CREST AND HERNIA IN FOWLS DUE TO A SINGLE GENE WITHOUT DOMINANCE

SOME five years ago I undertook an experiment with poultry involving the introduction of a number of genetic factors, regarded as dominants on the evidence of the crosses among domestic breeds, into a stock of wild jungle fowl, *Gallus gallus.* The experiment was designed to test a crucial point in dominance theory, for if the supposed dominants had become so through human selection during the process of domestication this fact could be demonstrated by showing that in wild stocks, which had not been subjected to this selection, dominance was absent, and the heterozygote was clearly intermediate between the two homozygous types. Until the present year, therefore, heterozygotes, each manifesting one of the group of factors to be tested, had been mated back to the wild stock.

This year, 1934, among other tests, heterozygous crested of the fifth generation were interbred and, at hatching, it was immediately seen that about a quarter of the offspring manifested cerebral hernia. The obvious inference that cerebral hernia is in itself a homozygous manifestation of the gene for crested can only be demonstrated with certainty by breeding from the chicks with hernia, if any survive. The conclusion is, however, supported by the data on hernia published by the earlier poultry geneticists, although the genie identity with crest seems to have been overlooked.

A number of crosses involving the crested varieties, Polish, Houdan and Silky, were reported by Davenport[1] in 1906. Only two of these seem, however, to have been carried as far as the second generation,

[1] C. B. Davenport, "Inheritance in Poultry," *Pub. Carnegie Inst. of Washington,* No. 52, 1906.

and the data given are difficult to follow, owing to inconsistencies in the numbers tabulated. His conclusions are: (1) That "cerebral hernia is inherited in Mendelian fashion with plain head dominant. Nevertheless, many of the plain head hybrids have the frontal eminence abnormally high—dominance is imperfect." (2) The crest is independent of the cerebral hernia." (3) "Crest is inherited in Mendelian proportions, and is dominant over crestless head. Even when the Silky is crossed with *Gallus bankiva* its crest is dominant. In this case the new characteristic, a positive variant, dominates over the ancient one; but the crest is diminished in the first generation; dominance is imperfect."

It is difficult to understand Davenport's statement that the crest is independent of cerebral hernia. For, of the two cases (Polish x Minorca) in which a second generation was bred, and classified simultaneously for the presence of crest and hernia, there appear in the table (p. 16) for the first cross: Normal, 21; crest without hernia, 34; hernia without crest, 3; crest and hernia, 12; total, 70.

Of the crested birds recorded over a quarter show hernia, whereas of the uncrested the proportion recorded is only one eighth. Moreover, it is evident that there is a deficiency in the numbers of crested recorded, the expectation out of 70 being 52.5. This discrepancy Davenport ascribes to misclassification of embryos, giving for comparison the numbers of crested and uncrested obtained from 52 chicks hatched. Since, therefore, 18 of the birds classified in his table must have died in the shell, at a stage when the crested character may be indistinguishable, the 3 birds said to have shown hernia without crest are easily explained. Hernia is immediately recognizable in the chick; I should, therefore, have no hesitation in interpreting Davenport's record of these broods as 21 normal, 34 heterozygous, 15 homozygous mutant, thus conforming entirely to a 1 : 2 :1 ratio. These

numbers are, however, apparently affected by copying errors, for in a later table Davenport gives 16 with hernia out of 70; while in an earlier table for crest he shows only 23 out of 75, instead of 24 out of 70 lacking crest.

Hernia also appeared in F_2 from Houdan (crossed with White Leghorn). In this case 11 with hernia appeared out of 45—these totals presumably including unhatched, as well as hatched chicks. Crest was classified for only 19 individuals, of which 6 were uncrested. The 13 crested individuals evidently included all those with hernia, which survived this stage, for it is stated that "hernia is never found dissociated from the crest." The number of the crested birds showing hernia is, however, not given. Both of Davenport's F_2 generations thus accord with the view that hernia in his material was manifested where the gene for crest was homozygous.

Punnett[2] comments on Davenport's statement as follows: "Davenport states in the same paper that hernia is never found dissociated from the crest, but as he himself records 3 cases of uncrested birds with hernia, his statement would seem to require modification." The statement quoted from Davenport, however, evidently only refers to his second cross (White Leghorn x Houdan), in the summary to which it occurs, and not to the first cross (Black Minorca x Polish). It is his general conclusion (p. 65) that "the crest is independent of cerebral hernia" that is doubtfully consistent with the experimental data he reports.

In connection with Davenport's table of the cross with Polish, it is interesting that if hernia is taken as diagnostic for homozygotes of the gene for crest, this gene is evidently linked with another, also showing lack of dominance, which gives when heterozygous a split comb, and when homozygous the obliterated comb of the Polish breed.

The 9 genotypes thus classifiable appear in Daven-

[2] "Heredity in Poultry," p. 103.

port's table with the frequencies shown in Table 1:

TABLE 1

	Normal	Crest	Hernia	Total
Single comb	12(9.004)	8 (7.097)	2(1.399)	22
Split comb	8(7.097)	17(20.806)	4(7.097)	29
No comb	1(1.399)	9 (7.097)	9(9.004)	19

The totals for the comb character are not altogether convincing, and suggest that some heterozygotes have been classified as having the single comb. Any such misclassification would tend to increase the apparent recombination frequency, which, as judged from the data, is between 28 per cent. and 29 per cent. The expectations in the table are for 28.27 per cent.

The earlier writers, such as Hagenbach and Darwin, took the connection between Crest and Hernia for granted. Among recent geneticists Dunn and Landauer[3] consider the point and report that all herniated fowls reared to maturity have developed a pronounced crest. They consider, however, that the characters are separable on the strength of one instance in which an uncrested fowl was believed to transmit hernia. The case would, however, be convincing only if uncrested birds showing hernia had been reared from the progeny.

In a recent letter, Dr. F. B. Hutt writes, "I have decided the same as you, that there is no difference between the genes," although in Hutt's material hernia seems not to be easily classified. Probably the largest factor in preventing, hitherto, recognition of the simple relation between these characters has been the genetic suppression of the hernia in the Silky breed used in many of the experiments. Back-crossing to the wild fowl is evidently capable after some generations of eliminating the cause of this suppression.

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[3] *Jour. Genetics, 22:* 95-101, 1930.