A CASE OF LEG-COLOUR ASYMMETRY IN THE FOWL.

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(With One Diagram.)

A PULLET derived from the mating Light Sussex ♀ and Rhode Island Red ♂, peculiar in that the left leg was yellow whilst the right was white, was sent to me by Capt. F. S. Pardoe, Horeham Road, Sussex, to whom I wish to express my thanks.

The hen was a typical F_1 ♀ out of this mating which involves the sex-linked characters silver and gold and the autosomal characters white and yellow epidermal pigmentation, save that the left leg exhibited the recessive epidermal pigmentation character yellow, although she herself must have been heterozygous for the dominant white.

Two possible and reasonable explanations of this peculiarity present themselves: (1) somatic chromosome elimination, (2) somatic gene mutation. Her Light Sussex mother was either homozygous, **WW**, or else heterozygous, **Ww**, for the white epidermal pigmentation character which she exhibited; her Rhode Island Red father was certainly homozygous for the allelomorphic recessive yellow, **ww**. Since this F_1 daughter had white epidermal pigmentation on at least one-half of her body, the fertilised egg in which she had her beginning must have had the constitution **Ww**.

If, during the first cleavage division of the fertilised egg which yields the two daughter cells from each of which one-half of the body is derived, through elimination, an autosome or part of an autosome carrying the gene for white epidermal pigmentation was lost, so that one of the resulting daughter cells was left only with the gene for yellow, then these two daughter cells and their descendants would differ in that one cell and the cells of one-half of the body would have the constitution **Ww**, whereas the other and its derivatives would have the constitution **w**. Thus, the epidermal pigmentation would be white on one side of the body, yellow on the other.

Alternately, both daughter cells might have the constitution **Ww**, but if in one of them the gene **W** mutated to become **w** the derivatives of the first would remain **Ww**, those of the second would become **ww**,

and so the epidermal pigmentation on one side of the body would be white, that on the other yellow.

On general grounds, and in the light of much experimentation, it is to be concluded that abnormalities in the distribution of the chromosomes (*e.g.* non-disjunction, elimination, fragmentation) occur more frequently than do specific alterations in the organisation of the chromatin in a particular locus (gene mutation). It is more probable therefore that aberration in chromosome distribution is the cause of the regional expression of a recessive character in this undoubted heterozygote.

It so happens that this is the seventh recorded case of lateral dissimilarity in the fowl. It will be profitable, therefore, to discuss the present case as one of a series which can easily be constructed.

Barrows (1914), confirmed by Biester (quoted by Knox), showed that the pigmentation of the legs of the bird is due to the presence of pigment either in one or both layers of the epidermis. There are two classes of pigments responsible for shank colour. Zooxanthin (yellow) in the chorium alone or in both layers of the epidermis yields yellow shanks. A lack of this pigment gives white shanks. Darker shades are due to the presence of melanin in the chorium, Malpighian and/or dermal cells. The nearer the surface the melanin is situated, the darker is the shank colour.

Hutt (1929 *a*), who showed that the size differences in cases Nos. 1 and 2 closely approximated the normal sex dimorphism in the appendicular skeleton of the fowl, discusses in another paper (1929 *b*) the first three cases and argues that if Lambert's case were due to non-disjunction, it would have been expected that in addition to the gene for white epidermal pigmentation, the chromosome lost from the cell giving rise to the yellow side would also carry other genes, and that the loss of some of them, particularly those affecting the size of the whole organism, or any part thereof, would also be expected to be shown as well as the absence of the dominant gene for white skin. He suggests that when there are lateral differences in both size and pigmentation, somatic non-disjunction is to be regarded as the cause, whereas when pigmentation differences alone are present, gene mutation is responsible. He chooses, therefore, to regard Lambert's case as an instance of gene mutation, the bird being either **Ww** or else **ww**, and either **W** mutating to become **w**, or **w** mutating to become **W**. But if one and the same cause can yield both kinds of differences, there is no reason for invoking the aid of two. Since all these cases cannot be interpreted as instances of gene mutation, it becomes necessary to enquire whether or not they can

	Author	Breed and genetic constitution	Sex	Lateral differences in gonad structure	Lateral differences in body size	Lateral differences in epidermal pigmentation	Suggested cause of abnormality
(1)	Macklin (1923)	Unknown. Barred Plymouth Rock?	?	Right testis Left ovotestis	Every bone on right larger than its fellow on the left side of body	Not recorded	Gynandromorph. Elimination of *X*-chromosome or fertilisation of binucleated ovum
(2)	Crew I (1928)	F_1 ♂ *ex* Light Sussex × Rhode Island Red mating. Heterozygous for white and yellow epidermal pigmentation	♂	—	Every bone on left larger than its fellow on the right side of the body	Left half of body white, right half of body yellow	Non-disjunction. Loss of autosome carrying genes for body side and white epidermal pigmentation
(3)	Lambert (1929)	Unknown	♀	—	—	Left half of body white, right half of body yellow	Non-disjunction or gene mutation
(4)	Knox I (1931)	F_1 *ex* Black Langshan × White Plymouth Rock mating	?	—	—	Left half of body white, right half of body yellow	Gene mutation or non disjunction
(5)	Knox II (1931)	F_1 *ex* White Wyandotte and Rhode Island Red mating	♀	—	—	Right shank green, left shank yellow	Gene mutation
(6)	Crew II (the present case)	F_1 ♀ *ex* Light Sussex and Rhode Island Red mating. Heterozygous for white colour	♀	—	—	Right shank white, left shank yellow	—
(7)	Knox III (1931)	Not recorded	Not recorded	—	—	Both shanks white, but one with a yellow streak	Gene mutation or non-disjunction

all be explained as the results of some aberration in chromosome distribution.

Macklin interpreted her case as an example of gynandromorphism due to the elimination of an *X*-chromosome in an *XX* zygote or to the fertilisation of a binucleated egg, though it is stated that the bird looked like a hen, with neck feathering suggestive of the male and tail feathers slightly longer than those of the normal hen. It had a cock's comb and exhibited male behaviour but was suspected of laying small eggs. It is to be noted that the presence of a testis on one side of the body and of ovarian tissue on the other does not in itself demand this explanation, for in the gynandromorph of *Drosophila*, with which this case is compared, the structure of the gonad is not necessarily in harmony with the remaining sex characters of the same side. If it is assumed that the condition of the gonads in this case is similar to that which is found in hens of normal proportions but suffering from pathological destruction of the left ovary, then gonadic abnormality can be left out of the discussion. In the light of the more recent work on experimental oophorectomy in the hen, there is no difficulty in assuming that testicular or testis-like material can develop following cessation of ovarian functioning, and that this can happen both in the hen of normal proportions and in the hen that is laterally asymmetrical. Certainly, this bird, its lateral asymmetry being disregarded, is very similar to the hen whose ovary is being destroyed by tumour growth. If this be granted, then cases 1 and 2 can be regarded as strictly comparable and an explanation common to both can be attempted.

In Macklin's case it is not necessary to postulate that it was an *X*-chromosome that was eliminated, for it is established that, in *Drosophila* at least, sex is not determined by the mere number of *X*-chromosomes but by the quantitative relation of sex and autosomes, so that the loss of an autosome, equally with the loss of an *X*-chromosome, can disturb the balance and result in sex abnormality. Furthermore, the fact that the size differences on the two sides of the body approximated those normally distinguishing male and female is in itself no proof that one side was male and the other female. We know that the loss of a *IV*-chromosome in *Drosophila* is attended by a diminution in body size. We do not know whether the bones on the larger side of Macklin's bird were equal in size to those of the male of some particular breed and those on the smaller side were equal to those of the hen of the same breed; for we can only assume from the photograph that this bird was a Barred Plymouth Rock. We know nothing of its origin or pedigree. In case 2

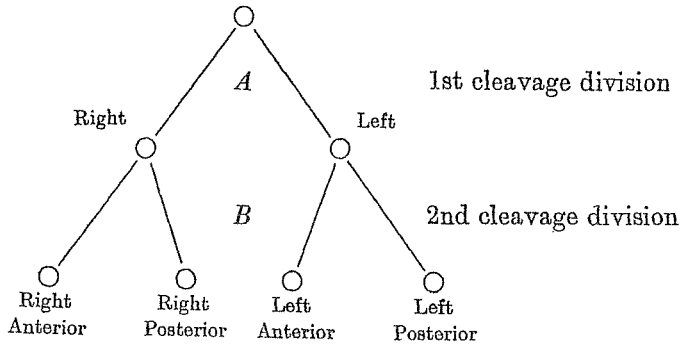
there was no indication that the two sides of the body differed in respect of sexuality: they certainly differed in respect of size, and the differences in size closely approximated the normal size differences in the appendicular skeleton which distinguish the sexes. The loss of an autosome carrying the gene for white epidermal pigment is a sufficient explanation of this case, if it is assumed that this autosome also carried genes for body size. The same explanation can be satisfactorily applied in case 1.

Under certain conditions, such lateral differences in size, due, it is suggested, to the loss of an autosome, could lead to the development of abnormalities in the distribution of sex-controlled as well as of autosomal characters. If the bird is a female, if the larger side of her body has a higher growth rate, and if ovarian deficiency is present, then, as is shown by the work of Lillie (1931) and his colleagues, it is to be expected that the plumage of the larger side of the body will be male whilst that of the smaller side will be female. For an explanation of gynandromorph conditions in the plumage and of the co-existence of testicular and ovarian tissues in one and the same bird, we must turn to the recently disclosed facts of avian sex physiology, but for an explanation of lateral asymmetry in size and in the distribution of non-sex-controlled autosomal characters, we must turn not to sex physiology but to genetics.

The remaining cases can be explained as instances of abnormality in chromosome distribution if it is assumed that not the whole but only a part of the autosome carrying the gene for white is lost (in case 5, the gene for green), and that the differences between cases 3 and 4 (complete half of body), 5 and 6 (shank) and 7 (part of shank) are reflections of differences in time during development at which the loss of part of this autosome occurred, the earlier it occurs the greater being the distribution of the recessive character.

This suggestion is in harmony with the information we possess relating to the early cleavage divisions of the hen's egg. There are only two cleavage divisions which are regular and invariable: these are the first two and these are always at right angles to each other and split the blastodisc into four blastomeres. The blastodisc lies on top of, and is continuous with, the main mass of the yolk, and so there can be no change in the relative orientation of the blastodisc and yolk mass. The round unfertilised egg entering the oviduct becomes compressed into an oval form, and as it passes down the oviduct rotates about its long axis, being at the same time coated with albumen, the inner layers of which themselves become spirally twisted to form the chalazae. As albumen and shell are deposited, the pressure on the yolk is released and the

yolk resumes its rounded shape, but its original orientation is maintained by the chalazae. The first cleavage division takes place in the upper part of the oviduct, that is to say, whilst the egg has an oval form. It is to be assumed that the first cleavage spindle is formed along the long axis of the oval blastodisc. If this be so then the first two blastomeres will be formed by a cleavage at right angles to the long axis of the egg. Duval (quoted by Lillie, p. 63) states that in 98 per cent. of cases this long axis is at right angles to the long axis of the embryo. If each lateral half of the chick is produced from one of the first two blastomeres, it follows that in 98 per cent. of chicks these two blastomeres represent the right and left halves of the embryo. The second division results in the production by each of these first two blastomeres of two others, one of which yields the anterior half of one side of the body, the other, the posterior half of the same side, so:



Chromosome elimination at *A* would yield dissimilarities involving the whole of the two sides of the body, *e.g.* size and skin colour or, if only a part of the chromosome were lost, skin colour alone. Chromosome elimination at *B* would yield dissimilarities involving only the anterior or posterior regions of the two sides. The seven cases can thus be explained as follows:

Case 1. Loss of whole autosome at *A*.
,, 2. ,, ,, *A*.
,, 3. ,, part of this autosome at *A*.
,, 4. ,, ,, ,, *A*.
,, 5. ,, ,, ,, *B*.
,, 6. ,, ,, ,, *B*.
,, 7. ,, ,, ,, later still.

Case 6, the hen recorded in this paper, was mated with a yellow-legged

Rhode Island Red cock, and produced twenty-six chicks, eleven of which had white legs whilst the leg colour of the rest was yellow. She bred true to her **Ww** constitution. Her single ovary on the left side of the body was not affected by the disturbance which resulted in the exhibition of the recessive yellow character of the leg of this side.

SUMMARY.

An F_1 hen out of the sex-linked mating Light Sussex ♀ × Rhode Island Red ♂ had legs of different colours, the right showing the dominant white, the left the allelomorphic recessive yellow. It is suggested that this regional expression of a recessive in a heterozygote is due to the loss of a part of an autosome during the second cleavage division.

Six other somewhat similar cases previously recorded and described as instances of either non-disjunction or gene mutation are discussed. The suggestion is hazarded that all of them can be regarded as examples of the loss of a whole autosome or of a part thereof.

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