DISCUSSION

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 genic 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, 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 × 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 × Houdan), in the summary to which it occurs, and not to the first cross (Black Minorca × 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

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

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

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 Davenport'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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THE NEWFOUNDLAND SEAL FISHERY

IN SCIENCE for August 24, Dr. C. Hart Merriam called attention to an announcement by the secretary of the Society for the Preservation of the Fauna of the Empire that the Newfoundland sealing industry is steadily declining and recommending that a sanctuary be provided for the protection of the seals. Dr. Merriam very properly pointed out that these ocean-dwelling seals breed only on ice floes and that a land sanctuary would not be possible.

Having before me the official records of the catch of the Newfoundland seal fishery for over a hundred years, it does not appear that the fishery has declined to a serious extent. During the period from 1860 to 1930 the average annual catch was 196,019. Due to unfavorable weather conditions in 1931 and 1932 there was a falling off, but in 1934, 223,708 seals were taken.

Prior to the middle of the nineteenth century large numbers of sailing vessels engaged in this fishery. At one time in the fifties, there were 400, it is said. The annual catch *occasionally* exceeded half a million seals. Later, when the sailing vessels were replaced by steamers, the hunting season was officially shortened with a view to conservation.

Sealing operations are now permitted only between March 10 and April 15. Owing to the present low price of seal skins and oil only nine steamers were employed in making the large catch of 1934—223,708 seals.

This long established seal fishery is unique in that it is based on the taking of *young* seals only, the number of adults captured being negligible. Adults take to the water at once, upon the approach of the hunters, the extremely fat, nursing young being unable to leave the ice floes on which they are born.

The catch has always been made on ice floes not far from Newfoundland. Doubtless both Harp and Hood seals, the two species on which the fishery is based, bring forth their young on ice fields more remote and more difficult to penetrate by vessels. It is evident that the survival of great numbers of breeding seals has hitherto sufficed for the notably prolonged maintenance of the fishery. I have records of catches dating back to 1795. With no heavier killing than that of the past decade the fishery may last indefinitely.

We have for some time urged that the control of sea lions on the Pacific coast be brought about by commercial use of the nursing young before they are old enough to take to the water, rather than by wanton destruction of breeding sea lions that sink when shot. Young sea lions represent a resource in usable leather and oil that has hitherto been wasted, the skins of adults not being utilized.

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OVERWINTERING OF APLANOBACTER STEWARTI

ATTEMPTS to solve the problems of dissemination and overwintering of *Aplanobacter stewarti* (E. F. Smith) McCul., the cause of bacterial wilt of corn, were for many years concerned chiefly with soil and seed transmission. Recently investigators have turned

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