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Dominance in Poultry

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VI—Dominance in Poultry

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1—Introduction

The following is a preliminary report of an experiment which the author has been conducting since 1929, intended to throw light on the nature of the dominance observed in certain well-marked breed characteristics in domestic poultry. The experiment is not yet completed. Consequently, while there are some results which scarcely admit of doubt, there are others on which I should not choose at present to speak with confidence. The resolution of questions at present doubtful, as well as the full confirmation of conclusions which may already fairly be drawn, will require, and should be possible in, two further years' experimentation. The present paper is therefore confined to those factors for which the situation seems to be already elucidated. I propose to defer discussion of the four factors responsible for feathered feet, pile or "dominant white" plumage, rose comb, and black internal pigment, and to give detailed data at present only for the three factors responsible for crest, polydactyly, and barred plumage.

2—Origin of the Experiment

The experiment originated in the observation that, while in the majority of domesticated plants and animals the characters of the domesticated races, when they had an unifactorial basis, were generally recessive to those of wild individuals, the genetic literature of poultry showed a large number of the characters of domestic breeds described as dominants. The contrast becomes more forcible when the experience obtained with wild species, brought into culture as suitable genetic material, is also taken into consideration. In *Gammarus chevreuxi*, for example, at least 16 mutations have been reported, as Mr. E. B. Ford informs me, all completely recessive to their wild allelomorphs. In *Drosophila melanogaster* (Fisher, 1928) out of 221 viable mutations producing visible effects, 208, or 16 out of 17, are reported as completely recessive, while in the remaining 13, though these are conventionally termed "dominants," dominance is really absent or, at most, incomplete; for the heterozygote, while it is distinguishable from the non-mutant homozygote, is also distinguishable, sometimes by a more pronounced difference, from the mutant homozygote, and is always clearly intermediate between these two forms. The

apparent peculiarity of domestic poultry was forcibly stated by PUNNETT (1923, p. 35), in terms of the theory that a recessive mutation represented a loss of germinal substance, in the assertion that for poultry something new had been added, possibly by means of hybridization with other species.

In 1928 the author put forward a theory to the effect that harmful mutations, not at first wholly recessive, which continued for a sufficiently long period, even with minute mutation rates, in a wild population would tend in the course of time to modify the species in which they occurred, by the cumulative selection of modifying factors, so as to become recessive or, in other words, to exert no appreciable effect when heterozygous. That certain mutations must be of very ancient occurrence is indicated by mutations, which can be proved to be homologous, occurring in nearly related species ; or more strikingly, perhaps, though the homology here cannot be experimentally proved, by the occurrence of such a mutation as albinism in different orders of mammals. Therefore it is a fair presumption that this harmful mutation has persisted in occurring throughout the history of the entire class, if not for a longer period, and has been as persistently eliminated by its injurious effects on the survival and reproduction of the affected individuals. At the time the theory of the modification of dominance was first put forward, few special facts could be adduced in its support, beyond the calculation that the magnitude of the selective forces in operation was of an order likely to have been effective. Several remarkable facts were, however, soon adduced by a number of different geneticists which strongly confirmed the view that dominance had in special cases, most notably, perhaps, in the polymorphic species, been modified so as to allow the heterozygote to resemble the more advantageous of the two homozygotes, and so to give dominance to the characters of the latter. This evidence has been summarized elsewhere (FISHER, 1930, 1931).

The existence of a theory of the evolution of dominance naturally gave special interest to the apparent exception exhibited by poultry. In 1929 the author put forward the tentative suggestion that the contrast was not so absolute as had been thought. The facts that, in addition to reputed dominants, poultry also exhibited, like other species, complete recessives, such as silky feathers and recessive white coloration ; and also lethals having a visible effect when heterozygous, such as " dumpy " ; and that the reputed dominants were definite breed characteristics belonging, for the most part or always, to long-established varieties, made it seem not improbable that human selection had played a part in producing the situations discovered by poultry geneticists. One circumstance in particular suggested a reason why in this species, rather than in others, human selection should have had this effect.

Man has, from the earliest times, valued and esteemed striking peculiarites in his domestic animals, and they have often been regarded with religious veneration, as, for example, the sacred white elephant, and the Apis bull, which was recognized, we are told, by peculiar markings. I am informed that in Malaya at the present day fowls exhibiting the mutation " frizzled," in which the feathers are curled

outwards, are esteemed to be particularly lucky in averting the effects of the " evil eye."

Whether valued, however, merely as ornaments and novelties, or for the superstitious awe which they were capable of exciting in species in which the matings were under control, recessive mutations, since from the first they will breed true and are in most species of by far the most frequent occurrence, would naturally supply the majority of the peculiarites of domestic breeds. If we consider, however, the early stages of the domestication of the jungle fowl in Burma, for example, or Assam, it is clear that the jungle tribes could not, and probably would not try to, prevent their flocks from being covered by wild birds. Very frequently, therefore, in such conditions the chicks reared would be of half wild origin, and (in such cases) any recessive peculiarities which might be favoured would be lost. If, however, a peculiarity were partially dominant it would reappear at least in half the chicks of the broods sired by jungle cocks. In these circumstances, therefore, a semi-dominant peculiarity would have a much better chance than a recessive one of becoming a characteristic of a domesticated strain. Moreover, if any valued peculiarity occurred in some, but not all, of the chicks in any year, it is certain that these would often have been regarded with attention, and the greatest care be given to preserve those in which the peculiarity was most strongly developed ; and this would constitute a selection of those heterozygotes in which the mutant was most dominant or least recessive. It seemed thus not improbable that human selection was responsible both for the high proportion of dominants or semi-dominants among the characteristics of domestic breeds, and for the relatively complete degree in which dominance seemed to be manifested among them.

On one essential point these views are capable of verification or refutation by direct experiment ; for, if human selection is in any way responsible for producing in the domestic breeds an exceptional genetic situation not previously existing in the wild jungle fowl, it should be possible, by introducing the mutant genes concerned, singly, into stocks of wild fowls, to ascertain the former reaction of the wild species to the mutants concerned. Such an experiment, if successfully carried out, should certainly be capable of deciding the main question of whether, in the wild jungle fowl, the reaction to the mutant genes tested was essentially similar to the reactions found in non-domesticated genetic material of other species, or whether it differed in any striking way from them. This question seemed eminently worthy of resolution. It must not be expected, however, that any experimental results should be capable of establishing as certainly true the conjectural details of the process by which, in early times, human selection, as has been suggested above, may be thought to have brought about its effects. Such conjectures, which are more anthropological than genetical in their nature, must continue to be judged on their inherent probability in the light of what is known of the habits and ideas of primitive peoples.

Moreover, if, as I venture to think the experiment has already indicated, the reaction of the wild jungle fowl to the reputed dominants is essentially similar

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to that found in *Drosophila* with those mutations in which dominance is absent, it does not rigorously follow in every particular case that the degree of dominance found in the domestic breeds has been modified during the period of domestication. The experiment I have carried out is competent only to reveal the reaction of a stock of wild origin. Whether this differs essentially from the reaction in domesticated breeds can be judged only when we know what reliance to place on the care with which these reactions have been studied and reported. It cannot, unfortunately, be doubted that statements respecting dominance are, in general, made with less care, and on the basis of less satisfactory evidence, than other statements in the genetic literature. It would, in the literature of poultry genetics, be easy to cite a number of examples, where factors have been first described as dominant, without reservation, which in reality were lethal, when homozygous, as in "dumpy," or in which the homozygote, as in "frizzled" and "spangled," is easily distinguishable from the heterozygote by the more extreme manifestation of the mutant character. The term, in fact, has often been used on the strength of experiments in which no single homozygote has been isolated for comparison with the heterozygotes. The more critical genetic literature of recent years has, indeed, shown that many of the reputed dominants of poultry should not, in any precise sense, have been so described, but that they are cases in which dominance is as clearly absent as in the oft-cited example of the Andalusian blue.

In the second place, where experimenters have been careful to enquire, by comparison with both homozygotes, whether the heterozygote really resembles one of these, or is intermediate, the comparison has been often made on material highly unsuited to this purpose. In such a comparison it is clearly desirable that the heterozygote and the two homozygotes described should differ as little as possible, and that not systematically, in other genetic factors affecting the character examined; but the vast majority of the statements with which we are concerned were based on a comparison of birds of different breeds, differing in a multitude of genetic factors, and with the first and second generations obtained by crossing them. It is clear that the parental birds, and their offspring of the first generation, will differ systematically in every factor in which the breeds differ; so that if, on crossing a breed with a large crest with a crestless breed, the first generation is crested, but with a smaller crest, the assertion that dominance is incomplete rests on the highly improbable assumption that the size of the crest is not influenced by any of the other factors in which the breeds may differ. The technique of introducing a new gene into an existing breed by repeated or alternate back-crossing, and so ascertaining its real effects viewed against a relatively uniform genetic background, seems not to have been practised by poultry geneticists, though it has been used (Timoféeff-Ressovsky, 1934) with success in studies of viability and fertility in *Drosophila*.

There are, however, as we shall see, demonstrable cases in poultry, where the reaction of the domesticated variety is certainly different from that of the wild species, and, for the rest, in considering, not individual cases, but the group of

reputed dominants as a whole, it would seem highly improbable that the frequency with which factors have been accepted as dominants was due only to the unsuitable material, and the uncritical use of terms on which their reputation often rests. So far as these factors are concerned, a bias towards calling the mutant genes recessive might as well have been evident as a bias towards calling them dominant.

The uncertainty of the status of the evidence on which different mutations were regarded as dominant presented an initial difficulty in the choice of what factors to work with. The author sought the advice of geneticists who had worked with poultry, and compiled a list of those that were regarded as good or reliable dominants. These were all included in the experiment, though one for black plumage was lost before a line for this character, free from others that might mask it, could be established. It may be mentioned that a factor having a similar effect, but evidently more recessive than dominant has reappeared in the last generation. This may or may not be the "dominant black" of the varietal crosses. With respect to several of the remaining seven factors, I have learnt, since the experiment was commenced, that in the opinion of other geneticists their claim to be regarded as good dominants in varietal crosses is not so good as had been previously represented. In no case, however, where it is claimed that the heterozygote can be distinguished from the homozygous mutant, does it appear that use has been made of this fact in linkage studies, for which such a discrimination, if it could be relied on, would be most valuable. Such a conflict of evidence and opinion is only what is to be expected if the dominance manifested by domesticated breeds had been brought about in their ancestors by human selection, for we should not expect to find, in a breed in which a certain peculiarity is absent, those modifying factors which would favour its dominance, unless this breed had been derived from one exhibiting the characteristic. Since the subject has been regarded as controversial, and since with increasing information the ground of controversy may be thought to change, I may quote a statement of the point of view from which the experiment was initiated from a book (Fisher, 1930, *a*) published in 1930.

> "It may be mentioned that my inference concerning the modification of dominance in mutant factors in the fowl, is open to the crucial test of introducing one or more of these dominants into a genuinely wild strain of jungle fowl. If my inference is correct, the mutant would then be found to be clearly intermediate, and not either completely dominant or completely recessive. Through the kindness of the Zoological Society of London, and the generosity of Mr. Spedan Lewis, it has been possible to start this experiment; the result cannot, of course, be known for several years."

I may add that I have at no time suggested that the result of back-crossing would be to make the mutant completely recessive. On the contrary, it was an essential part of my view, as first stated in 1929, that the lack of complete recessiveness in crosses with the wild stock was an important factor in enabling a mutant to become a characteristic of one of the anciently established breeds.

3—Method of the Experiment

The experiment as planned consisted in introducing various genes reputed to be dominants into a stock of wild jungle fowl. Repeated back-crossing was practised in order to diminish, as rapidly as possible, the proportion of the ancestry contributed by the domesticated breed, from which the mutant gene was obtained. Where it is desired to introduce a recessive gene into a stock in which it is not already available, it is necessary to employ alternate generations in back-crossing and inbreeding respectively. In the generations devoted to inbreeding, the proportionate contributions in the ancestry of the two original strains are unchanged. The purpose of such inbreeding is merely to ensure the inclusion in future generations of the recessive gene, by recovering recognizable homozygotes from heterozygous parents. Such homozygotes may then be back-crossed, yielding known heterozygotes, with a contribution to their ancestry of the strain from which the mutation was derived only half as great as before.

When dominance is absent, or when the gene to be introduced is dominant, the heterozygotes are distinguishable from the non-mutant homozygotes, and it is possible, in each generation after the first, to breed heterozygotes back to the non-mutant strain; obtaining in each generation approximately half heterozygotes, which are available for future breeding, and half non-mutants, which are discarded. The offspring of the first generation will then be all heterozygotes, owing half their ancestry to the strain from which the mutant was derived, and in subsequent generations a half of the progeny will be heterozygotes, owing to this strain $\frac{1}{4}$, $\frac{1}{8}$, $\frac{1}{16}$, $\frac{1}{32}$nd of their ancestry. Apart from the selected gene, and other genes linked with it, one part in 32 of the germ-plasm of the progeny of the fifth generation may, on these grounds, be expected to be of domesticated, and 31 parts in 32 to be of wild origin. These proportions will remain unchanged on inbreeding heterozygotes of the fifth generation, to obtain birds of the homozygous mutant type. It may, therefore, be expected that any observable differences between the heterozygotes and homozygotes will appear in the sixth generation, sufficiently little obscured by the interaction of other factors. It will be noticed, however, that the previous generations provide experience only of the appearance of the heterozygotes, and throw no light on what further modifications to expect in the manifestation of the homozygotes. These must be recognized, in the sixth generation, and pending further tests, merely by the appearance of birds showing a more extreme development of the mutant character than has been found in previous generations.

The possibility that such novelties as appear may be due partly to other factors should be considered. A gene unlinked to the one selected will, as has been seen, occur as a heterozygote in the fourth generation in one case out of eight. In one case out of 32 it will be transmitted to both of the two parents of the sixth generation, and in one case out of 16 to one of them. If transmitted to both, a quarter of the young will be homozygous and a half heterozygous for the secondary gene. If transmitted to only one of them there will be no homozygotes, but half the young

will be heterozygous. Consequently, there will be one chance in 128 for a bird of the sixth generation to be homozygous for a gene derived from the domesticated stock, unlinked with one selected. In six other cases out of 128, it will be heterozygous for such a gene, but then it will exhibit no character which has not appeared regularly in its ancestry, and probably also in many collaterals. The chance of a new character being due to anything other than homozygosis of the gene selected is only one in 128, and such cases, if they occurred, would only be deceptive if they affected the same character as the primary gene.

With respect to genes lying in the same chromosome as the one selected, and linked with it in inheritance, the probability of inclusion is necessarily higher, and would become extremely high if the linkage were very close. The chance rises to an even one (0·5) if the recombination fraction is about 10%, so that approximately we may think of the sixth generation as containing, in addition to one thirty-second part of the rest of its nuclear material of domesticated origin, a length of chromosome of about 10 units on either side of the gene selected. The linkages so far found in the fowl have on the whole given rather high recombination fractions, so that the tract included by reason of linkage is probably only a small part of the chromosome concerned.

Unfortunately, there are other considerations which may be practically important in determining the proportion of domesticated germ-plasm. Of these, two may be considered:—

(1) the purity of the wild stock employed, and
(2) the action of selection during the experiment.

With respect to the wild stock, it may be said that it was possible to use menagerie stock of good repute, which there is no reason to think was not derived exclusively from wild jungle fowl. On the other hand, in both cases the history of the material was unknown, and the possibility of some contamination with domesticated breeds could not be excluded. The only breeds, however, which could with any probability have been confused with the wild fowl in an earlier generation are those, such as the black-red game breeds, whose origin from the wild is most probably distinct from those of the fancy breeds, and contamination with which is therefore least to be feared. Selection during the experiment has probably been a far more potent factor. During the earlier generations a pen of wild fowl was invariably bred alongside the experimental lines. These regularly were less fertile and suffered a heavier chick mortality than those of partially domesticated origin, and these difficulties increased generally in the experimental lines with the increasing proportion of wild blood. It is, consequently, highly probable that the birds which survived well and were fertile, and consequently contributed most to the experimental lines, were those which contained the largest proportion of germ-plasm of domesticated origin. Sometimes also the selective process by which the lines were propagated may have contributed to the same result. Manifestation of the mutant character in the heterozygotes was extremely variable. With polydactyly

it is certain that the gene was occasionally carried by birds which were entirely normal in structure, for the character appeared from such birds in the line bred for feathered feet. In the latter factor also all external signs of feathering may disappear as the chicks grow up, so that it is possible with both of these factors that heterozygotes were sometimes discarded as normals. Such a selection would tend to enhance the manifestation of the mutant character by conserving subsidiary factors, which may have been accumulated because of this effect in the domesticated races.

The effect of involuntary selection is thus to diminish the probability of the experiment demonstrating a clear difference between the reactions of wild and domesticated strains, first by retarding the replacement of germ-plasm of domesticated origin by that of wild origin, and, secondly, by retaining an undue amount of the modificatory variance by which otherwise decisive results might be masked. The effects of five generations of back-crossing may not be so great or so clear as if the mutant gene alone could be examined in a purely wild strain. They are probably nearly as effective as any longer experiment conducted in the English climate without elaborate artificial control of environment. If substantial modification has taken place, it will be sufficient if the experimental procedure is competent to reveal, not the whole, but a large part of the effect.

The experiment is designed to bring to light only large and obvious differences between the genotypes. It is sometimes asserted, and it may be true, that if sufficiently large numbers could be examined, all genotypes would be found to be distinguishable, and all dominance to be incomplete. In this research it has not been possible to work on such a biometrical scale. Only small numbers of the homozygotes could be bred, and unless the differences they show are large and consistent they cannot be recognized. This is no serious drawback. If the mutants of our domesticated breeds were generally so nearly dominant as to be distinguishable only by the aid of biometric refinements, they would still constitute an anomaly inexplicable by the evolutionary hypothesis. It has therefore been thought sufficient to work on a scale which should reveal large and consistent differences, when they exist, but which is inadequate to detect, or to disprove the existence of many smaller differences, which may exist, but which, if they do, would not greatly affect any judgment as to the completeness of dominance.

4—Crest

The gene for crest, like several others used in these experiments, was obtained from Japanese Silky pullets, bought from English breeders in 1929, and mated with a wild jungle cock kindly lent to me for the season by the Zoological Society. From that time the experiment proceeded as planned, giving, in 1933, crested birds of the fifth filial generation, which were mated in 1934 with a view to producing homozygotes for the mutant gene. The numbers bred in the different years varied greatly, principally owing to the fact that initially the entire stock

was crested, while in later years, when the different "dominants" had been separated, only a single pen was given to this factor. In this, as in some other lines, breeding and survival were capricious, and the line came very near to extinction.

The four pullets used for the original cross in 1929 exhibited the five "dominant" characters—crest, black pigment, rose comb, polydactyly, and feathered feet—in addition to the recessive silky feathers characteristic of the breed. They appear to have been all heterozygous for three of these factors, rose comb, polydactyly, and feathered feet, though homozygous for crest. From the first generation onwards, owing to the change in the structure of the feathers, the manifestation of the gene for crest has been of very different appearance from that shown in the Silky, being a tuft of somewhat erected and elongated feathers, growing on the fore-part of the skull, not at all resembling the little mop of loose feathers of the Silky breed. In length and erectness there was, in the second generation, noticeable variation, but not enough examples were reared in later generations to detect any trend in the average of these characters. A comparison of the specimens preserved from each year shows no important change in the manifestation from the first generation onwards. If anything, the length tends somewhat to decrease.

In 1930, nine crested pullets of the first generation were mated to cocks of a wild strain, kindly presented by Mr. Spedan Lewis, who has otherwise most generously supported the experiment. All subsequent matings to the wild were made using this strain. A large progeny was reared in this year, of which 62 survived to be classified for crest, as is possible at about 12 weeks from hatching, 27 being crested and 35 without crest. In this generation the chickens were segregating in all of the 5 factors introduced in this cross. Crest appeared to segregate independently from the other 4. Since 3 of the 9 first-generation pullets had not feathered feet, the data for this factor are of little value. For black internal pigment (G), recognizable in the chicks by green feet, there appeared :—

Normal	G	Cr	GCr	Total recombinations	Total chicks
19	16	17	10	33	62

For rose comb (R) there survived :—

Normal	R	Cr	RCr	Total recombinations	Total chicks
23	12	14	13	26	62

and for polydactyly (P) :—*

Normal	P	Cr	PCr	Total recombinations	Total chicks
20	15	14	13	29	62

* Hutt (1933) cites data showing 243 recombinations out of 524 between crest and polydactyly. The data given above bring the totals to 272 out of 586. The significance of the discrepancy is somewhat increased by the addition, from $\chi^2 = 2{\cdot}76$ to $\chi^2 = 3{\cdot}01$. It should scarcely be regarded as significant unless further data raise its value to 4 or more.

The values for rose comb give a hint of linkage, but one that is not statistically significant on the numbers recorded.

Six of the pullets of the second generation, used in the third cross with wild cocks, were crested, but of these only one was free from the other dominants. Two carried G, two more carried P, and the remaining one R. Pullets with more than two dominants, including crest, were not used in this generation, but the pens in which the different dominants were bred could not be separated. One of the pens in this year (1931), however, happened to have crested hens only, and these gave in the third generation 15 which survived to be classified, of which only 4 were crested. In 1932, 2 crested pullets, now free from other mutants, gave, in 4 broods, 19 chicks, 7 of which survived to be classified. Of these, the fourth generation, only 1, a male, was crested. This male was mated in 1933 to pullets of the wild strain, but the 2 broods obtained gave only 3 chicks of the fifth generation, all crested, of which the 2 survivors, fortunately a cock and an hen, were mated together in 1934.

The possibility that novelties appearing in the sixth generation, the first in which the offspring could be homozygous, might be due to factors other than the mutant gene studied, has already been discussed. It was not, however, expected that the homozygotes would reveal themselves, save by a greater elongation or erectness of the head feathers, which would not ordinarily be apparent for some months after hatching. The first brood of 1934, of 3 chicks only, showed, indeed, nothing unusual, but the three subsequent broods all showed one or more chicks with a marked cerebral hernia. The record of the character in the 4 broods is shown in Table I.

TABLE I

Brood	Normal	Hernia	Total
F	3	0	3
M	8	3	11
V	7	1	8
X	3	1	4
Total	21	5	26

The proportion of 5 out of 26 agrees, within less than its standard error, with the one-quarter of homozygotes expected on breeding together two heterozygous birds. I should, therefore, on the evidence of the experiment alone, have been confident in identifying the chicks showing cerebral hernia with the expected homozygotes. The evidence of the experiment cannot, however, be considered in isolation, because (i) both crest and hernia have been used in other genetical work, and (ii) the original Silky birds used in 1929, which, as we have seen, were homozygous for crest, showed no external signs of cerebral hernia. We must, therefore, enquire first how it should have happened that crest and hernia have been regarded by poultry geneticists for many years as due to independent factors, if in reality they were caused by the same gene.

DARWIN (1868) gives an extensive description, with illustrations, of the domed and perforated skulls of a number of crested breeds. That he associated the hernia with crest cannot be doubted. It is important that he particularly emphasized the great differences in the deformation of the skull to be found in different breeds.

> "From the foregoing facts we see in how astonishing a manner some of the bones of the skull vary in Crested fowls. The protuberance may certainly be called in one sense a monstrosity, as being wholly unlike anything observed in nature; but as in ordinary cases it is not injurious to the bird, and as it is strictly inherited, it can hardly in another sense be called a monstrosity. A series may be formed commencing with the black-boned Silk fowl, which has a very small crest with the skull beneath penetrated only by a few minute orifices, but with no other change in its structure; and from this first stage we may proceed to fowls with a moderately large crest, which rests, according to BECHSTEIN, on a fleshy mass, but without any protuberance in the skull. . . . Lastly, when we come to fowls with a largely developed crest, the skull becomes largely protuberant and is perforated by a multitude of irregular open spaces. The close relation between the crest and the size of the bony protuberance is shown in another way; for Mr. TEGETMEIER informs me that if chickens lately hatched be selected with a large bony protuberance, when adult they will have a large crest."

The protuberance in the external form of the head may be clearly seen in the photographs, fig. 1, of chicks of the sixth generation (1934) showing hernia, in comparison with others, normal in appearance, from the same broods. In one that died at 19 days from hatching, fig. 2, the protuberance has no bony roof, but is due to a protrusion of the brain between the two halves of the frontal bone, forcing them apart and giving the posterior portions of the bone, which are still united along the median line, a reversed curvature or crumple. A second specimen died at 82 days, when the crest was already apparent, and is also without any bony roof. Of the remaining three, two, both crested, were killed in September by a weasel, which also ate the head of one of them. The surviving bird is a pullet with an exceptionally large crest.

The observations of DARWIN leave no doubt that some degree of cerebral hernia is characteristic of the crested breeds, as it should be if hernia is a manifestation of the gene for crest when homozygous. They show further that great differences in manifestation are due to other heritable factors in which the breeds differ, and that in some, at least, of these the manifestation has been much modified by human selection. In the light of the present experiment, in which, it will be remembered, the gene for crest was actually derived from Silky fowls, it would appear that these have undergone a modification even more striking than, though in a different direction from, the prominent domed skull produced in the Polish. The primitive reaction, as observed in wild stock, is seen to be a bursting of the brain case between the anterior portions of the frontal bones, possibly repaired to some extent in later

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FIG. 1—Chicks bred from crested parents in the sixth generation, 1934, showing hernia, in comparison with normal chicks from the same brood

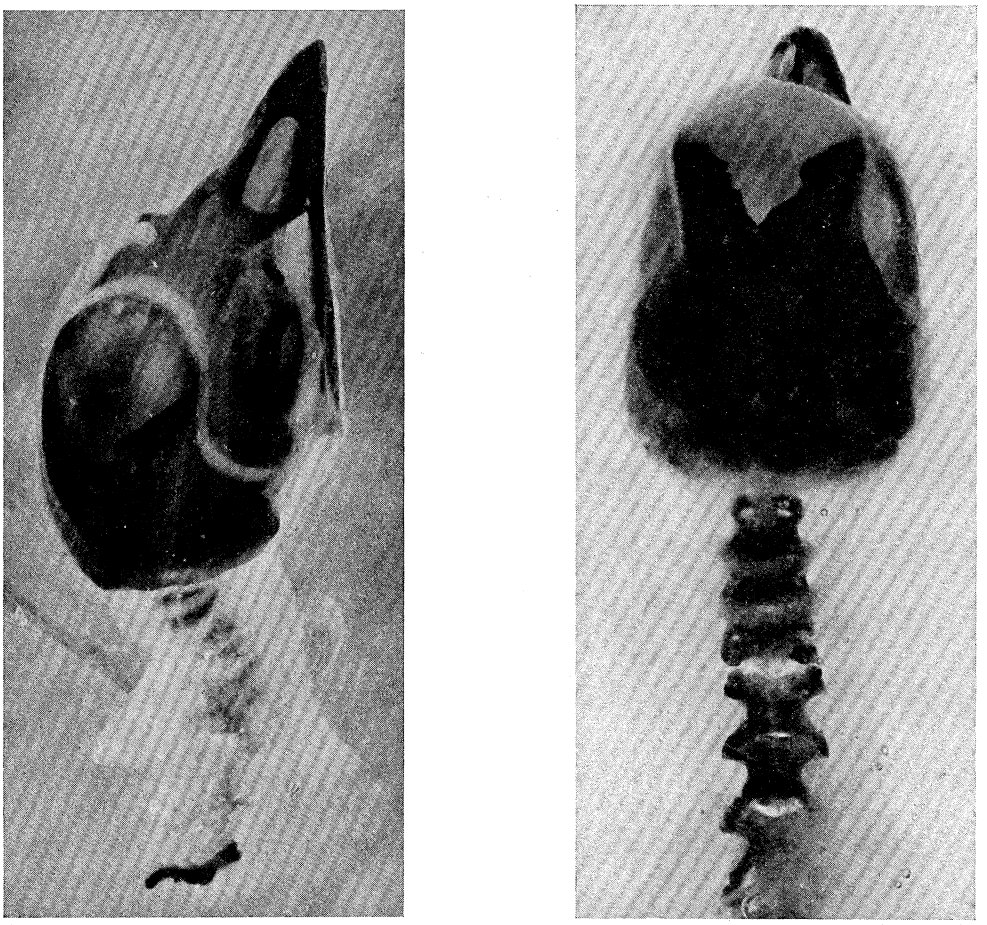

FIG. 2—Photographs in glycerine of herniated chick, 1934, from crested parents. Died at 19 days from hatching

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life by the completion of the skull by secondary ossification over the greater part of the hernia. In the Polish the high dome of bone has been exaggerated, thus giving a greater erectness to the crest feathers growing over the forepart of the head. In the Silky the hernia has been largely suppressed with a consequent approximation in appearance between the heterozygous mutant and the homozygote, or, in other words, rendering the mutation apparently dominant. In the wild fowl the mutation, if it had been first studied in this breed, would have been more likely classified as a recessive than as a dominant, for its homozygous manifestation is clear at hatching and persists throughout life. It is further shown in the greater erectness of the crest, which also covers a larger area, and seems to be longer ; while the heterozygous manifestation of a less prominent crest is only recognizable after some months. Properly, however, the mutation should not be described either as dominant or recessive, since the heterozygote is, when mature, clearly distinguishable from both homozygotes. If the terms dominant and recessive are used at all in such a case they should only be applied to particular aspects of the mutant gene's effects. In the skulls, I have observed no difference between the heterozygous crested and the uncrested birds. Thus it might be said that in the rupture of the brain-case the mutant is recessive, being analogous in this respect to the more harmful effects of most other mutations ; but that in the crest itself it is semi-dominant. Crest, therefore, supplies a further example of a phenomenon, the importance of which was first recognized by Ford (1930) in *Drosophila*, namely, that with mutants having two or more effects, those which are definitely deleterious may be completely recessive, even though semi-dominance is shown in other and apparently less harmful effects. We may thus suppose that relatively severe counter selection against cerebral hernia in the wild population is responsible for its recessiveness, while the mere erection and elongation of the feathers of the head have been less, if at all, harmful.

The conception that crest and hernia are due to two distinct and independent genetic factors seems to have originated with Davenport (1903). Davenport reports three crosses involving the crested varieties, Polish, Houdan, and Silky, but of these only the first two seem to have been carried as far as the second generation. With Houdan crossed with White Leghorn, of 45 chicks in the second generation 11 manifested hernia. Crest was classified for only 19 individuals, of which 6 were uncrested. The 13 crested birds evidently included all the survivors with hernia, for it is stated that " Hernia is never found dissociated from the crest." The number of these survivors is, however, not stated. This experiment is in perfect accord with the view that hernia appeared in birds homozygous for crest.

The experiment with Polish, crossed to Minorca, was on a more extensive scale, but the record of it is difficult to interpret, owing to inconsistencies in the numbers stated in different tables. In the second generation is appears that 52 chickens were hatched, and 23, which died in the shell, were also classified for crest. Of these embryos, 12 are stated to be crestless, and the author calls attention to the probability that in some of these the crest may have been indistinguishable. As

has been stated, in my own material no crest is visible before 10 or 12 weeks from hatching, and the deficiency of crested birds among the embryos in DAVENPORT's experiment strongly suggests that the classification of these must have been uncertain. The simultaneous classification for crest and hernia is, however, given for 70 birds, of which at least 18 must have been embryos, as follows :—

Normal	Crest without hernia	Hernia without crest	Crest and hernia	Total
21	34	3	12	70

Since hernia is readily recognizable in the chick, while crest is not, I should have no hesitation in interpreting DAVENPORT's record of these broods as :—

Normal	Heterozygous	Homozygous crested
21	34	15

conforming satisfactorily with a 1 : 2 : 1 ratio. The 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, as lacking crest.

It is apparently solely on the questionable classification of these 3 birds that DAVENPORT bases the statement in his general summary : " The crest is independent of the cerebral hernia."

The evidence on which DARWIN's views were set aside must be thought surprisingly slight. Nevertheless, it should be remembered that the period at which DAVENPORT wrote was one in which extraordinary confidence was felt in the evolutionary importance of simple genetic results, and in which the work of earlier writers was too commonly disregarded.

DAVENPORT's statement with respect to the cross with Houdan that " Hernia is never found dissociated from the crest " was misinterpreted by PUNNETT as applying to the whole of his experiments (1923, p. 103).

> " DAVENPORT states in the same paper that ' the hernia is never found dissociated from the crest,' but as he himself records three cases of uncrested birds with hernia, this statement would seem to require qualification."

In his general conclusion, however, DAVENPORT had stated that " The crest is independent of the cerebral hernia." PUNNETT's criticism was, therefore, unjustified. His comment on DAVENPORT's work undoubtedly enhanced the impression that DAVENPORT's experiments provided solid ground for separating crest from hernia, by ascribing to these three admittedly doubtful observations a scientific decisiveness which they do not possess.

At the time DAVENPORT wrote linkage had not been discovered. It is, therefore, interesting that his record from this same cross displays a clear instance of linkage involving the crested gene, which strongly confirms its identity with that causing hernia. In the Polish fowl the comb is almost completely suppressed,

but in the heterozygotes the comb was split posteriorly in the form of a Y. In this comb character, therefore, dominance is absent and the heterozygote can be distinguished from both homozygotes. Fortunately, DAVENPORT gives a table showing the simultaneous classification of 70 birds with respect to comb, crest, and hernia, which are arranged in Table II.

TABLE II

	Normal	Crest	Hernia	Total
Single comb . .	12 (9·00)	8 (7·10)	2 (1·40)	22
Split comb . .	8 (7·10)	17 (20·81)	4 (7·10)	29
No comb . . .	1 (1·40)	9 (7·10)	9 (9·00)	19

The totals for the comb character suggest that some heterozygotes may have been classified as having the single comb. Any such misclassification would tend to lower the apparent linkage, or to increase the apparent recombination frequency which, as judged from the data, is between 28% and 29%. The expectations inserted in Table II are for 28·27%. It will be seen that these expectations agree closely with the numbers observed. Both the combinations crestless-single and hernia-combless are relatively frequent, which would be an unintelligible accident if hernia were not due to the same gene as crest, or to one closely linked with it. Again, the combinations normal-combless and hernia-single are the least frequent in the table, having together only 3 representatives, in perfect correspondence with the same conclusion. From DAVENPORT's data alone it might have been concluded, had genetic linkage at that time been discovered, that hernia and crest must be due either to the same gene or to closely linked genes, both linked with the gene for split comb.

A more recent experiment, which was followed up on a large scale, was reported first by DUNN and JULL (1927), and later discussed, with additional data, by DUNN and LANDAUER (1930). This experiment is notable both for the further information it provides on linkage, and for the confirmation of the view that the genetic basis for hernia is present in normal birds of the Silky breed, but is suppressed by the special genetic constitution characteristic of the breed. The evidence for partial suppression in the second and later generations from a cross with Silky is especially valuable.

A Silky cock was mated to White Leghorn hens. The 39 birds surviving to be classified in the first filial generation were all crested. The crests varied considerably in area, erectness, and length. These birds mated *inter se* gave 46 crested to 16 uncrested, an entirely normal 3 : 1 ratio, indicating crest as a simple dominant. The authors remark :—

> "The most interesting and unexpected result of the cross of Silky by Leghorn was the appearance in F_2 of several chickens with pronounced cerebral hernia, similar to that which occurs in Polish fowls. . . . Neither of the parent types nor any of the immediate ancestors of the Leghorns had such cerebral hernia, and I have been unable to find any reference to its occurrence either in Silky or Leghorn fowls."

Nineteen out of 155 chicks and embryos showed hernia, 12 of these survived hatching, but only one lived to maturity. Sex was ascertained in 16 cases, there being 14 females, and only two males, both of which died as embryos. It will be noted that in this generation hernia appears in nearly a quarter of the females, but is almost entirely suppressed in the males. The authors recall that DARWIN quotes BLUMENBACH (1805) as stating that at that time the skull of the Polish fowl was protuberant only in the female. DARWIN (1868) remarks :—

> " Hence there can be no doubt that this remarkable character in the skulls of the Polish fowls was formerly in Germany confined to the female sex, but has now been transferred to the males, and has thus become common to both sexes."

Theoretically, the sex difference in DUNN and JULL's second generation might be due to autosomal factors, if the hernia is more easily suppressed in the male than in the female. The data can equally be explained as due to a sex-linked factor derived from the Silky, having a semi-dominant effect analogous to that which will be shown to be produced by the barred gene. No single interpretation can, however, be established from the data.

DUNN and JULL show that hernia is linked with pile, or " dominant white." This linkage is interesting on two points, first as showing that the gene for hernia itself cannot be sex-linked ; had it been, it could not have been transmitted through a succession of females, as in my own experiment ; and, secondly, by making it possible to test further the conclusion that crest and hernia are due to the same gene, as we have done in the case of linkage with the Polish comb in DAVENPORT's data. Unfortunately, recessive white was introduced also into the cross through the Silky parent. Of 83 birds classified for colour and hernia, using females only, but including 3 birds with hernia unclassified for sex, DUNN and JULL give :—

	Observed	Expected
White normal	63	60·73
White hernia	5	6·70
Coloured normal	3	1·52
Coloured hernia	12	14·05
	83	83·00

For comparison I have appended expectations based on 5% recombination between pile and hernia, a value which agrees generally with the other data of the experiment. Owing to the inclusion of recessive white, and to the variable manifestation of hernia, no very precise linkage value can be obtained from any part of the experiment. For example, the value estimated only from the data shown above would be 7·63%.

Let us turn to the observed relation between crest and colour. The 62 birds classified for crest were :—

	Observed	Expected
Crest white	38	34·90
Crest coloured	8	11·60
Crestless white	16	15·47
Crestless coloured	0	0·03
Total	62	62·00

The numbers set out as expected again refer to expectations on the basis of 5% recombination. It will be seen that there is no significant discrepancy. No argument can be based on the observed numbers opposed to the view that crest, equally with hernia, is linked with pile. The evidence for linkage in this case is weaker, partly because of the smaller numbers, but principally because the genes concerned are now in repulsion, instead of, as before, in coupling. It is a fact far too frequently overlooked that data from F_2 generations in repulsion give far less information on linkage than would the same number of observations from a cross in coupling. The data for crest and colour do, however, provide positive confirmation of our view, in the entire absence of coloured birds without crest, which with close linkage should be very rare. Without linkage the expectation in this class is 2·91.

The enlarged material, published in 1930, throws no further light on the linkage of crest with colour, but clearly confirms the linkage of colour with hernia, and gives much further information on the incidence of suppression. The new data for the second generation show 12 males out of 146 as having hernia, which we may interpret as manifestation in 33% of the birds carrying the necessary genes. Of the females, there were 39 out of 192, or 81% manifestation.* Heterozygous hens mated to herniated cocks of the second generation gave 25 out of 95 with hernia, or 53%. The same heterozygous hens mated to an F_3 cock with pronounced hernia gave 28 out of 60, or practically complete manifestation (93%). Matings between herniated fowls of the third generation gave 52 with hernia out of 61, or 84%. All the normals of known sex were males. The authors interpret these results as meaning that the gene for hernia occurs in the Silky breed :

> " But that in the absence of other factors necessary for its expression it seldom reaches a recognizable degree of development."

They further note :—

> " The expression of hernia is not only very variable in the embryo and newly hatched chick, but the character may become progressively less marked with advancing ossification of the cranium, especially in males. Thus males recorded as having pronounced hernia when hatched may show no protuberance at all as adults and may be classed as normals."

* These percentages are calculated on the numbers of herniated birds expected. Percentage manifestation in this sense corresponds to what TIMOFEEFF-RESSOVSKY has termed " penetrance."

In regard to the possible connection with crest, the authors remark :—

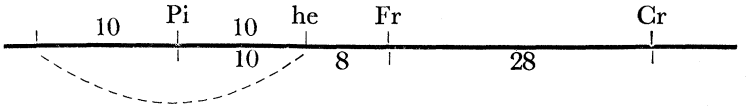
> " In our material all herniated fowls reared to maturity have developed a pronounced crest. Nevertheless, there is probably not a true genetic association or identity between crest and hernia, for we have found one instance in which an *uncrested* fowl has transmitted hernia."

No further details of this case are given, and evidently no strain showing hernia without crest has been produced from this bird, as could have been done if she really contained the gene for hernia without that for crest. Without such confirmation this single example remains inconclusive. That the authors were also influenced in their conclusion by the belief that their data showed that crest was not closely linked with pile, or " dominant white," is shown by the following further comment :—

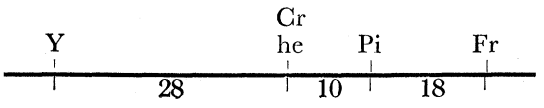
> " The possibility of a close linkage between the two genes is made improbable by the fact that, although hernia is very closely linked with the recessive allelomorph of dominant white, crest appeared in our previous data to be independent of this colour gene."

It has, however, already been shown above that the data for crest and colour are entirely consistent with very close linkage between these factors.

The identification of the genes postulated for crest and hernia establishes and simplifies an interesting linkage-group in poultry recently proposed by Hutt. In 1932, Suttle and Sipe had shown that crest was linked with frizzled plumage. In extensive and consistent back crosses they obtained 67 recombinations out of 239 or 28·3% ; exactly the same value, though much more precisely determined, as we have obtained from Davenport's data for the recombination between crest and the Y comb. In 1933, Hutt published data showing a linkage between frizzle and pile with only 18% recombination, thus connecting the linkage found by Suttle and Sipe (1932) with the linkage between pile and hernia noticed by Dunn and Jull. Believing, however, that these writers had shown that crest was not closely linked with pile, Hutt proposed the serial arrangement



in which frizzle (*Fr*) lies between pile (*Pi*) and crest (*Cr*), and in which the position of hernia (*he*) is placed ambiguously in either of two loci 10 units away from pile. The ambiguity disappears when crest and hernia are placed at the same locus. Indeed, Dr. Hutt informed me, when in 1934 I communicated to him the occurrence of hernia in my crested line, that he had already convinced himself that the genes for crest and hernia must be identical. The arrangement of the genes must therefore, be



in which I have added the Y comb of DAVENPORT's experiment on the side away from frizzle, with which, if it is (as seems improbable) the split comb of the experiments of LANDAUER (1932), and SEREBROVSKY and PETROV (1930), it appears not to be closely linked. Since in at least three out of these four factors dominance is absent, and the heterozygote is clearly distinguishable from both homozygotes, the group supplies attractive facilities for linkage studies provided that the suppressor or suppressors of hernia are excluded from the experimental material.

It has been mentioned that HUTT (1933) called attention to the possibility that polydactyly is loosely linked with crest and pile. Tests with frizzle, or with the Y comb from Polish, or with both, would now seem to offer an opportunity of testing this suggestion.

5—POLYDACTYLY

As a dominant, polydactyly in poultry has not enjoyed the same unquestioned reputation as has crest. BATESON early found instances of five-toed birds bred from four-toed parents. It was not that dominance was imperfect in the sense that the heterozygote could be distinguished with any confidence from both the homozygous forms ; but rather, on the contrary, that it might be indistinguishable from either. As BATESON puts it (1909, p. 53) :—

> " Imperfection of dominance does not even obscure the application of Mendelian analysis. The cases in which difficulty does arise are those in which dominance is irregular and the recessive class cannot be distinguished with certainty. In the fowl, for instance, the extra toe is usually a dominant, but in some strains there is irregularity, and birds without the extra toe may nevertheless transmit it."

In spite of this irregularity, polydactyly has until recently been habitually termed a dominant. Since the commencement of my experiment, however, two publications have appeared dealing with the results obtained with this factor, which demonstrate their variability in different crosses among domesticated breeds, and which, without further investigation, might have sufficed to show that dominance in this character has been much modified in the course of the formation of these breeds.

The first of these, by PUNNETT and PEASE (1929), is particularly valuable for containing a summary of numerous progenies raised by the authors, and by previous workers in different crosses. In conclusion, the authors state :—

> " In the first place we feel little doubt that there is a definite factor for polydactyly. It is evident also that this factor can be carried by an apparently normal four-toed bird. This is probably due to the existence of a factor (or possibly factors) inhibiting the action of the factor for polydactyly, and this factor (or factors) may be carried by normal recessive four-toed birds. Further, since the evidence points to polydactylous birds being also capable of carrying

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the inhibitor, we must suppose that we are also concerned with some other factor, or factors, rendering possible the manifestation of the polydactylous effect in spite of the presence of the inhibitor. But although we have devised various schemes along these lines we have not found one which we consider satisfactory."

An entirely different interpretation of the data discussed by PUNNETT and PEASE was shortly afterwards put forward by HUTCHINSON (1931), who shows that the facts are consistent with the theory that dominance is controlled by genetic factors, and that different degrees of dominance are found in different strains. Without postulating that either homozygote can be made by an inhibitor, or super-inhibitor, to resemble the opposite kind of homozygote, HUTCHINSON shows that in some crosses there is apparently complete, or nearly complete, dominance of the polydactylous condition; in others, it is the four-toed condition which is completely or nearly completely dominant; while in a large number the heterozygotes are divided more or less unequally, some being four- and some five-toed, owing to the segregation of factors controlling dominance. Factors controlling dominance can, of course, be introduced with either the four- or the five-toed parent.

If an inhibitor existed capable of reducing homozygous polydactyl (PP) birds to the four-toed condition, its existence could be proved unequivocably by establishing a strain homozygous for polydactyly and for the inhibitor (IIPP), and therefore breeding true to the four-toed condition, which, when crossed with other strains lacking both these factors (*iipp*), and also invariably four-toed, would give regularly in the second generation a proportion of five-toed young, Further, if there existed, as PUNNETT and PEASE suggest, an inhibitor of such an inhibitor, permitting the manifestation of polydactyly in PP birds, even in the presence of the inhibitor, a strain breeding true for polydactyly could be made up containing both these inhibitors (SSIIPP), which, when mated with polydactyl strains containing neither of them (ssiiPP) would, in the second generation, invariably yield a proportion of four-toed young. The exact proportions to be expected in these two tests will depend on the dominance relations assumed for the inhibitors. But, whatever assumptions of this kind be made, it must be possible, on this view, both to produce five-toed young by crossing four-toed strains, and to produce four-toed offspring by crossing five-toed strains. Neither of these has been done. Until geneticists, who suspect the existence of an inhibitor in their stock, show that they can be done, there is no ground for going beyond the simple view that the apparent irregularities in the inheritance of polydactyly are due solely to modification of the heterozygotes, as proposed by HUTCHINSON.

The interest of the experiment now reported lies, therefore, in only three features: (1) in ascertaining experimentally what are the dominance relationships in a strain of wild fowls similar, presumably, in this respect to the wild fowls from which the domesticated breeds were derived; (2) in examining these relationships in material genetically purer, or presumably freer from genetic modifiers, than the products of breed crosses; (3) in examining, incidentally, whether this four-toed strain

contains any inhibitors capable of masking the manifestation of the polydactyly introduced into it.

The four Silky hens used were apparently all heterozygous for polydactyly, for they gave 17 four-toed and 17 five-toed young in the first generation. For the second generation, nine five-toed pullets were mated with a wild cock, and gave, out of 144 classified, 81 five-toed and 63 four-toed. Among the 81 are included 2 having actually only four toes on each foot in which the hallux was elongated, and contained two joints in place of one, as in fig. 3*a*L, where this feature is shown on one foot of a bird clearly polydactylous in the other. The excess of 9 over the expected frequency, 72 five-toed, is not statistically significant, but it leaves little room for the view that many birds carrying the mutation failed in this generation to manifest the polydactyly. Nevertheless, at least one such case seems to have occurred, for polydactyly appeared in the third generation in the pen reserved for feathered feet, and has never been completely eliminated from this line.

With respect to the evidence on linkage provided by the second generation, recombination with G (black internal pigment), manifested by green feet at hatching, showed even more than half the progeny in the recombination classes. The totals are :—

Normal	G	P	GP	Recombinations	Total
21	42	45	36	87	144

With rose comb, one bird was lost before classification for comb, but the remaining 143 gave

Normal	R	P	RP	Recombinations	Total
35	32	30	46	62	143

The deviation is in the direction of linkage, though not statistically significant.

Two polydactyl crested and two polydactyls without other dominants were mated for the third generation, and produced 12 polydactyl young ; in addition, 3 more appeared from four-toed mothers with feathered feet. Of these, seven polydactyl females were mated in 1932, giving 11 five-toed and 14 four-toed young. Four pullets of the fourth generation mated to a wild cock in 1933 gave 4 polydactyl and 6 normal. Of those with five toes, one male and two females survived, and were mated together in 1934, with a view to producing homozygotes for the polydactyl gene.

The segregation in different years is shown in Table III.

TABLE III

	Polydactyl	Normal	Total
1929	17	17	34
1930	81	63	144
1931	—	—	—
1932	11	14	25
1933	4	6	10
Total	113	100	213

The totals for four years, 113 : 100 have the appearance of an entirely undisturbed 1 : 1 ratio. Nevertheless, the data are consistent with the view that, whereas in the first two generations polydactyly was manifest in nearly all heterozygotes, yet in the later generations a certain proportion of heterozygotes were normal in appearance. There is an apparent excess of normal birds. In 1934 also, when the expectation was 1 in 4, there were 11 normal out of 31 birds. The comparison of normals expected and observed for the last three years is made in Table IV.

TABLE IV

	Expected	Observed	Sampling variance
1932	12·50	14	6·25
1933	5·00	6	2·5
1934	7·75	11	5·81
	25·25	31	14·56

The aggregate discrepancy is 5·75, but the standard error is 3·82, so that even if all heterozygotes were recognizable the discrepancy would not be beyond the fluctuations of random sampling.

The structure of the polydactyl foot varies considerably, and the variety of types is at first confusing. In order to have a basis for comparison with possible homozygotes, to be bred in 1934, during the last three years the feet of polydactyl birds have been prepared for examination by staining the bones with alizarin, and rendering them transparent by treatment with potash and, finally, with glycerin; 25–30 pairs of feet were thus examined from the second to the fifth generation. These are illustrated in fig. 3 *a–f*.

Disregarding minor differences the feet are of four main types which may be described as follows : (A) Normal feet in which the hallux, comprising one phalanx and a claw, is supported by a short metatarsal resting against the cannon-bone, to which the other three toes are articulated. In addition to the claw this type has only two bones in the region of the hallux (fig. 3, *c*R, *d*R, and *f*R). The types with three bones consist either (B) of a single hallux with two phalanges (fig. 3, *a*L), or (C) of a duplicated hallux of which one portion is articulated to the metatarsal while an additional bone, also carrying a claw, either floats freely by its side or arises from it as a spur (fig. 3, *c*L and *f*L). In type (D) there are five bones which, in every case except one, consist of two parallel metatarsals, which may or may not be fused, bearing respectively an extra hallux of two joints, and a normal hallux of one. In one exceptional case, found in an embryo, the two metatarsals are placed end to end and carry two extremely dwarfed terminal toes with one and two joints respectively. Type D (fig. 3, *a*R, *b*R and L, *d*L, *e*R and L), is by far the most common in heterozygotes, 10 birds out of 25 having this type on both feet. On a count of 50 feet, 56% were of type D, 28% of type C, and 14% of type A.

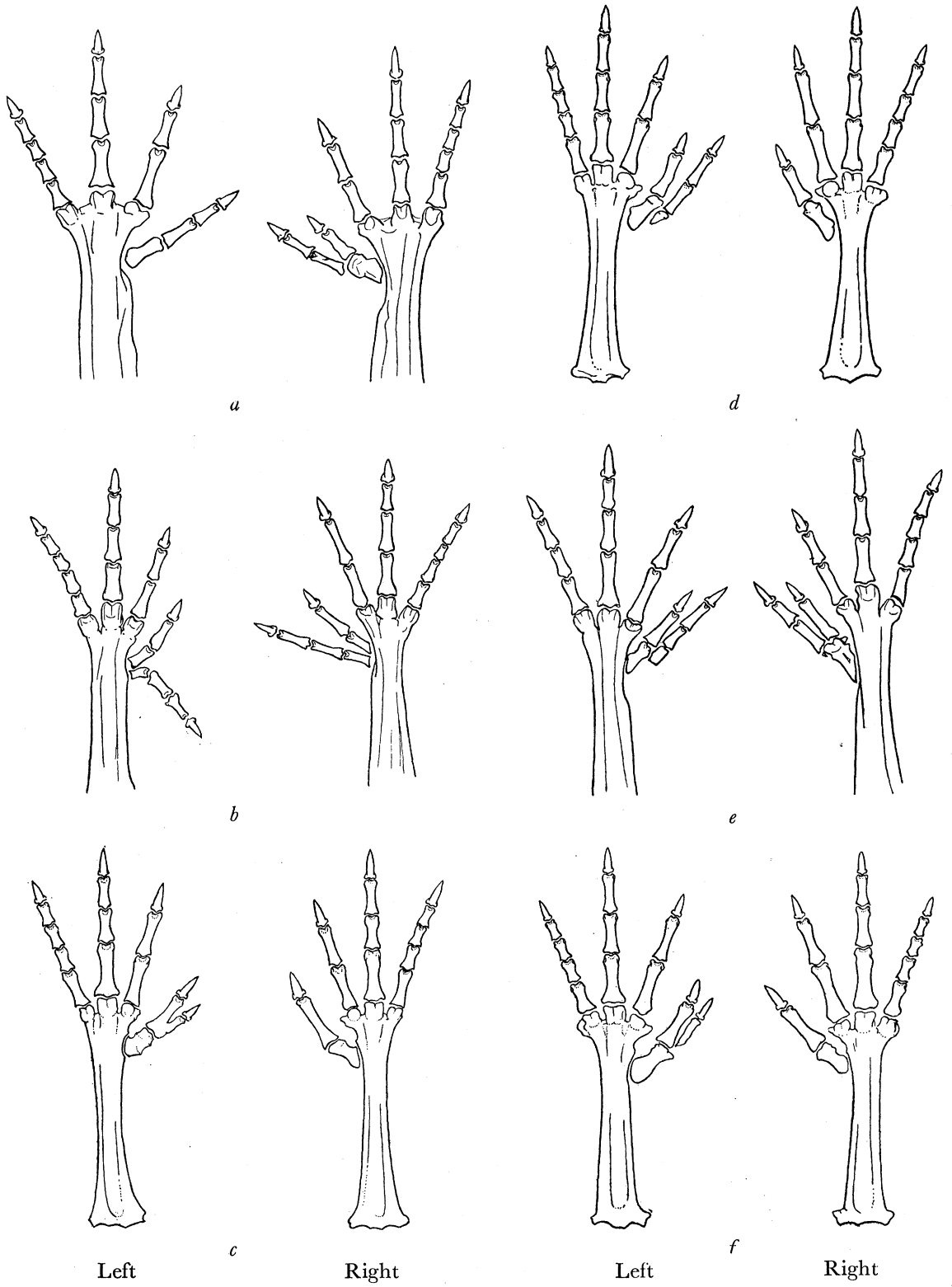

FIG. 3—Six pairs of polydactyl feet, illustrating types A, B, C, and D
Polydactyl ♀ D, 1930-31; *b* polydactyl ♀ B, 1930-31; *c* polydactyl ♀ A, 1931-32; *d* polydactyl ♂ E, 1932; *e* polydactyl ♀ C, 1930-31; *f* polydactyl ♂ F, 1932

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Only one foot was of type B which has evidently been quite rare in the later years of the experiment.

The two feet of the same bird are usually similar, but the different types are not equally frequent on the two sides. Bond (1920) has emphasized the fact that when there is asymmetry, polydactyly is more fully developed on the left foot than on the right. In confirmation of this I find that all seven of the normal feet found on polydactyl birds were on the right side, paired in five cases with type C, and in two cases with type D. On the other hand, asymmetry of birds having five bones on one side and three on the other is, in my material, equally frequent in both directions. In addition to the feet of birds recognizably polydactyl, there would in a complete census be at least a few cases in which both feet were normal, or of type A, and these would somewhat lower the average number of bones. The number of such birds would doubtless be much greater than it is were it not for the readiness of the left foot to be disturbed from the normal pattern.

Taking the two feet together and counting two bones for A, three for B or C, and five for D, the distribution of 25 heterozygotes is :

Number of bones both sides together	Frequency
5	5
6	3
7	2
8	4
9	1
10	10

The bird with nine bones had a right foot classed as D in the classification used above, but in which the proximal ends of the phalanges are fused and the metatarsal must be counted as single. The average number of bones in this series is just less than eight. If three normal birds were added with four bones each, the average would be brought down to $7\frac{1}{2}$. The average, therefore, probably lies between $7\frac{1}{2}$ and 8 units. The frequency distribution is shown in fig. 4.

As has been stated, of 31 birds bred in 1934, 11 were normal ; the remaining 20, however, showed, in four of the broods, exceptional manifestations of polydactyly, of kinds which had not appeared in previous generations. Six birds out of 20 showed these novel features, which are just such as might be looked for from an enhancement of the action of polydactyly. The most characteristic of these is the appearance of three phalanges in the extra hallux. This may be accompanied by a hallux with one (fig. 5, AL and BR) or with two (fig. 5, CR) phalanges. In one bird the hallux is almost completely suppressed, and has no bone, though there are two metatarsals, fig. 5, CL. In another case the claw of the extra hallux is duplicated, fig. 5, BL, showing a tendency towards the formation of three toes in place of the hallux. As will be seen from the figures, the new and presumably

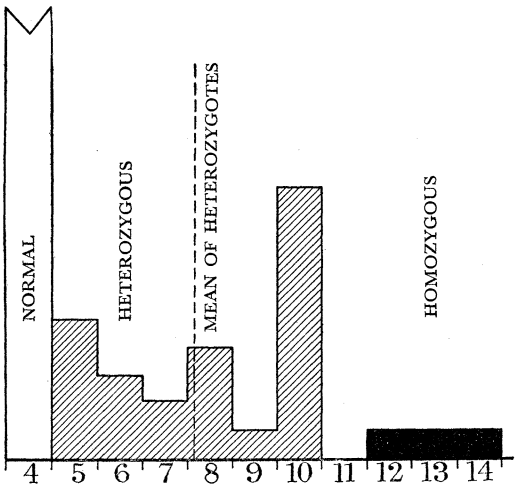

FIG. 4—Frequency distribution of number of bones, taking both feet together, in the halluces of 25 heterozygotes for polydactyly, in comparison with homozygous normal and homozygous polydactyls

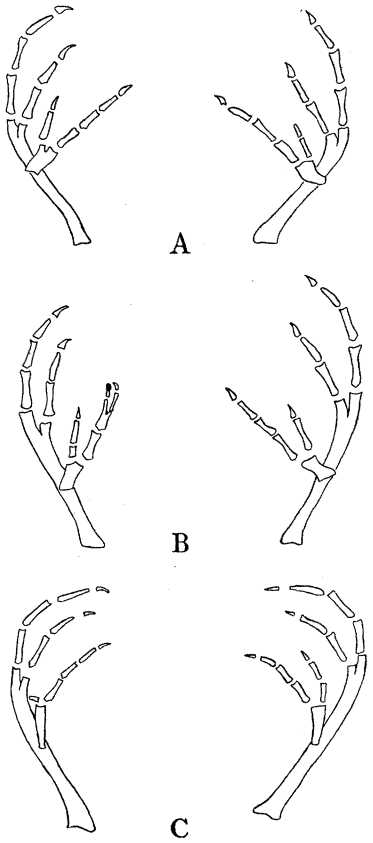

FIG. 5—Homozygous PP chicks from heterozygous parents, 1934. A, brood T, 1934, 37 days; B, brood W, 1934, 41 days; C, brood N, 1934, 10 days

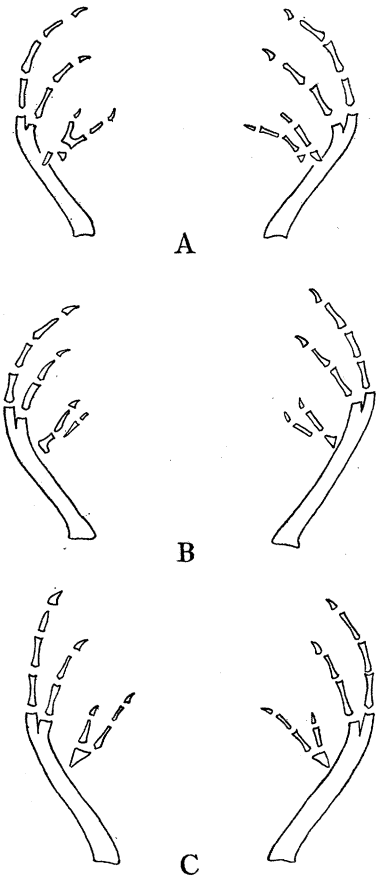

FIG. 6—Heterozygous (Pp) chicks from heterozygous parents, 1934. A, brood N, embryo, both feet type D; B, brood W, 5 days, both feet type C; C, brood B, 9 days, both feet type D

homozygous types are as distinct from the heterozygotes as these are from the normal. No doubtful or intermediate structure has appeared in the limited material so far studied, though it is not improbable that such would occur among a larger number. For comparison, fig. 6 shows similar sketches of heterozygous birds from the same broods.

In addition to the three-jointed toes, a second feature which regularly appears in the homozygotes is the backward elongation of the metatarsal, which, instead of resting against the cannon-bone, projects externally along or across it. The number of bones in the region of the hallux counted on the same convention as before in the specimens so far available are 12, 13, and 14. The average is evidently at least as far removed from that of the heterozygotes (between $7\frac{1}{2}$ and 8) as these are from the normal number of four.

6—Barred

The mutant barred manifests itself by transverse white bands on the feathers, the intermediate spaces being also generally paler than in unbarred birds. Unlike the other mutants included in the experiment, it is sex-linked. Since in birds the female is heterogametic, only two types of hens are known, barred or not-barred, according as each has received from her father a mutant or non-mutant gene. Cocks, on the other hand, which receive sex-linked genes from both parents and transmit them equally to their sons and daughters may be homozygous, either barred or normal, or heterozygous through having received different genes from their two parents. Dominance can, therefore, only show itself in the males. Moreover, the mutant gene cannot be introduced by breeding with wild cocks for more than one generation in succession, since it is never transmitted from mother to daughter. In most years, therefore, the line was continued by breeding a heterozygous barred cock with wild hens.

Since barred does not occur in the Silky breed, an attempt, which proved unsuccessful, was made in 1929 to mate the wild cock with a barred Plymouth Rock hen which was evidently too big for mating to be successful. Later in the year a small barred pullet was kindly presented by Mr. M. S. Pease, and produced a brood of six, including three barred cocks. Besides barred this pullet contained genes for silver, rose comb, polydactyly, and a black affecting the plumage of many of her descendants; but these were all discarded in subsequent generations.

A cock from this mating heterozygous for the sex-linked factors barred and silver and for the autosomal factors rose comb and polydactyly, mated in 1930 to wild hens, gave an exceedingly varied progeny, several of which showed black plumage. In all 13 were unbarred and 8 barred. Two of the barred were cocks. Of these, one, apparently normal in all respects, save for having a brown breast, mated in 1931 to wild hens gave, in three broods, 5 normal and 9 barred. Of the latter 3 cocks and 3 hens survived to be sexed.

In 1932, the three barred pullets mated with a wild cock gave 6 normal females and 5 barred males. In these three years the low viability and backwardness of the barred males was very noticeable, and in 1932 in particular, at ages when the males normally should be growing away from their sisters, the backwardness of the barred males was particularly noticeable. One, however, from the first brood, attained adult plumage, and was fit to mate in 1933, although a second male from the same brood was extremely small when killed in the spring of that year. In 1933, however, a succession of good broods were obtained comprising, in addition to 17 normal, 14 barred, of which 6 females and 5 males lived to be sexed. One male was again fit to breed from, and was mated in 1934 to 5 barred females, with a view to producing homozygous cocks.

The five preliminary years thus produced 44 normal and 39 barred birds, in good accordance with the expected equality. Of these, 35 normal and 31 barred came from barred sires. Whereas, of the normals 12 males and 12 females survived to be sexed, of the barred birds there were 10 males and 18 females, and of the males, the majority were very ill-developed. There was therefore some reason to fear that the homozygous males when produced might be nearly inviable.

The first brood, however, in 1934, of three chicks, contained two which were unmistakably males homozygous for barred. The third chick of the brood, a heterozygous male, had, like the barred birds bred in previous years, chick plumage nearly of the wild type with a pale spot in the middle of the dark band which normally runs from the forehead down the back of the neck. The two others were unmistakably lighter, having large white patches on the sides of the head and neck, including the ears, light wings, and on the body the dark lateral stripes quite obliterated. Fig. 7 shows one of these chicks, which died at 38 days, together with one of the ordinary barred, either a female or a heterozygous male, and one with the wild down plumage. It will be seen that the homozygote is much more distinct from the heterozygote than the latter is from the wild.

The five broods reared in 1934 contain 25 living chicks, 9 normal, 11 barred and 5 homozygous males. Including chicks dying at hatching and in the shell, there were 12 normal, 20 barred, and 5 homozygotes. There thus seems to be some deficiency of homozygotes, but it is not accounted for by deaths at or shortly before hatching.

As the illustration shows, the contrast in chick plumage found on introducing barred into a wild strain is practically identical with that discovered by Punnett and Pease (1930) in material used as the basis for the new breed known as the Cambar. This breed has been developed at Cambridge for the special property that, unlike other pure breeds, the sexes can be distinguished at hatching. This property is evidently also possessed by the wild jungle fowl when the mutant barred is introduced into it. In such a strain the mutant is not a dominant, but is more nearly a recessive, though the heterozygote is distinguishable from the wild throughout life. The contrast with the homozygous male is much more pronounced. It is interesting that the barred female does not resemble the homozygous male, but is,

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in the chick stages, little if at all different from the heterozygote, a feature suggestive of the possibility that all females transmit the normal allelomorph of barred from mother to daughter.

7—Summary and Conclusions

The reputed dominant mutant for crest in poultry appears to be due to the same gene as the reputed recessive mutant for hernia. In wild stock the heterozygote

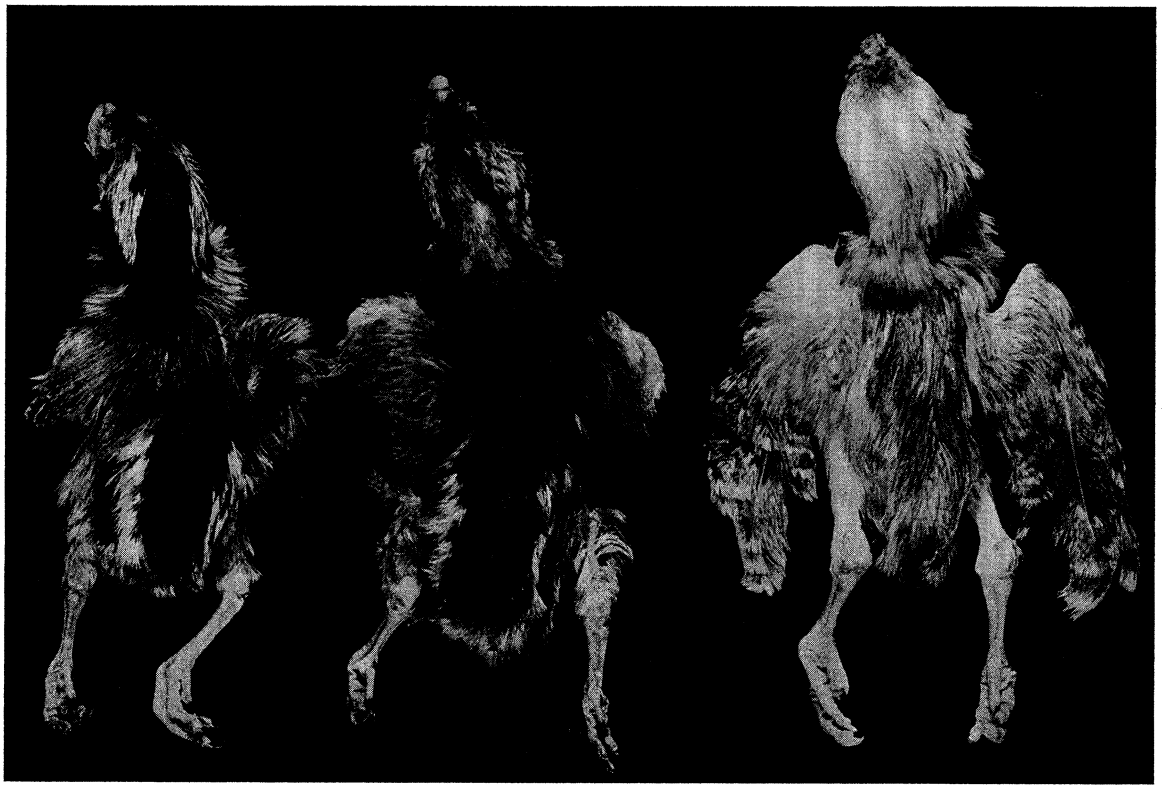

Fig. 7—Homozygous barred male (right), but slightly feathered, and still showing the down pattern on head and body, contrasted with heterozygous male, or barred female (centre), and normal unbarred (left) in down plumage

differs obviously from both homozygotes, in possessing crest and in lacking hernia respectively.

During the period of domestication hernia has been suppressed in the Japanese Silky variety by an hereditary factor, or factors, which suppress the hernia more readily in the male than in the female. Other crested breeds, such as the Polish, appear to have been formerly affected by a similar suppression which has been eliminated since breeders have aimed at a prominent domed skull as a fancier's point.

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In a breed with hernia completely suppressed, the mutation would appear to be a simple dominant, whereas in the wild fowl the injurious manifestation, hernia, is completely recessive, while the crest is intermediate, or partially dominant, in the heterozygote.

The belief of DARWIN and earlier writers that hernia was connected with crest was abandoned by geneticists early in the 20th century on inadequate evidence. With two incomplete exceptions, the modern data conform completely with the view that crest and hernia are due to the same gene. This view also explains other significant features in the data ; notably the complete absence of grown birds showing hernia without crest, and the linkage relations with the Polish comb, and with pile.

The identification of the genes postulated for crest and hernia confirms, and to some extent modifies, the autosomal linkage group proposed by HUTT. The four factors for Polish comb, crest and hernia, dominant white or pile, and frizzle, apparently lie in that order. Dominance is absent, apart from the action in some strains of suppressors of hernia, in at least three of these four cases.

So far as the present material is representative, polydactyly, when introduced into the wild jungle fowl, is a typical example of the mutations with intermediate heterozygotes in which dominance is absent.

The heterozygote is distinctly variable, having four to ten bones, counting both feet together, in the region of the hallux, and therefore occasionally overlapping the normal homozygote. The most frequent number is ten, and this it apparently does not exceed, nor does it overlap the mutant homozygote by the production of 3-jointed halluces. The variability of the heterozygote is suggestive, in view of the modifiability of its average expression, as shown by Hutchinson, in different breeds. The homozygotes also are variable in structure, but the variation observable is not so much in the degree of expression of the character as in the various ways in which the developing tissues adjust themselves to the abnormal situation produced by the mutant genes. In the wild stock it would certainly seem possible, without appreciable loss of accuracy due to misclassification, to discriminate the three genotypes, in linkage studies, or for other purposes. The difficulties encountered in varietal crosses seem to be wholly due to the segregation of modifiers affecting the degree of dominance.

No inhibitor of polydactyly was introduced by the wild cocks used for crossing.

Barred is more nearly a recessive than a dominant mutation ; the heterozygous males are more like the wild than like the barred homozygotes.

The barred hen when young is indistinguishable from the *heterozygous* male. The barred mutation thus introduces a sexual differentiation.

In breeds in which this sexual differentiation does not exist, barred may be described as a dominant, though perhaps as always to some extent an imperfect one.

The sexual differentiation found by PUNNETT and PEASE, and on which is based the breed known as the Cambar, is, in the chick stages, the same as that found in a wild stock into which the barred gene has been introduced.

The three factors reported on in this paper thus entirely bear out the suggestion that the supposed " dominants " found in domesticated breeds of poultry show distinct lack of dominance when introduced singly into a wild strain. Any dominance, therefore, which is shown by them in breed crosses must be due to modification during the period of domestication.

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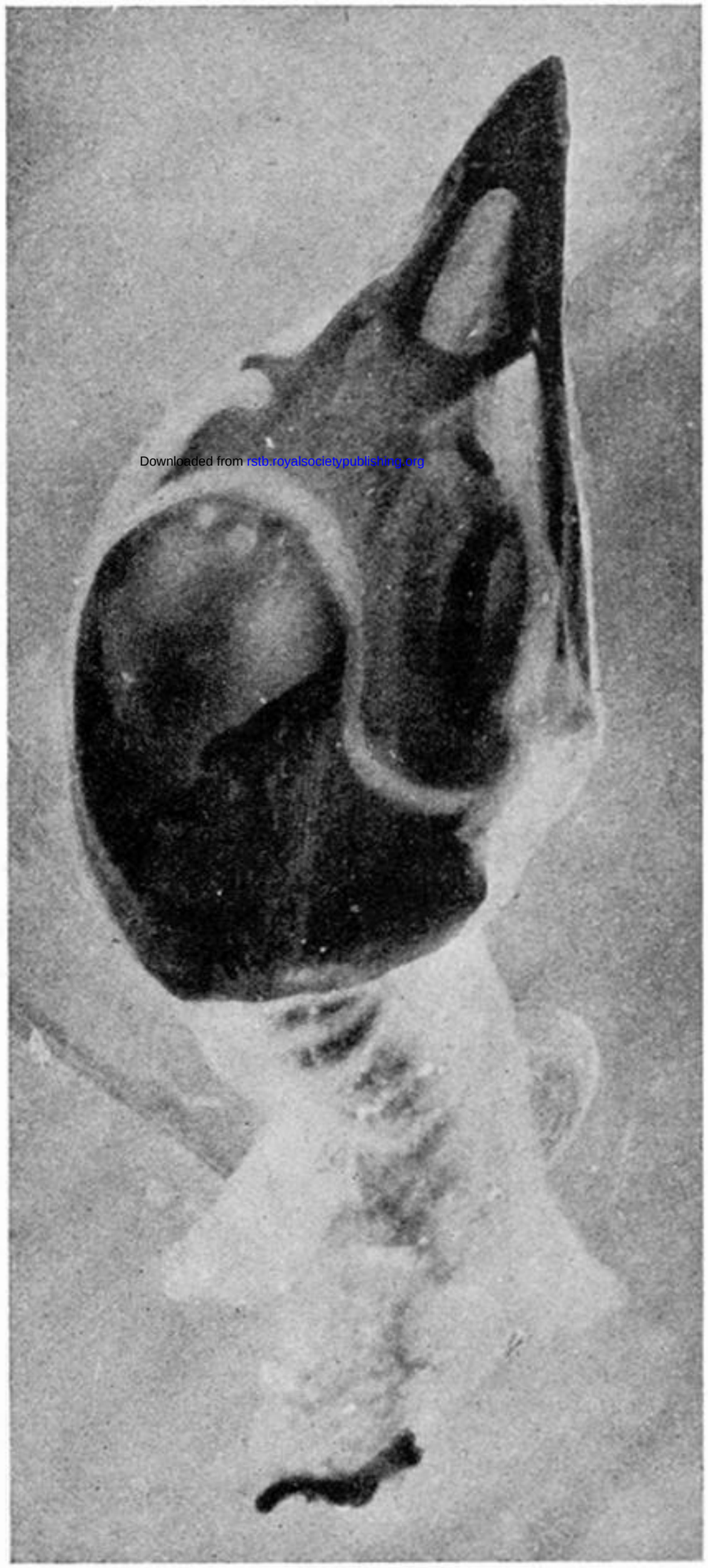
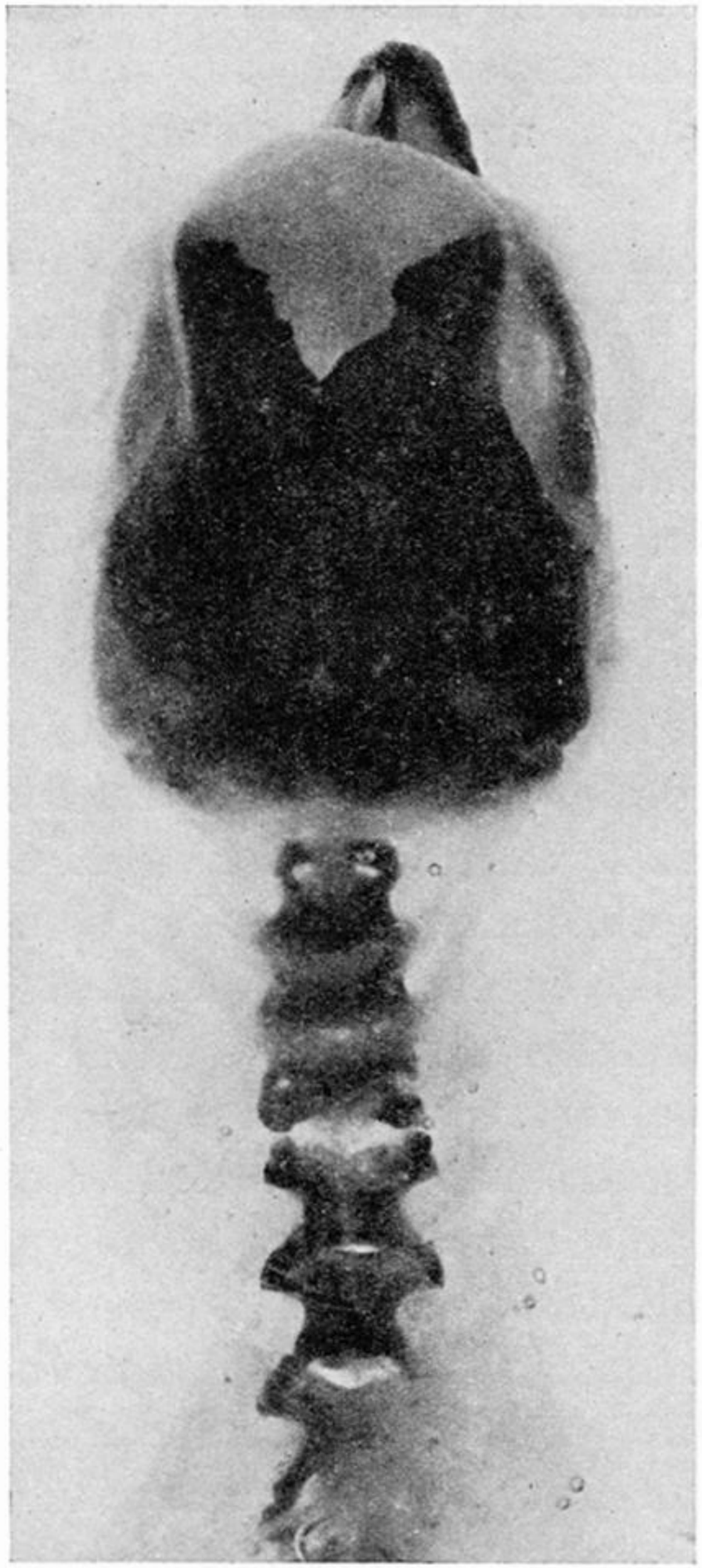
G. 1—Chicks bred from crested parents in the sixth generation, 1934, showing hernia, in comparison with normal chicks from the same brood





IG. 2—Photographs in glycerine of herniated chick, 1934, from crested parents. Died at 19 days from hatching

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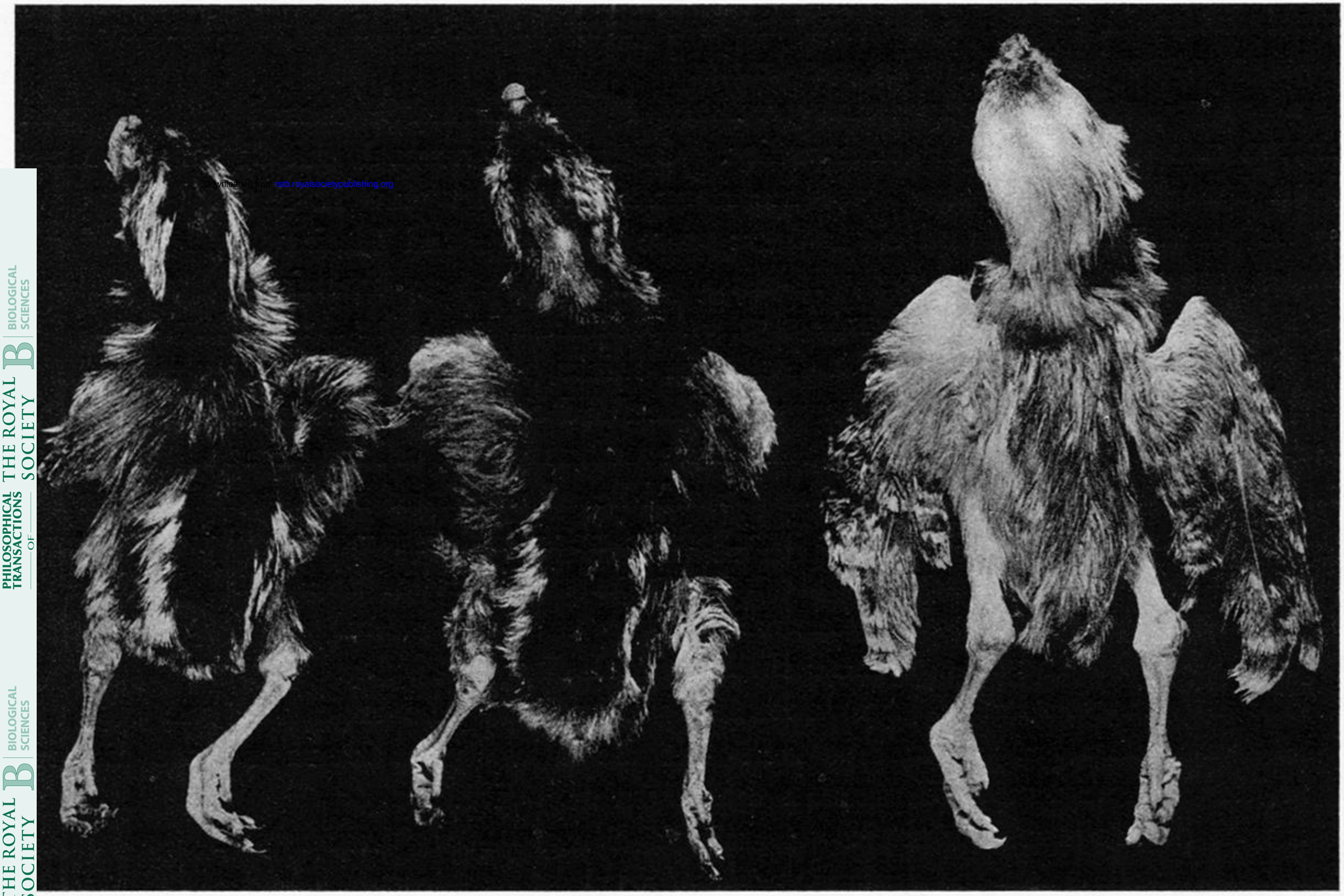

G. 7—Homozygous barred male (right), but slightly feathered, and still showing the down pattern on head and body, contrasted with heterozygous male, or barred female (centre), and normal unbarred (left) in down plumage