SOMATIC MOSAICS IN THE DOMESTIC PIGEON

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Received August 4, 1939

MOSAIC or chimeric effects not of artificial origin may conveniently be divided into two classes, those which are frequent, and customarily associated with special genotypes, and those which are rare and unpredictable. Variegation and eversporting are examples of the former class. In the pigeon a condition known as "flecking" is of this sort. The other class includes gynandromorphs and chimeras generally.

These phenomena have received little attention in the pigeon. We shall present a review of the known facts, together with new cases which have come under our observation, and suggestions as to the possible causes involved.

FLECKING ASSOCIATED WITH SEX-LINKED FACTORS

In the wild type or "blue, black-barred" pigeon (*Columba livia*) no flecking has been observed. Flecking is found associated with three sex-linked color factors, which, as indicated later, are probably all alleles. Each of these is dominant to its wild-type allele, and each is responsible primarily for a particular kind of "bleaching" effect, that is, the plumage is lighter in color than that of the wild type. The apparent mutation, inactivation, or loss of such a factor in areas of irregular size and distribution in the feathers produces the flecking.

The most common of these factors is the "dominant red" of COLE and KELLEY (1919), or, as we shall call it, more specifically, "ash-red" (figure 1). HAWKINS (1931) demonstrated that the factor for sex-linked recessive "chocolate-brown" plumage color is a third allele at the ash-red locus. He symbolized these alleles, in descending order of dominance, B^A (ash-red), B (wild type), and b (chocolate-brown). HAWKINS reviewed the literature on flecking and reported new observations of his own. He found flecks only in heterozygous males, and suggested that flecking is merely a consequence of heterozygosity. Thus, males of the constitution B^AB showed only flecks of B phenotype, while males of the constitution B^Ab showed only flecks of the b phenotype; females, since they possess but one sex chromosome, cannot be heterozygous in the strict use of the term.

However, STEELE (1926) had definitely stated that he had observed "dunnish" flecks in dominant red females. We also have found flecking in many such females, and the flecks are invariably of the b type. HAWKINS'

[1] Papers from the Department of Genetics, Agricultural Experiment Station, No. 246.

suggestion that flecking is due to heterozygosity as such is therefore inadequate.

The sex difference in flecking was vaguely recognized for a long time by breeders (Cf. DARWIN, 1900, Vol. I, Chap. 5, p. 167), but the first clear outline was given by STEELE (1926). He stated that males more often exhibit flecking than do females; that flecked males generally show more flecks than do flecked females; and that flecks in females are not as dark in color as those in males. HAWKINS (1931) attempted to find whether an

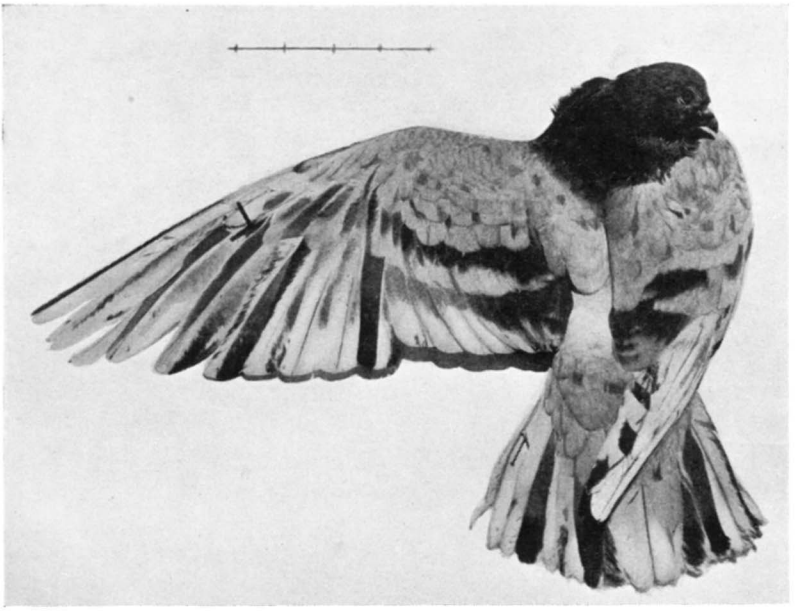

FIGURE 1.—Male D325Z, age 8½ years, showing large amount of black flecking. Genotype: $B^A B$ (ash-red).

endocrine differential is involved by castrating flecked males and observing subsequent feather regeneration. No change in fleck color or in general incidence of flecking was found.

Age has an important influence on flecking. The sex differences are already apparent in the juvenile plumage, but the flecking tends to increase in amount with each moult, so that, in old males particularly, it is extremely marked (figure 1).

The second most common factor associated with flecking is "almond," symbolized *St* by WRIEDT and CHRISTIE (1925). The color is basically yellowish-ashy to white, depending on associated factors. This factor is characteristic of the almond Tumbler, Oriental Roller, and Magnani Modena breeds. Flecking is abundant in both sexes, but as GHIGI (1908)

has shown, there is a marked sex-difference in quantity, as with ash-red. Unlike ash-red, the flecks in almond females are not chocolate in color, but show black (wild type) pigment. The flecking increases with each moult, as in the case of ash-red; old males may become more black than almond in appearance.

The third factor associated with flecking is "faded," *Of*, (HOLLANDER 1938). It has since been proved to be sex-linked. The general effect of this factor is much less obvious than almond or ash-red; the plumage is only slightly bleached. Flecking in faded birds is about the same in amount as that in heterozygous young ash-red males. It differs from ash-red and almond in that no sex difference or age difference has been apparent, but as in almond, the females have black pigmented flecks.

The relationships of all these three factors to one another are not yet fully determined but allelism is strongly indicated. Linkage tests of almond with another sex-linked factor, the recessive "dilution," *d*, were made by WRIEDT and CHRISTIE (1925); crossing over was around 50 percent. The amount of crossing over between ash-red and dilution was also very high in tests made by COLE and KELLEY (1919) and subsequent investigators. Since almond and ash-red both appear to lie at a distance from dilution, they probably are close neighbors of one another, if not actually allelic. FELDMAN (unpublished)[2] made a test of this possibility. Males heterozygous for ash-red and almond were produced; these resembled almond except that the flecking was of the ash-red color. Preliminary breeding tests with these males gave no crossing over.

We have obtained the male heterozygote of almond and chocolate. In this case a most unexpected result appeared: the flecks are chiefly chocolate, but a few are wild type. No linkage tests have yet been attempted. Faded has not yet been tested with almond or with chocolate, but it should be noted that FELDMAN obtained faded originally in the son of an almond ("Parlor Tumbler") female. By the rules of sex-linked inheritance, this son should have been almond; the best explanation so far advanced is that almond gave rise to faded by mutation. In preliminary linkage tests with dilution, faded has given a high rate of crossing over, while with ash-red it has given only non-crossovers. Males heterozygous for ash-red and faded resemble ash-red in general but their flecks are of the faded coloration.

In view of the above indications that almond and faded are situated at the B^A locus, we shall for purposes of discussion assume that all are alleles, and refer to them as the *B* series.

[2] The work referred to here was done by DR. H. W. FELDMAN at the University of Michigan He has generously given us valuable birds of the almond and faded types, as well as access to his records.

FLECKING ASSOCIATED WITH AN AUTOSOMAL FACTOR

Only one autosomal factor has been found accompanied by flecking. This factor is "grizzle" (*G*), also dominant to the wild type, and responsible for a whitening of the plumage. In a few heterozygotes we have observed large portions of feathers clearly lacking the grizzle factor, and therefore quite comparable to flecking associated with the sex-linked factors. It is difficult however to be certain that small areas lack grizzle. No further study of the flecking with this factor has been undertaken.

MUTATION IN THE SEX-LINKED B SERIES

This series may be symbolized provisionally in descending order of dominance, B^{St} (almond) $>B^{A}$ (ash-red) $>B^{Of}$ (faded) $>B$ (wild type) $>$ b (chocolate). The general effect of the genes dominant to wild type is to reduce the black pigmentation gradually to light gray or white in some areas and to red or yellow in others. These light colors serve as a contrasting background for dark flecks of a more recessive color. On the hypothesis that the dominant gene is completely lost or inactivated in the area of the fleck, the color of the fleck should be governed by the remaining allele. Thus if the gene B^{A} were eliminated in the heterozygote B^{A} B the fleck pigmentation should be wild type, while in the heterozygote B^{A} b the fleck pigmentation should be chocolate. In females of any of the three dominant types chocolate flecks (the "residual" condition) would be expected on the assumption that b is the lowest possible allele at this locus, but such is not the case for almond or faded. Furthermore, the male almond-chocolate heterozygote, B^{St} b, has not only flecks of the expected chocolate pigmentation but also some of wild type. The conclusion seems unavoidable that there is not actual loss or complete inactivation of the dominant allele in all cases. This also precludes an explanation based on the loss of an entire chromosome and further evidence against such loss is found in males heterozygous for both the ash-red and the dilution loci of the sex chromosome: B^{A} D/B d. The flecks in such males were always black. If the whole chromosome containing B^{A} were lost also in some cases, the flecks should then show the dilution phenotype, provided that this factor can produce its effect autonomously in development. That it can so act is indicated later in connection with chimeras.

A more plausible genetic basis for the flecking seems to be offered by the hypothesis of somatic mutation of labile genes. Such genes have been shown to exist in a number of species of animals and plants. In this case it is only necessary to assume that the dominant genes tend to mutate to alleles lower in the series, but not always to the same one even in a single bird. Presumably these mutations occur in homozygotes equally as often as in heterozygotes, but no visible result would be expected unless both

genes in the cell mutated. Similarly, in males heterozygous for two of the dominants, mutation of the lower allele would never be observable unless by coincidence the higher gene mutated also. There is no evidence that either the wild type or chocolate alleles are at all labile; apparently mutation is a characteristic of the genes dominant to wild type and the higher the allele in the series, the more labile it is. Since almond heterozygotes show such a large amount of flecking, we would not be surprised if almond homozygotes should show a few flecks; the mutation rate seems so high that both genes might well occasionally mutate in the same cell. However, no observations on mature male almond homozygotes have yet been made.

In table 1 is summarized present knowledge of the *B* series genotypes and phenotypes, with notes on the flecking.

FLECKING IN OTHER BIRDS

The pigeon is not alone among birds in exhibiting flecking. Essentially the same phenomenon, under names such as "fault feathering" or "exceptional feathers," is found in the "blue" domestic duck, the slate turkey, and several sorts of chickens, such as Barred Plymouth Rocks, Andalusians, and dominant whites. In certain of these forms it has received considerable attention both from breeders and from investigators, and has been recognized as a special phase of the problem of coloration because of the high degree of irregularity. In all these species, as in the pigeon, the genetic factors involved are more or less dominant to the wild type; furthermore, the flecking does not occur in homozygotes, except in the white Andalusian fowl. The Andalusian, seeming exceptional, requires special discussion. SEREBROVKSY (1926) states that flecking in the heterozygous ("blue") Andalusians is always black (wild type allele), while it is "blue" in homozygous whites, and much more striking than in heterozygotes. He explains these facts by the assumption that "loss" of one allele occurs. The homozygote having two labile alleles, blue flecking should be twice as abundant as in heterozygotes. We have examined a number of Andalusians, and find SEREBROVSKY'S treatment satisfactory. We may add that within large blue flecks (in homozygotes) black flecks are occasionally to be found. These would indicate "loss" of both alleles. The reason for the peculiar status of the Andalusians seems simply the fact that the factor involved is only partially dominant to the wild type allele, the heterozygous condition being phenotypically distinct.

The Andalusian factor and dominant white in fowls are not sex-linked, and apparently sex-linkage is not involved in the duck or turkey. But most of the investigation of the flecking has centered around two independent dominant sex-linked color factors, "bar" and "silver," in the chicken. SEREBROVKSY (1926) and HERTWIG and RITTERSHAUS (1929)

TABLE 1

Genotypes and Phenotypes of the B Series.

GENOTYPE	BASIC COLOR	FLECKS	EXPECTATION ON BASIS OF LOSS
		Almond Genotypes	
Males			
$B^{St}\ B^{St}$	*	*	Creamy; no flecks
$B^{St}\ B^{A}$	Creamy	Ash-red	As found
$B^{St}\ B^{Of}$	*	*	Creamy; faded flecks
$B^{St}\ B$	Creamy	Black	As found
$B^{St}\ b$	Creamy	Chocolate and black	Only chocolate flecks
Females			
$B^{St}-$	Creamy	Black	Chocolate flecks
		Ash-red Series	
Males			
$B^{A}\ B^{A}$	Ash-red	None	As found
$B^{A}\ B^{Of}$	Ash-red	Faded	As found
$B^{A}\ B$	Ash-red	Black	As found
$B^{A}\ b$	Ash-red	Chocolate	As found
Females			
$B^{A}-$	Ash-red	Chocolate	As found
		Faded Series	
Males			
$B^{Of}\ B^{Of}$	*	*	Faded, no flecks
$B^{Of}\ B$	Faded	Black	As found
$B^{Of}\ b$	*	*	Faded; chocolate flecks
Females			
B^{Of}	Faded	Black	Faded; chocolate flecks
		Black Series	
Males			
$B\ B$	Black	None	No flecks
$B\ b$	Black	None	Chocolate flecks if mutation occurred
Females			
$B-$	Black	None	Chocolate flecks if mutation occurred
		Chocolate	
Males			
$b\ b$	Chocolate	None	As found
Females			
$b-$	Chocolate	None	As found

* Not studied or insufficient information.

observed the frequencies of each kind of fleck in Barred Plymouth Rocks and crosses possessing both these factors, and in each sex. SEREBROVSKY concluded that segregation of whole sex chromosomes, with or without crossing over, would adequately explain the facts, while the latter authors decided that only part of the chromosome—usually the part containing

the bar factor—was lost. In either case, a genetic change in the soma appeared to account for the exceptional feathers.

HERTWIG and RITTERSHAUS gave as further evidence of a genetic change that, after plucking, the fault feathers are replaced by new ones of the same type. JUHN (1933) has demonstrated however that this result is not regularly found. She concludes that her findings "do not support the genetic interpretations advanced," which, she adds, "are untenable from an embryological point of view," though she does not specify in what way. Her explanation of the exceptional feathers is that a physiological threshold allows the recessive factors in the birds' make-up to function at times, depending on "the interaction of the genetic factors . . . with variable morphogenetic factors such as rate of growth . . . " In other words, a physiological control of the action of these genes is postulated, the dominant being expressed under certain local internal conditions, and suppressed under others.

MONTALENTI (1934) has also investigated feather succession in Barred Plymouth Rocks. He found that follicles from which "abnormal" feathers are plucked always regenerated abnormal feathers, though in most cases the successive feathers differed considerably. If the feathers were completely abnormal (black, in these chickens) they were generally followed by new ones of exactly the same sort. MONTALENTI sums up his observations and deductions as follows: "It appears . . . that the *range of action of the genes for the barring* in mosaic feathers may vary considerably in successive generations. Sometimes the barring does not appear at all in some of them, although this factor is potentially present in the follicles concerned, as it is proved by its manifestation in successive generations of feathers." (Italics ours.)

THE DEVELOPMENTAL BASIS OF FLECKING

We have concluded so far, on the basis of fleck coloration, that flecking in pigeons is the result of genetic change in the soma, and that this change is probably mutational in nature. We must next examine the facts of feather development to determine whether this conclusion can be accepted.

We have observed successive feathers from a large number of follicles in different specimens of the *B* series. As in chickens, there is a great deal of variability in the successive feathers; nevertheless, where the fleck is so large as to extend from the tip to the base of the feather, and especially if an entire vane or more is involved, repetition in successive feathers is obvious (figure 2). The smaller the flecks the less tendency there is to repeat in detail, but the same absolute variability would be expected to produce more apparent alteration of the small flecks than of the large

ones. At any rate, we may conclude that in the case of the great flecks, a relatively permanent differentiation exists in the feather primordium with respect to potentialities for color production.

If we interpret LILLIE and JUHN'S latest (1938) contribution correctly, they now consider growth of the feather to be mainly axial, rather than concrescent as earlier assumed. Any genetic difference existing in the "collar," such as might occur from mutation, should therefore result in a

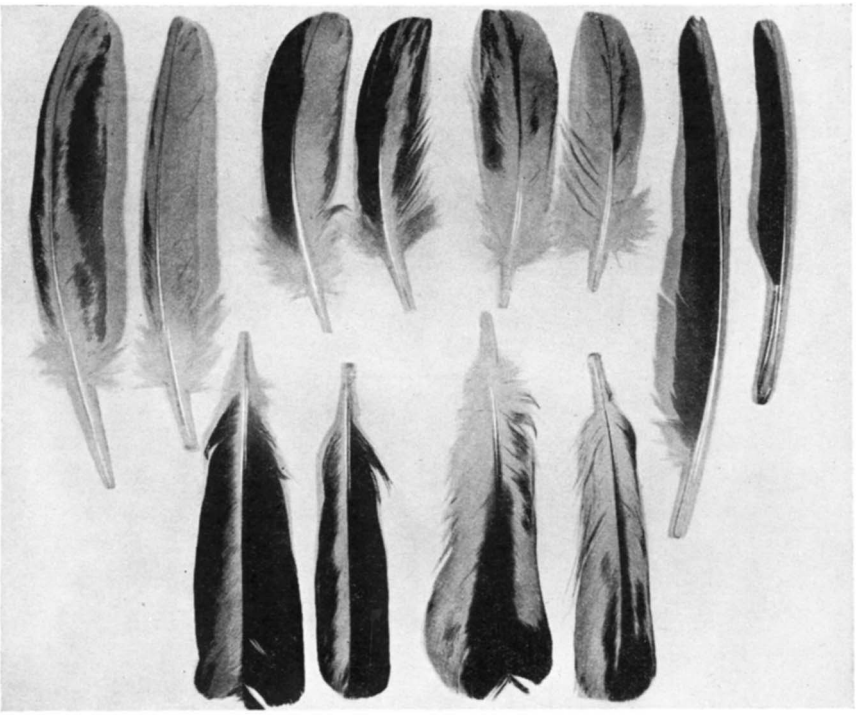

FIGURE 2.—Pairs of successive feathers from six follicles from aged flecked male, 2515E (genotype B^A B). In each pair, the younger feather is to the right of the older. Above, wing feathers; below, tail feathers. The first feathers were all pulled the same day, and the successors a month later. Note that the more outstanding fleck areas (black) tend to be repeated, with fair fidelity in most cases, and that the fleck areas in general run longitudinally.

corresponding longitudinal streak in the completed feather. This condition is often approximated by the larger flecks, and the structural difference shown in figure 15 gives striking evidence of definite axial growth. The small flecks appear as irregular islands. The irregularities in shape and size, as well as in replacement, may be accounted for by one or more of several processes: 1. New mutations which would affect very small areas may be occurring intermittently in the active part of the collar. 2. Irregularities of outline and lack of continuity, producing flecks rather than continuous streaks, may result from uneven relative growth of different

derivative cells in the collar. 3. As DANFORTH (1939) has emphasized, the early precursors of the pigment cells are apparently capable of migration; furthermore, these specialized cells are large and have long branching processes which deposit pigment some distance from the original position of the cell (GREITE 1934).

That physiological differences and changes may have an influence on the incidence of mutation and possibly on the relative growth of the mutated portions is as yet undeniable. Sex and age differences in the quantity of flecking, as described in connection with ash-red and almond, point to some measure of metabolic control. Subsidiary genetic factors, subject to selection, also may have an influence on the degree of flecking, as is shown by differences in specimens physiologically comparable.

CHIMERAS

Gynandromorphism is a well-known type of chimera in domestic fowls, according to CREW and MUNRO (1938), but no case has been reported in pigeons. The lack may be due to the difficulty in recognizing sexually abnormal pigeons; external differences between male and female are so slight that a gynandromorph might easily be overlooked.

Chimeras of feather color and structure have, however, occasionally been observed. The flecking and chimera types have much in common but chimeras differ from the flecking type in being relatively rare and unpredictable in occurrence, and in having larger areas affected, including whole groups of feathers, and even considerable portions of the body. For convenience, and without any necessary implication as to cause, these will be referred to as the "mutant areas." They are, in general, though not always, similar to flecking in being attributable to some sort of loss of the dominant allele in a heterozygote. To account for the large areas involved, it must be assumed that the "mutation" occurred earlier in ontogeny than is the case with flecking, possibly in some cases even as early as the first cleavage division.

For convenience of treatment, the pigeon chimeras which have come to our attention (26 in all) are classified below according to the principal factors involved.

1. *Chimeras involving ash-red* (B^4).

LYELL (1877, p. 48) states that he once bred a "mealy with black shoulders." It is clear from his other descriptions that by "mealy" LYELL refers to the ash-red condition, while the mutant area is of the *B* phenotype. He did not give sex or pedigree of the specimen.

Two somewhat similar male mosaics have been studied at this laboratory. The first, male 2067B, was a crossbred Tumbler produced by an

ash-red male and a black female. Most of the plumage of this specimen was typically ash-red with black flecks, but on the crop, back of the head, and inter-scapular region were large patches of black feathers. These patches remained during the six years of the bird's life. A progeny test indicated that his constitution was $B^{A}B$, as was to be expected. The second mosaic, 2411A, exhibited much more extensive B areas (figure 3).

FIGURE 3.—Diagrams of male chimera 2411A. In these and following diagrams, black represents the dominant allele, stippling represents the recessive allele, and white represents white-spotting. Dominant allele here, B^{A} (ash-red); recessive allele, B (blue checker).

This bird was a Homing pigeon obtained from a Milwaukee, Wisconsin, breeder; the parents were said to be an ash-red male and a wild-type-colored female. The mosaic was tested with a wild type female; of the five offspring obtained, two were typical ash-red, and three showed wild-type pigmentation. Thus the gonads were, at least in part, of the constitution $B^{A}B$.

LEVI and HOLLANDER (1939) report two additional cases, with illustrations. Both were $B^{A}B$ males, by pedigree and, in the one case tested, by progeny test. Both showed a large amount of B plumage, and both possessed white-spotting, extensive in one. A most unusual feature of one of these mosaics was the change, in the transition from juvenile to adult plumage, of a large part of the B plumage in the tail back to the proper color, ash-red. Such reversion has not been observed in any other chimera.

A male bird (E284E) of genotype $B^{A}B$ had relatively few and small flecks in its juvenal plumage. Now, in its first adult plumage, it has abundant flecks and by the time it is several years old it will presumably be fully as flecked as D325Z, shown in figure 1. In addition, E284E has on the left side of its head a black patch which extends from near the base of the beak, beneath the eye to the occiput (figure 4). The total area of this patch is less than might occur on a single tail feather; it differs from the latter, however, in that it involves a good many contiguous feathers instead of a single one, and its extent has not increased with the molt.

This specimen may accordingly be included as a chimera.

In the above six cases, the mosaicism can be accounted for by the hypothesis that the B^A factor, in heterozygotes, has mutated to B (or b) or become lost or otherwise inactivated, as in the case of flecking, but at some time early in ontogeny.

A more complicated problem is presented by male 2721E, bred at this laboratory. In addition to the sex-linked ash-red factor, an autosomal feather pattern factor is involved. The bird's father was a dun of the T

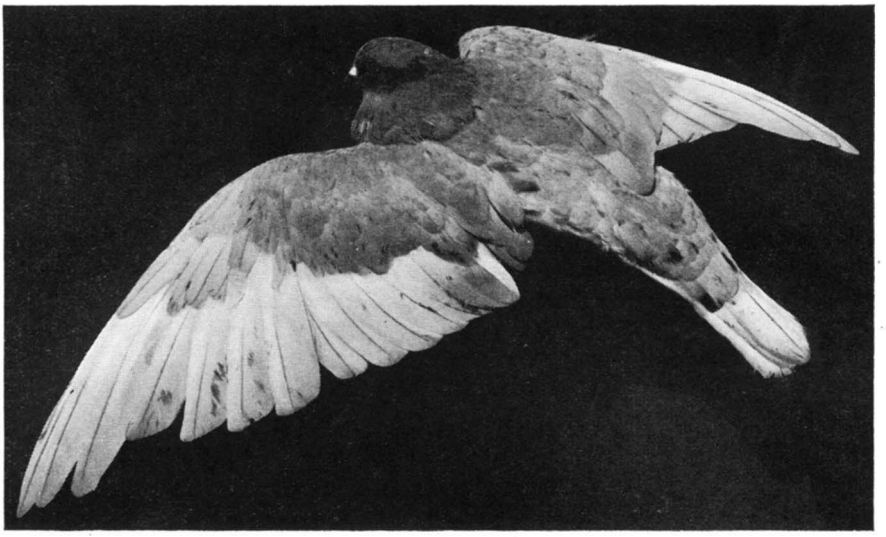

FIGURE 4.—Young ash-red male E284E (genotype B^A B) showing black flecks on individual feathers and a mutated patch of black feathers on the head. The latter presumably represent a single early mutation, making this bird, according to definition, a chimera.

pattern, genotypically $B\ d/B\ d;\ C^T/C$. The mother was a typical ash-red, of the constitution $B^A\ D/-;\ c/c$. The mosaic is ash-red in the checker pattern, with flecking, and is therefore genotypically $B\ ?/B^A\ D;\ C/c$. On the right wing there is a large patch of brownish feathers (enclosed by a black line, figure 5) whose color we have not been able to identify with certainty. The feathers of this area have a few flecks, which are black as in other parts of plumage. Furthermore, the feathers appear to be of the T pattern, rather than checker. Possibly the brownish color arose by recessive mutation of B^A to some new allele in the series, but a simultaneous mutation of C to C^T in another chromosome seems highly improbable. No very satisfactory explanation of this mosaic has been deduced. Polyspermy might perhaps be invoked but this would involve other complications. No breeding tests were made.

2. *Chimeras involving chocolate (b).*

A male mosaic showing irregular areas of *B* and *b* plumage coloration, together with a good deal of white spotting, is reported and figured by LEVI and HOLLANDER (1939), in the King breed. By pedigree, the bird's genotype was *Bb*. This case would be most simply explained by the loss of *B* in certain areas. This is the only case we have of the mutation of *B*.

FIGURE 5.—Right wing from male chimera 2721E. The island of unusual brownish feathers is outlined by a black ink line. The surrounding feathers are ash-red (B^A *B*) with a few black flecks; a single black fleck also occurs in a lesser secondary covert in the island.

3. *Chimeras involving dilution (d)*

Two mosaics of dilution and its normal allele are known, both in the Carneau breed. KEESLING (1924) described a specimen whose father was yellow (dilution with autosomal recessive red: *dd ee*) and whose mother was red (*D- ee*). It was "a cock of beautiful type and good size; has yellow head with small red spots, red breast and neck, yellow wings and back, red wing flights and tail. The markings are sharply defined." From this description it seems that a high degree of symmetry existed. The constitution of the bird is, by pedigree, *Dd*, so that the appearance of *d* areas is unexpected, but may be accounted for by mutation of *D* to *d*.

A quite similar case, though not symmetrically marked, was observed by one of us (W. F. H.) at the Middleton Squab Farm of Norristown, Pa. This bird also was a male, apparently breeding normally. There was a

moderate amount of white spotting in the plumage. Nothing further is known about this bird.

4. *Chimeras involving grizzle (G)*

A single grizzle mosaic female crossbred Homer-Carneau, *Gg* by pedigree, is reported by LEVI and HOLLANDER (1939). Most of the bird is grizzle but the factor appears to be lacking on most or all of the left wing. There was a moderate amount of white spotting.

5. *Chimeras involving recessive red (e)*

a. *With recessive mutant areas.* METZELAAR (1926, p. 34) mentioned a recessive red mosaic. In correspondence of January 24, 1925, he described it in detail: "Crossing a pure recessive red Carneau with a brown silver King, a female young was obtained which shows both parental colors in a piebald form. The flights are pure red, so is the neck; the rest of the body is brown-barred but for a few flecks in the areas with clumped pigment. These flecks are red; not clumped but spread pigment. They are irregular islands of spread pigment within the clumped brown region." The brown in this mating is sex-linked chocolate, and is to be expected in female offspring. The red, however, is unexpected, but may be accounted for by assuming mutation of *E* to *e* in a bird of the composition *b/-; E/e*. Here again is a case with (apparently) a fair symmetry. The bird has been mounted, and is at present in the Museum of Zoology at the University of Michigan.

Two other mosaics involving recessive red were given us in 1934 by DR. H. W. FELDMAN from the University of Michigan colony. These were male sibs, 2708.1 and 2708.2, F_2's from recessive red grizzle white-spotted Tippler×black-laced Blondinette. Both were black with some grizzling and white-spotting apparent; in one, scattered patches of red feathers occur over the neck, crop, and scapular regions; in the other, only a few scapulars are red. Although neither specimen was progeny-tested, their genotype was probably *Ee*, and the red areas may be accounted for simply by loss of the normal allele.

Two similar cases have appeared at this laboratory. A female, 1158H, whose skin in juvenile plumage was preserved, closely resembles the above males but no entirely red feathers are present; instead, there are large red segments of the interscapular feathers and elsewhere. She also had the typical juvenile reddish edging on most other feathers. She was heterozygous for *e*, as the father was recessive red and the mother black. The remaining case is a male, 2688H (figure 6), which has a rather large area of recessive red color, and also a moderate degree of white spotting. No breeding tests were made, but as both parents were heterozygous for *e*,

this specimen in all probability is also. Here again the appearance of red may be attributed to loss of the normal allele.

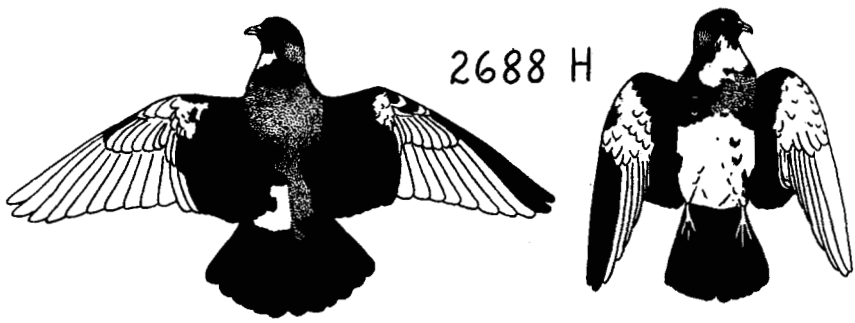

FIGURE 6.—Diagrams of male chimera 2688H. Black areas represent wild type allele (blue checker); stippled areas recessive red.

b. *With dominant feathers.* HORLACHER (1930, p. 341) mentions four recessive red specimens with one or more black feathers. Here we have

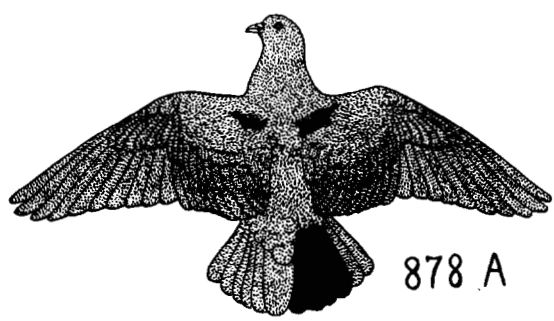

FIGURE 7.—Diagram of male chimera 878A. Stippling, recessive red; black, wild type allele (black).

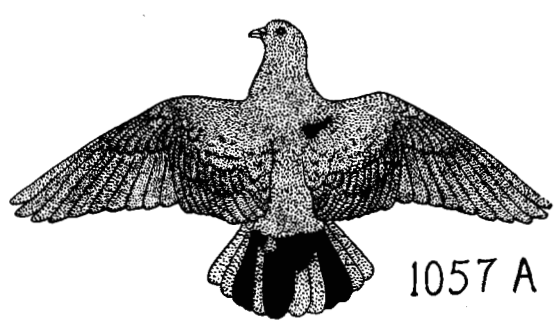

FIGURE 8.—Diagram of female chimera 1057A. Key same as in figure 7.

what appears to be a different sort of chimera from those treated above. Further information is available for two of these. One was a male, 1875C. Both parents were recessive red, and the first description of the specimen

(six years before HORLACHER'S description) made no mention of any black feathers; HORLACHER found a single black wing covert in this bird. The other mosaic, 1057A, was a female (figure 8). This specimen has been

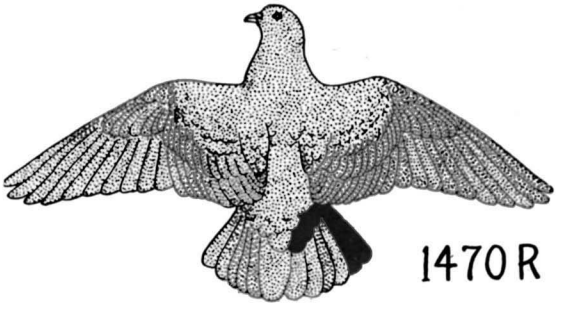

FIGURE 9.—Diagram of female chimera 1470R. Key same as in figure 7.

preserved; there are black feathers in the interscapular region and in the tail. No progeny test was made.

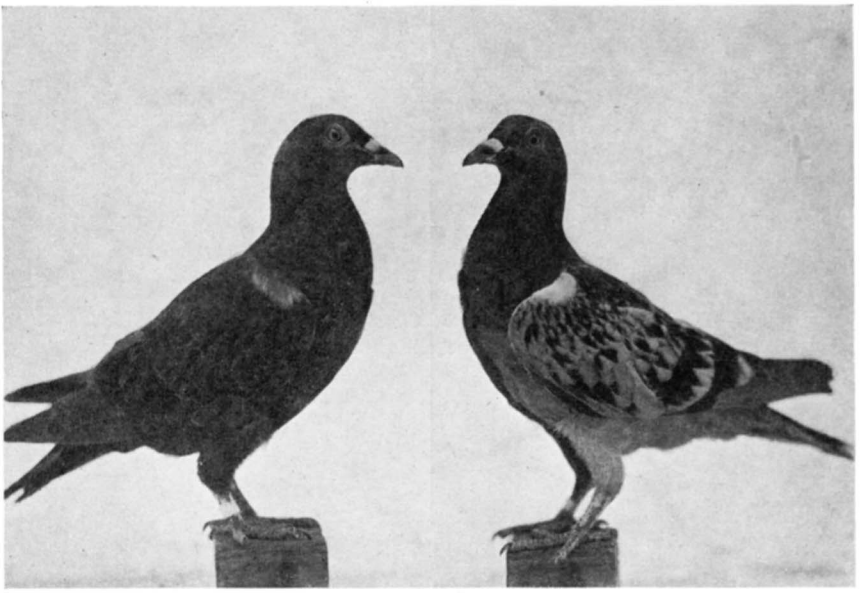

FIGURE 10.—Right and left aspects of "half-sider" *S* mosaic. Right side spread black, *S;* left side blue checker, *S*.

Two additional examples very similar to 1057A have been produced in the Wisconsin colony. One was a male, 878A (figure 7), and the other a female, 1470R (figure 9). These three mosaics are somewhat related to one another, the females being first cousins. Furthermore, a paternal uncle of the females, 925B, was described as recessive red with two black

as for example in gynandromorphism or in birds not genetically heterozygous, or where size or other changes are involved. Somatic segregation was suggested by ASMUNDSON (1938) to explain a chimeric turkey. Such an explanation could possibly apply to some of the pigeon chimeras. Dominant mutation or polyspermic effects have not been suggested in other birds; WRIGHT and EATON (1926) found a dominant somatic mutation in a guinea pig, and polyspermy has been found to occur in *Drosophila pseudoobscura* by CREW and LAMY (1939).

TABLE 2.

Summary of Chimeras.

		Mutant Area
1. Involving ash-red (B^A)		
LYELL (1887)	?	Simple recessive, B^A to B
2067B	♂	Simple recessive, B^A to B
2411A	♂	Simple recessive, B^A to B
LEVI AND HOLLANDER	♂	Simple recessive, B^A to B
LEVI AND HOLLANDER	♂	Simple recessive, B^A to B
E284E	♂	Simple recessive, B^A to B
2721E	♂	Complex; 2 factors involved
2. Involving chocolate (b)		
LEVI AND HOLLANDER	♂	Simple recessive, B to b
3. Involving dilution (d)		
KEESLING (1924)	♂	Simple recessive, D to d
HOLLANDER	♂	Simple recessive, D to d
4. Involving grizzle (G)		
LEVI AND HOLLANDER	♀	Simple recessive, G to g
5. Involving recessive red (e)		
(a) with recessive mutant areas		
METZELAAR (1926)	♀	Simple recessive, E to e
2708.1	♂	Simple recessive, E to e
2708.2	♂	Simple recessive, E to e
1158H	♀	Simple recessive, E to e
2688H	♂	Simple recessive, E to e
(b) with dominant feathers		
1875C	♂	Dominant, e to E
1057A	♀	Dominant, e to E
T49D	♂	Dominant, e to E
T55C	♂	Dominant, e to E
878A	♂	Dominant, e to E
1470R	♀	Dominant, e to E
925B	♂	Dominant, e to E
6. Involving S ("spread black")		
Chicago "half-sider"	?	Simple recessive, S to s
D935K	♀	Complex
7. Involving silky (L)		
SPRUIJT (1931)	♂	Dominant, l to L

For purposes of ready reference the chimeras described in this paper are summarized in table 2.

Simple recessive mutation, without the necessity of invoking chromoso-

mal loss, somatic segregation or other aberrancy, will explain the majority of the pigeon chimeras; mutation from recessive to dominant will explain all the others except 2721E and D935K, which are more complex. The fact that all the chimeras involving sex-linked characters are males may indicate that the explanation is really not so simple. Moreover, factors are involved which are ordinarily stable (*B* and *D*) as well as one (B^A) which, according to the flecking results, is regularly labile.

Polyspermy might possibly account for 2721E (group 1) and the reds with small black areas (group 5, b) but the results of polyspermy should be detectable in almost any mating of heterozygote with recessive homozygote and hence it cannot be very prevalent. Evidence for it has not been found in hundreds of suitable matings. Furthermore, there appeared to be some tendency for the black-on-red condition to occur in related birds. In this respect it differs from the other chimeras.

D935K (group 6) is the only known chimera in pigeons involving body structural characters and, in this case, feather structure as well. Apparently a single loss can account for this case only if *S* and white spotting, and presumably factors for structural development as well, are assumed to be on the same chromsome. There is no independent evidence to support this assumption. It would appear likely, however, that in this case a whole autosome, or more, was eliminated early in the development of the embryo.

The somatic nature of all the above pigeon chimeras is evidenced by failure of the condition to be transmitted in all cases when breeding tests were made. The tests were not always conclusive, it is true, but none gave any indication that the gonads were affected, even when the major portion of the body appeared "mutated," as in 2411A (see figure 3).

White spotting was present in at least 10 of the 26 chimeras described but probably as an incidental association. There are many essentially symmetrical patterns of white spotting in pigeons which breed more or less true. Although the developmental and hereditary basis for white spotting has not yet been successfully analyzed, it seems safe to conclude that it is not chimerism of the above sort because of its relatively predictable inheritance. Such marked asymmetry of the white as occurred in D935K is altogether unusual and is undoubtedly part of the mosaic pattern in this bird. This is the only specimen of this type that has come to our attention, but possibly the "scherzo" pigeons mentioned by Italian writers, as quoted by HOLMES (1921), are of this sort.

THE DEVELOPMENTAL BASIS OF CHIMERISM

No obvious law has been found governing the distribution of the differing areas in chimeras. In other species of birds CREW and MUNRO (1938)

found that sharp left-right asymmetry was common, but in pigeons we have found only one such case. Blotchy effects are most common and to a certain extent comparable cases may be cited in such forms as guinea pigs and Drosophila. In some cases the mutant areas are of more or less central location (for example, 2688H, group 5, and 2411A, group 1); in METZELAAR'S mosaic (group 5) they are distal. Only the nape of the neck and the interscapular region seem to be affected with any considerable regularity.

The interpretation of these heterogeneous arrangements in terms of development is not easy, especially as cell lineage in the definitive differentiation is obscure. Also, in considering color, the peculiar behavior of the pigment cells must be considered. Studies in this field, as summarized by DANFORTH (1939), indicate that in the embryo the "pigmentoblasts" have neural origin and migrate to their final positions. If the migration is irregular it may help to explain the irregularity of chimerism. At any rate, several discontinuous areas are presumably not to be interpreted as due to separate mutations. It would be difficult to explain why, considering the rarity of chimeras, a bird which had one mutation in its early development should have several simultaneous ones.

Unlike flecking, the color arrangements in chimeras have proved permanent through successive molts except in one case (reported by LEVI and HOLLANDER, group 1). In this case part of the mutated feathers in the tail changed in a reverse direction, back to the dominant color. We have no explanation of this case, but until more is known concerning color determination we may assume that it might harmonize with genetic interpretation.

In the *S* mosaics (group 6) clear striping effects, both of color and structure, were noted. The striping is more clean-cut than in flecking, and fits excellently the theory of axial cell-lineage in the feather. The *S* factor affects not pigment quality, but the arrangement or pattern of the pigment in the cell, and this may well be determined by the tissues in which the pigment cells reside rather than by the pigment cells themselves. The tissue cells having no tendency to wander in the growing feather, mosaicism would be expected to show up as sharply distinguished axial striping, and the same is true of a structural difference.

This study of chimeras in pigeons demonstrates that the factors involved are autonomous in development, that is, two different alleles are able to express themselves clearly in the same bird. Where the differing areas adjoin, there may be mixture, but no true blending of colors or structural differences. Because of this independence chimeras may have certain advantages over the transplantation technique for the study of tissue relationships, since no tissue antagonism is to be expected.

GENERAL DISCUSSION

Comparison of flecking and chimerism

Flecking, like most cases of chimerism in pigeons, consists of irregular areas of recessive color in heterozygous birds which otherwise are of the dominant phenotype. The flecks rarely involve whole feathers but consist of patches on the feather, often of very small size. This would seem to indicate that the mutations occur late in ontogeny, indeed within the individual feather germs. The mutant areas in chimeras, on the other hand, must have their origin in mutations which have occurred much earlier, possibly in some cases even as early as the first cleavage of the egg (figures 10 and 11). If it occurred still earlier, namely in the germinal tissue of the parent, a mutant *individual* might result, and it is possible that Feldman's original "faded" came about in this way (see p. 18).

Mutations at the early stages which produce chimeras are relatively rare, even with genes, such as B^4, which show high inconstancy in the feather follicles. It must be supposed, therefore, that there is some internal condition at this late stage which predisposes to a high frequency of mutation in certain labile genes. Furthermore, the tendency would appear to increase with age, as indicated by the greater amount of flecking in old birds. The mutant areas in chimeras, on the other hand, have not been observed to change with successive molts,[3] which would indicate again that the spot is determined by a single earlier mutation.

The difference between flecking and the larger areas of the chimera is usually clear enough but the actual size of the spot in a chimera, as shown in E284E, figure 4, may be smaller than a large single-feather "fleck" (fig. 1). The difference is that the former covers an area of several feathers, so it is obvious the causative change ("mutation") must have occurred before differentiation of the individual feathers. Furthermore, even when it is small, the chimera spot appears to be constant through successive molts.

Mutation or physiological control

That internal physiological conditions may normally determine whether or not a gene will exhibit its characteristic phenotypic expression is definitely shown in an ordinary Barred Plymouth Rock feather, for example, where the action of the gene is alternately expressed and suppressed. The work of LILLIE and his associates has demonstrated that not only barring, but other patterns as well may be controlled with considerable accuracy by changing physiological conditions. It is not to be supposed in either of these cases that there has been corresponding genetic change in the cells concerned. Probably the same is true for all regular patterns. It has been

[3] One "reverse" exception, group 1, p. 25.

suggested that in the case of piebald patterns the white areas may be regions of somatic genetic change, but there seems to be no direct evidence in favor of such an explanation. Why then may not the flecking and chimerism described in this paper be explained as direct physiological response to local internal environment rather than to localized genetic change? Probably the strongest argument at the present time for the latter interpretation is the great irregularity of occurrence of the flecks and spots and the way they fit into a logical genetic scheme. Furthermore, the initial changes, particularly in the case of the larger chimera spots, must have occurred well back of the point where they are expressed, and they are not synchronized as the responses to direct physiological conditions so commonly are. The "half-sider" described and illustrated (figures 10 and 11) could scarcely be referred to a general physiological differentiation in the first two blastomeres, which was so permanent as to continue through all subsequent somatic cell generations. Such a result would, however, naturally follow from a genetic mutation in one of these blastomeres. From the half-sider to the bird with ordinary flecking there seems to be a series, representing mutations at different stages of development, which make it logical to apply the same interpretation to the latter type of marking. This does not preclude the possibility that changes in internal environment, such as that accompanying ageing, may have an influence on the frequency of somatic genetic change.

SUMMARY

Three sex-linked color factors in the pigeon, all probably allelic, and each dominant to wild type, are found accompanied in heterozygous males and the hemizygous females by a sort of variegation which we have termed flecking. This is usually most marked in males and in aged birds. Large flecks tend to be repeated in successive feathers from the follicles; small flecks less noticeably so. Flecks of recessive color have also been observed in connection with an autosomal factor, grizzle, but no special study of this condition has been made.The assumption of frequent recessive somatic mutation seems to account adequately for flecking.

Chimeras are treated in seven main groups based on the factors involved. The areas in chimeras which are discordant with the regular phenotype of the bird are commonly larger than the flecks, involving considerable patches of feathers. They may even include as much as half the bird, as in a "half-sider" described. Like the flecks, they mostly occur in heterozygotes, and in the majority of cases are recessive. These cases can also be explained as the result of recessive somatic mutations which have occurred relatively early in the ontogenetic development.

A few of the chimeras are not susceptible of this simple explanation.

These appear to involve dominant mutation, or possibly polyspermy. One multiple chimera described involves at least two color effects and two structural effects (counting arrangement of pigment as structural). It is suggested that at least one autosome was lost in this case.

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