|}]$, with $d = L/(m - 1)$. The matrix $\mathbf{V}$ is an example of an autoregressive matrix, which means that element $\mathbf{V}_{ij}$ is proportional to the product of elements $\mathbf{V}_{ik}$ ($k = 1, j - 1$). This is true also if markers are not spaced evenly along the chromosome.

Because of the special form of $\mathbf{V}$ (an autoregressive matrix), its inverse is always a tridiagonal matrix with elements

$$V^{11} = V^{mm} = 16/(1 - e^{-4d}),$$

$$V^{ii} = 16(1 + e^{-4d})/(1 - e^{-4d}) \quad (\text{for } i > 1 \text{ and } i < m),$$

$$V^{ij} = -16e^{-2d}/(1 - e^{-4d}) \quad (\text{for } |i - j| = 1), \text{ and}$$

$$V^{ij} = 0 \quad (\text{for } |i - j| > 1).$$

The covariance between X_k and Z is

$$y_k = [\tfrac{1}{2}(1 - e^{-2d(k-1)}) + \tfrac{1}{2}(1 - e^{-2d(m-k)}]/(16L)$$

Multiplying $\mathbf{V}^{-1}$ with $\mathbf{y}$ gives, to a constant of proportionality,

$$b_1 = b_m \propto \tfrac{1}{2}(1 - 2e^{-2d} + e^{-4d}), \quad \text{and}$$

$$b_k \propto (1 - 2e^{-2d} + e^{-4d}), \quad \text{and}$$

$$b_k/b_1 = b_k/b_m = 2.$$

For $t > 1$, the inverse of $\mathbf{V}$ is not tridiagonal, but off-diagonals for $|i - j| > 1$ are relatively small, and the relative weights for b_1 and b_m are very close to 1/2. This was found empirically by calculating the regression coefficients for various combinations of t and m.

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Guinea Fowl Plumage Color Inheritance, With Particular Attention on the Dun Color

R. G. Somes, Jr.

The genetic basis of the dun-dundotte plumage color of the pearl guinea fowl was determined, and the relationship of this locus with that of the locus that causes the blue coloration was also studied. The dun color was shown to be the result of a single autosomal recessive gene, to which the gene symbol *d* was assigned. This locus was shown to be independent of the blue gene locus, and when both loci were homozygous for their mutant genes, a very dilute blue-dun color was produced. These results lead to a hypothesis of gene action for both the blue and dun genes.

The domestic pearl guinea fowl is a descendant of the native guinea fowl of West Africa (*Numida melegris*). This species has been domesticated for many centuries and was used by the Romans and Greeks as a table bird. During this extensive time period, at least four mutant genes whose influence is on plumage coloration have appeared and been conserved. Named color varieties have been developed around these four mutations and their various combinations, such that 17 distinguishable named phenotypes now exist (Table 1).

The Italian geneticist Alexandro Ghigi presented the first information on guinea fowl plumage color genetics at the 2nd

Table 1. Guinea fowl plumage color mutants

Phenotypes	Genotypes	Figure 1 reference
Pearl, pearl grey, grey, speckled, wild-type	M^+/M^+, I^+/I^+, D^+/D^+, w^+/w^+	A
Royal purple, violet	m/m, I^+/I^+, D^+/D^+, w^+/w^+	B
Lavender, lilac, light grey	M^+/M^+, i/i, D^+/D^+, w^+/w^+	C
Coral blue, sky blue, blue coral	m/m, i/i, D^+/D^+, w^+/w^+	D
Dundotte, chamois, buff dundotte	M^+/M^+, I^+/I^+, d/d, w^+/w^+	G
Dun, buff	m/m, I^+/I^+, d/d, w^+/w^+	H
Porcelain	M^+/M^+, i/i, d/d, w^+/w^+	I
Opaline	m/m, i/i, d/d, w^+/w^+	—
White	$-/-$, $-/-$, $-/-$, W/W	E
White-breasted pearl, splashed	M^+/M^+, I^+/I^+, D^+/D^+, W/w^+	F
White-breasted purple, lakenpur	m/m, I^+/I^+, D^+/D^+, W/w^+	—
Silverwing	M^+/M^+, i/i, D^+/D^+, W/w^+	F
Coral white	m/m, i/i, D^+/D^+, W/w^+	—
Dundotte white	M^+/M^+, I^+/I^+, d/d, W/w^+	—
Buff white	m/m, I^+/I^+, d/d, W/w^+	—
Porcelain white	M^+/M^+, i/i, d/d, W/w^+	—
Opal white	m/m, i/i, d/d, W/w+	—

World's Poultry Congress (Ghigi 1924) with a much later follow-up report at the 13th World's Poultry Congress (Ghigi 1966). Ghigi discussed three of the known color mutants: the sky blue color, white, and the loss of the pearl-like spots, or the "margarogene factor," as he called it. Of a fourth mutant color, a yellowish-white or light dun color, which Ghigi called "chamois" in his 1924 report, he stated "I do not yet know the gametic formula for the chamois. I simply say that a homozygous variety is in question which differs from the white and that the French classification which in their exhibitions constantly classes it with the white, is wrong." To my knowledge, the inheritance of this color has only been briefly reported (Somes 1988, 1990), and thus one of the purposes of this article is to cover the inheritance of this trait more fully.

Materials and Methods

A number of crosses were made over a period of 3 years using the pearl, blue, purple, and white color phenotypes in order to verify their genetic basis as they were reported by Ghigi (1924, 1966). The pictures in Figure 1 show the chick down and adult plumage colors of these various phenotypes.

The genetic basis of the color that Ghigi called chamois, which in this article is referred to as dun, was studied in a series of 16 crosses over a 3-year period (Tables 2 and 3). Many of these crosses were such that they also demonstrated the relationship between this gene and the gene for blue color (Table 3). Figure 1G,H,J-7,J-8 shows the chick down and adult plumage colors produced by the gene that causes the dun color.

Results

Ghigi Results Verified

The series of crosses designed to verify Ghigi's results did in fact do that, and thus the data are not presented here. However, in the following phenotype descriptions, the inheritance of the various mutant genes are presented. The phenotype known as pearl is the wild-type. This phenotype is a dark grey-black ground color covered with white spots or "pearls" as they are generally called (Figure 1A). Day-old chicks are a dark grey-black with reddish-brown streaks on the body and head (Figure 1J-4). The dark middle head streak is very large.

The purple phenotype, generally referred to as royal purple, is the result of an autosomal recessive gene (*m*), which prevents formation of the pearl markings, or the margarogene factor, as Ghigi called it. Adults of this color type are a very dark black-violet color with only a few pearl markings showing on the sides and under the wings (Figure 1B). Day-old chicks are a bit lighter than the wild-type pearl chicks on the dorsal surface and are white on the ventral surface and wings. The middle head streak is smaller and more wavy than that seen on the pearl chick (Figure 1J-3).

The blue coloration of the lavender (with the pearl markings, M^+/M^+) (Figure 1C) and the coral blue (lacking the pearl markings, m/m) (Figure 1D) phenotypes is the result of a single autosomal recessive gene (*i*) that reduces the intensity of the ground color to bright blue. Adults are a clear sky blue color with the lavender phenotype being uniformly flecked with white pearl markings (M^+/M^+), whereas the coral blue lacks these markings (m/m). Day-old blue chicks are a bright ash color with the head streaks of the lavender chick being similar to that of pearl chick (Figure 1J-2), and the head streaks of the coral blue chick similar to that of the purple chick (Figure 1J-1). The difference in head streaks reflects the presence or absence of the *m* gene.

The white plumage color, which is completely white in the adult, is a buff color in the chick and is the result of an incomplete dominant gene (*W*) (Figure 1E). When heterozygous for this gene (W/w^+), birds have a variegated tuxedo-type pattern in which the ventral surface shows varying degrees of white and the dorsal surface is the characteristic color determined by the other color genes in the genome. Figure 1F shows this tuxedo-type pattern on the lavender (left) and pearl (right) backgrounds. Figure J5 shows the ventral surface and Figure J6 the dorsal surface of this pattern on day-old chicks that are the purple color.

These three mutant color genes (*m, i,* and *W*) singly and in combination can produce nine distinct phenotypes, which along with their genotypes are listed in Table 1. The names listed for these phenotypes have been obtained from several sources (Ghigi 1924; Greenwood 1987; Van Hoesen and Stromberg 1975), but other names may possibly be in use for some of these phenotypes.

Genetics of the Dun Color

The intensity of color in the dun (without pearls markings) and dundotte (with pearl markings) phenotypes varies tremendously ranging from so light a color as to be almost white to a darker shade as seen in Figure 1G,H. Day-old chicks likewise have a wide range of color intensities (Figure 1J-7,J-8). Those shown in Figure 1J-7,J-8 are on the darker side.

The genetic data that explains the inheritance of the locus that controls the dun color are presented in Table 2. This color is shown to breed true and to be inherited as an autosomal recessive trait. The gene symbol, *d,* is proposed for this trait.

Because blue coloration was also inherited as an autosomal recessive, it became desirous to see how these two genes would react in the double homozygous state (i/i d/d). Over a 3-year period, 12 crosses were made that involved both of these genes. These crosses not only verified the genetic basis of these two genes, but they also produced a new phenotype, which was the double homozygote. These

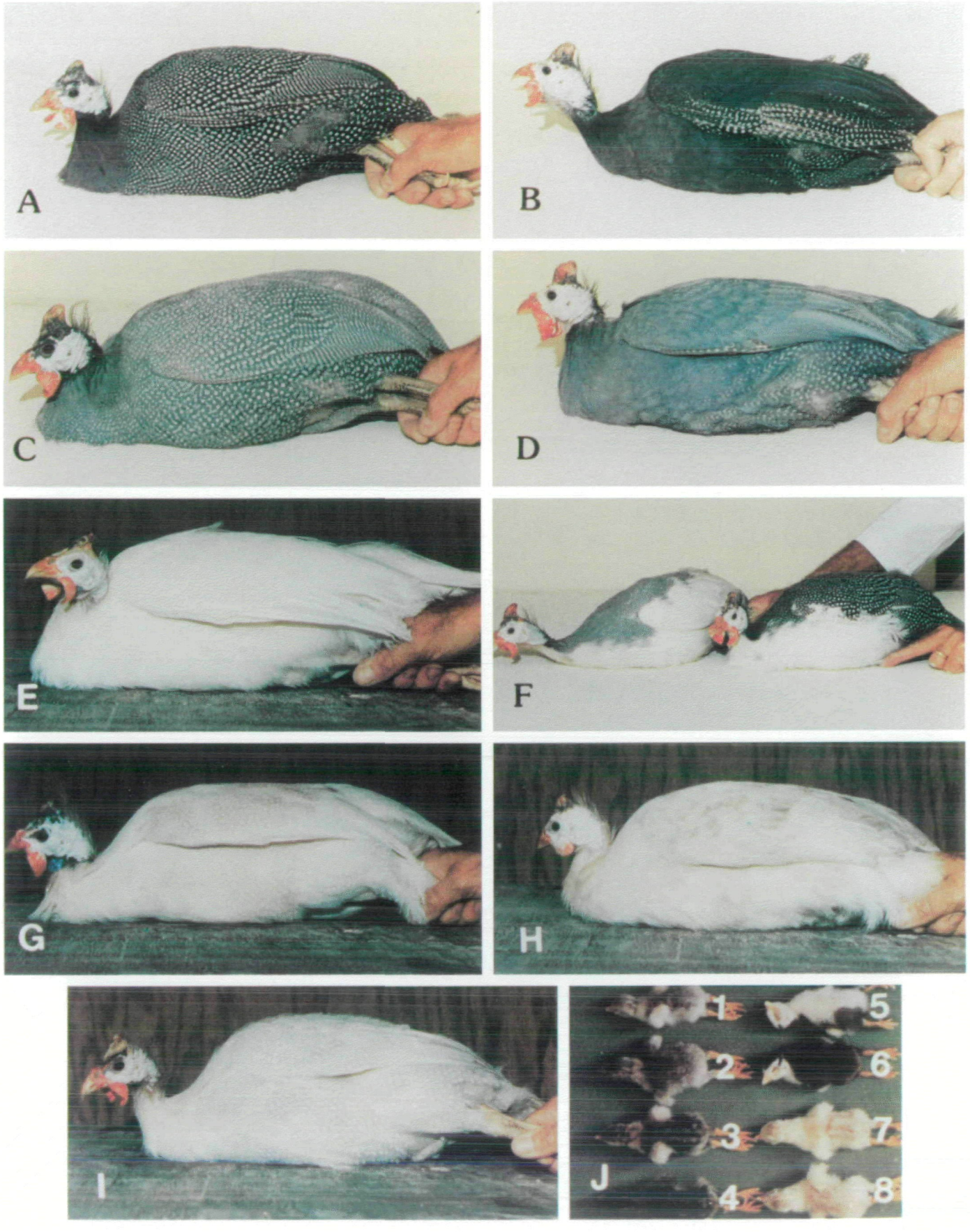

Figure 1. Plumage color phenotypes of adult guinea fowl: (A) wild-type pearl, (B) royal purple, (C) lavender, (D) coral blue, (E) white, (F) heterozygous for white gene on lavender and pearl backgrounds, (G) dundotte, (H) dun, (I) blue-dun, and (J) day-old guinea fowl chicks—(J-1) coral blue, (J-2) lavender, (J-3) royal purple, (J-4) wild-type pearl, (J-5) ventral surface of white-breasted purple, (J-6) dorsal surface of white-breasted purple, (J-7) dun, and (J-8) dundotte.

Table 2. Guinea fowl crosses involving the dun color trait

Crosses			Progeny phenotypes						
Male		Females	Pearl	Dun	Blue	Total	Ratio	χ^2	*P*
Dun	×	dun	—	23	—	23	all	0.00	1.00
Dun	×	pearl	7	—	—	7	all	0.00	1.00
Pearl	×	dun	20	—	—	20	all	0.00	1.00
F_1	×	F_1	37	11	—	48	3:1	0.11	.74

data are presented in Table 3. It is evident from the various segregations that these two genes are not alleles and that the blue gene (*i*) is epistatic to the dun gene (*d*). When both genes are homozygous, a new phenotype is produced, which is referred to here as blue-dun and is shown in the last cross in Table 3 to breed true. This is the phenotype that some have referred to as opaline or porcelain (Table 1). I have used the term blue-dun in this article because it is more descriptive of the phenotype than either opaline or porcelain. The adult phenotype of this new color is dun with a very light blue "wash" over it. The example shown in Figure 1I is more intense in color than most. In this example, the blue wash almost covers the dun color. The chicks are very difficult to distinguish from dun-colored chicks, and so classification at 1 day of age had to be verified at a later age.

Discussion

The guinea fowl has four known loci that influence its plumage color. All of these loci are autosomal with three being recessive (*d, i, m*) and the fourth incompletely dominant (*W*). The *W* locus inhibits the production of all pigments when homozygous, thus producing an all white bird. When heterozygous, this gene's pigment inhibiting ability is restricted to only the ventral surface, thus producing a tuxedo-like phenotype. The *W* allele at this locus is therefore a pigment-inhibiting gene.

In the wild-type pearl guinea fowl, the dominant allele (M^+) at the margarogene factor locus inhibits pigmentation in such a way that rows of small white spots or "pearls" are present on all the feathers. The recessive allele at this locus (*m*) prevents this particular systematic pigment inhibition from taking place, and birds are basically the dark black-violet ground color all over. Thus, this mutant gene (*m*) functions in such a way as to disrupt the normal pattern producing pigment inhibition that is characteristic of the wild-type and therefore leads to a nearly solid colored plumage.

The blue ground color that is produced by the mutant *i* allele at the I^+ locus is a bright sky blue color and is much clearer and brighter than the blues normally seen in other galliform species. This blue color is more like that seen in the budgerigar, the common parakeet. Blue color in feathers is not due to blue pigment, but is due to structural changes that cause light scattering or diffusion of the light, the so-called Tyndall blue (Simon 1971). Most blue feathers in galliform species are a dark blue-gray color and not bright sky blue as in the guinea fowl. The *i* gene's function is thus one of replacing the dark black-violet ground color of the wild-type with a sky blue color. However, the exact structural change that occurs is unknown at this time.

The dun ground color was shown in this study to have a simple autosomal recessive type of inheritance (*d*). Its action would appear to be one of inhibiting the production of eumelanin, at least when in combination with the I^+/I^+ and I^+/i genotypes. It completely inhibits the dark black-violet pigment that is characteristic of both M^+/M^+ and *m/m* birds. Whether the dun-colored pigment is produced instead of the black eumelanin or is normally present but covered by eumelanin and thus not normally seen is not known.

A surprising result from this study was the appearance of blue (although diluted in intensity) when the dun genes were in combination with the genes for blue (*d/d i/i*). It indicated that the dun gene acted differently when in the *i/i* genotype than in the I^+/I^+ or I^+/i genotypes. This result was not expected, but this difference may suggest the mode of action of the blue gene which results in the production of the blue phenotype. Simon (1971) stated that electron micrographs of feathers from birds that are bright blue, when viewed in transverse sections, show the feather branches to be filled in their centers with densely packed dark melanin particles. Suspended in the horny keratin outer layer of the feather branches are cells in which are suspended tiny melanin particles scattered throughout the cells but not densely packed. These tiny melanin particles in these cells refract the light and reflect the blue light, whereas all other colors are absorbed by the dark melanin in the center of the feather branches. He also reported that in the case of the budgerigar there is a mutant gene that inhibits the production of the dark melanin in the center of the feather and when this happens the bright blue is replaced by a pale blue.

Because the blue feather color of the

Table 3. Guinea fowl single pair matings involving the dun and blue color traits

Crosses			Progeny phenotypes							
Male		Females	Pearl	Dun	Blue	Blue-dun	Total	Ratio	χ^2	*P*
Blue (*i/i, D⁺/D⁺*)	×	Dun (H)[a] (*I⁺/i, d/d*)	20	—	17	—	37	1:1	0.24	0.62
Blue (H) (*i/i, D⁺/d*)	×	Blue (H) (*i/i, D⁺/d*)	—	—	92	19	111	3:1	3.71	0.05
Dun (H) (*I⁺/i, d/d*)	×	Dun (H) (*I⁺/i, d/d*)	—	34	—	6	40	3:1	2.13	0.15
Dun (H) (*I⁺/i, d/d*)	×	Blue (H) (*i/i, D⁺/d*)	6	6	10	5	27	1:1:1:1	2.18	0.54
Blue (H) (*i/i, D⁺/d*)	×	Dun (H) (*I⁺/i, d/d*)	17	16	27	11	71	1:1:1:1	7.59	0.06
Blue (H) (*i/i, D⁺/d*)	×	Pearl (HH)[b] (*I⁺/i, D⁺/d*)	25	9	25	4	63	3:1:3:1	2.23	0 53
Blue (*i/i, D⁺/D⁺*)	×	Blue-dun (*i/i, d/d*)	—	—	17	—	17	all	0.00	1.00
Dun (*I⁺/I⁺, d/d*)	×	Blue-dun (*i/i, d/d*)	—	5	—	—	5	all	0.00	1.00
Pearl (HH) (*I⁺/i, D⁺/d*)	×	Blue-dun (*i/i, d/d*)	5	5	6	1	17	1:1:1:1	3.47	0.32
Blue (H) (*i/i, D⁺/d*)	×	Blue-dun (*i/i, d/d*)	—	—	18	18	36	1:1	0.00	1.00
Dun (H) (*I⁺/i, d/d*)	×	Blue-dun (*i/i, d/d*)	—	13	—	12	25	1:1	0.04	0.84
Blue-dun (*i/i, d/d*)	×	Blue-dun (*i/i, d/d*)	—	—	—	23	23	all	0.00	1.00

[a] (H) = heterozygous for other color, dun or blue.

[b] (HH) = heterozygous for both dun and blue.

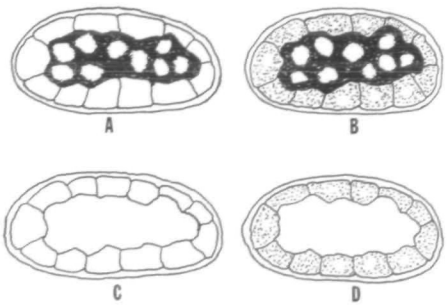

Figure 2. Transverse sections of feather branches with proposed melanin pigment distributions to account for the observed affects of the genes for both the blue and dun colors: (A) pearl and royal purple, (B) blue, (C) dun, and (D) blue-dun.

guinea fowl is similar to that of the budgerigar, I would suggest that the mode of action of the guinea fowl *i* and *d* genes may be the same as the budgerigar genes mentioned by Simon (1971) in the previous paragraph.

If this suggested mechanism is in fact true, then the difference between the blues and the darker colored pearl and royal purple guinea fowl is the presence of tiny melanin particles in the cells of the horny keratin outer layer of the feather branches of the blue-colored birds, whereas in the darker phenotypes these melanin particles are absent. Both color types however have densely packed melanin particles in the center of the feather. The action of the dun gene would then be to remove the densely packed melanin particles from the center of the feather branches. In the case of the blue phenotype, the color would now change to a pale blue color. These proposed gene actions are illustrated in Figure 2.

If the dun color is in fact the result of the lack of the densely packed melanin particles, then this would also imply that the dun-type pigment is normally always in the feather and that it is only able to be seen when the darker pigment is not produced. Proof of this proposed mechanism for the gene action of both the blue and dun genes (*i* and *d*) could easily be verified with some electron micrographs of these features. However, I am now retired, and so this proof must be left for someone else to obtain. Some other mechanism may be responsible for the results seen in this study, but I feel that the proposal suggested here best fits the results seen in this study and previously reported feather pigment work done by others and reported in the literature.

Not only has this study determined the inherited basis of the dun feather color, but it has led to suggested mechanisms for the gene action of both the blue (*i*) and dun (*d*) genes.

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The Genetics of the Mimetic Coloration in the Butterfly *Heliconius cydno weymeri*

M. Linares

The genetic bases of the wing color pattern in the neotropical butterfly *Heliconius cydno weymeri* were investigated. Evidence from F_1 broods of wild-caught females indicates that the studied subspecies is composed of two mimetic forms, *weymeri* and *gustavi*, which differ mainly by single allele substitution of major phenotypic effect. Three additional Mendelian genes are hypothesized to possess alleles that contribute to Müllerian mimicry with two alternative model species (mimicked by the two mentioned polymorphic forms), and a fifth one is hypothesized to possess alleles that are mimetically irrelevant. Segregation occurred at all five putative loci. Most of the broods show ratios consistent with simple Mendelian segregation. Broods inconsistent with simple Mendelian inheritance can be explained by (1) a possible epistatic interaction between some of the hypothesized loci and/or the modifier effect of two addtional genes; and (2) a possible effect of sex on the expression of one of the hypothesized loci. There is evidence that the genetic system has evolved epistatic interactions in order to facilitate mimetic resemblance. There is no evidence of linkage between mimetically relevant loci except for one pair of these. This is the first report on the genetic bases of the wing color pattern variation of the species *Heliconius cydno.*

Evolutionary biologists have debated for a long time whether adaptations result from the accumulation of many allele substitutions of small effect ("micromutationism") or from the accumulation of gene substitutions of large effect (which I could call "macromutationism"; Charlesworth et al. 1982; Goldschmidt 1940; Orr and Coyne 1992). In order to account for the evolution of adaptations, it is fundamental to elucidate the genetic bases of concrete examples of these biological attributes and infer the nature, number, and magnitude of the gene substitutions that conform them.

A good example of an adaptation is Müllerian mimicry which is the phenotypic close resemblance between two, or more, distasteful relatively distantly related species. The wing pattern of *Heliconius* butterflies represents an excellent case of Müllerian mimetic coloration shaped mainly by natural selection (Benson 1972; Brown et al. 1974; Mallet 1986, 1989; Mallet and Barton 1989a,b; Mallet et al. 1990). These insects can be cultured and used for studying the genetic bases of a major adaptation (Müllerian mimicry), through hybridization experiments and genetic analysis, providing valuable information that may contribute to resolve the debate between micro- and macromutationists mentioned above. In this article I present results on the genetic bases of the Müllerian mimetic wing color pattern variation in the butterfly subspecies *Heliconius cydno weymeri* (for a list of studies involving *Heliconius* genetics, see Mallet 1993). Furthermore, this is one of the most detailed studies on the genetics of a *Heliconius* species, involved in Müllerian mimicry simultaneously with another member of *Heliconius* and one of the subfamily Ithomiinae (see below), in which the polymorphism does not seem to be maintained through the interaction of natural hybridization between differentiated subspecies, and selection on mimetic patterns, as in most of races of *Heliconius* studied by Turner and