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FECUNDITY IN THE DOMESTIC FOWL AND THE SELECTION PROBLEM

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I

In the December number of the American Naturalist Professor W. E. Castle directs a vigorous attack against the present writer’s work on fecundity. Any one reading Professor Castle’s article could scarcely fail, I think, to carry away the impression that the whole of the writer’s studies of the past eight years on fecundity in the domestic fowl are to be regarded as essentially valueless. I assume that it was not the intention to convey this impression. The fact, however, appears to be as here stated. With such a conclusion I can scarcely be expected to agree. I shall therefore attempt, in the following pages, in the first place, to call attention to some points regarding my own work which Professor Castle appears to have overlooked, and which seem calculated to give it at least some slight degree of significance, and in the second place, to set forth very briefly my reasons for venturing, in the present state of knowledge, to hold a different opinion from his in regard to some phases of the selection problem.

II

The general plan of Professor Castle’s paper appears to be to make a comparison between his selection experiments with rats, and my selection experiments with poultry, to the very great disadvantage of the latter. To this general comparison no general comment on my part can be made, except assent to Castle’s conclusion that his

1 Papers from the Biological Laboratory of the Maine Agricultural Experiment Station, No. 94.
work on the selection problem is vastly superior to my own. Since the subject of such comparison has been opened it gives me great pleasure to pay tribute, in all sincerity, to Professor Castle’s splendid series of experiments on selection in rats. In respect of the numbers of animals involved and their superior adaptability for such an experiment, his work with rats altogether transcends anything which has been done with fowls. These selection experiments constitute an achievement of which their author may well be proud. I have ventured to disagree with Professor Castle’s interpretation of the results for reasons which will presently be stated. But this difference of opinion, I would most strongly emphasize, concerns only the interpretation. We are at one in our high admiration of the factual basis afforded by the rat experiments.

III

Granting all this, however, it seems to me that possibly the case against my studies of fecundity in toto is not quite so bad as Castle makes it out to be. Let us examine his points seriatim. In the first place the strictures upon the character egg production on p. 714 seem to me to overdo the matter a bit. It is of course true that it is a character confined in its expression to one sex, though that it is also a character which is transmitted by the other sex even Castle somewhat grudgingly admits (p. 715). It also is a character which comes to expression only in the adult. Of this Castle makes a great point throughout his paper, emphasizing that this means that only a small proportion of all offspring born can take part in selection experiments. From the standpoint of methodology this point has nothing like the significance which Castle attributes to it, for the very simple reason that in all breeding experiments, his own included, there is a vast amount of random sampling between the population of parental genes and the population of offspring somata. When Professor Castle breeds a pair of rats only a very few
sperm and ova out of the vast hordes the parents produce take part in the production of the resulting litter. He operates, of course, upon the basis that those germ cells which do take part in the formation of the litter constitute a random sample of the whole population. When I put pullets into the house to test their egg production I operate on precisely the same basis, viz., that I have a random sample of the family from which they are taken. As a matter of fact, I have been at great pains to ensure that the sampling should be random. In all of my studies on the inheritance of fecundity I have regarded this as a point of paramount importance, and have never made use (except occasionally for confirmation of points already made out on other material) of families in which I had not either tested all the daughters as to egg production or a sufficiently large random sample to be fairly representative of the family. Further I have repeatedly made careful ad hoc investigations of the adequacy and randomness of my sampling.

Castle’s next point is, as the matter stands, apparently well taken. He quotes (p. 715) a statement which I somewhat rashly made to the effect “that phænotypic variation of the character fecundity in fowls, markedly transcends, in extent and degree, genotypic variation.” Professor Castle’s treatment of this statement is perfectly legitimate. If it were true, as stated, it would admit of being turned around as it is in Castle’s next sentence, and then it surely would be silly to talk about either selection for this character or about its Mendelian inheritance. What I should have said when I wrote that unfortunate sentence, but did not, was that phænotypic variation may transcend genotypic in fecundity, not that it always or regularly does. Because it may I wanted to point out the need for great care in respect of environmental conditions in interpreting results with this character. The real point is this: Long experience in working with winter egg production in poultry has convinced me that under properly controlled environmental conditions this character is as
definitely and regularly controlled by hereditary factors as is the plumage color and pattern. On the other hand, it is a character which is rather particularly sensitive to environmental influences in one direction, namely, downward. I can breed a flock of birds which I know will be high winter layers if properly fed, housed and managed. But if these birds are starved, housed in a damp cold place and otherwise maltreated they will lay but a few if any eggs. Under such conditions the genotypic condition would be swamped by the environment. It was this sort of thing I had in mind when I made the statement that Castle quotes. It should be particularly noted, however, that this is a somewhat one-sided matter. I can (because I have done so) breed a flock of pullets lacking totally the factors for winter production. With such birds nothing can be done in the way of feeding or management which will make them lay before some time in February or March when the spring cycle begins.

Now all my work on fecundity has been done in a public institution. Egg production is a commercially valuable thing. We have had to submit the results of our breeding operations, in the shape of the birds themselves, to the practical test of farmers, poultrymen, etc. In doing this there has always been vividly before my mind the fact that unless the birds were given proper feed and care, no matter what the genes they carried, they would not lay many eggs.

On the other hand the degree of expression of the character in birds carrying the factors for high fecundity may be favorably influenced by exceptionally favorable circumstances, though the possible effects in this direction are, according to my observations, much smaller in amount than in the opposite direction.

While Castle’s comments on the unfortunate sentence under discussion are technically perfectly legitimate, I do not think he is quite fair to the essential underlying point of genetic epistemology, namely, the impossibility of judging the genetic constitution by the somatic appearance.
This of course is the reason for the progeny test. I do not think I am in any sense exaggerating if I say that it is one of the chief results of the Mendelian method of studying inheritance to show that in many cases and for many characters it is impossible, in the absence of a progeny test, to be sure of the genetic constitution of the individual from an examination of the soma alone. I fancy that if I cared to be fussily nasty in my controversial methods I could cite page after page from Professor Castle’s Mendelian writings where even he, in order to be quite sure about the genetic constitution of an individual, has had to breed it. This is all I mean by the progeny test. Why am I and my fowls held up to scorn and ridicule because I say that it is frequently impossible to tell the genetic constitution of a fowl with respect to fecundity without breeding it? Surely fecundity in poultry and coat color in rats only differ in this respect in degree, if they differ at all, not in kind. I think if any one will read pp. 604 and 605 of my last Naturalist paper, which is the immediate objective of Professor Castle’s attack, he will have to admit that the interpretation which I give of the earlier results is not entirely senseless, and might indeed explain them. In any case, it is in thorough accord, methodologically considered, with the very best current Mendelian usage, including that of Professor Castle himself.

IV

This brings us to the most serious phase of Castle’s attack, namely that in which he denies the validity of my conclusions respecting the inheritance of the character fecundity in fowls.

On the top of p. 716 he asserts that I “assume” that two Mendelizing factors are concerned in the inheritance of fecundity, “but without any sufficient published evidence for either conclusion.” As I have published3 many pages of evidence in demonstration of my conclusions on this point, one can only infer from this statement of

Castle's that he regards that evidence as totally worthless. It has not so appealed to other workers.\(^4\) Furthermore I think it can be shown that methodologically my treatment of the problem of inheritance of fecundity stands on precisely the same plane as Mendelian work in general, and Professor Castle's Mendelian work in particular. This I shall now try to do.

The essence of a test of a Mendelian hypothesis lies in this: the genetic constitution of the parents of an array of offspring necessitates that the individual offspring bearing different segregating characters, or different segregating categories of the same character, shall occur in definite numerical proportions. If the observed numerical proportions of the offspring agree, within the limits of error due to random sampling, with the proportions expected from the Mendelian hypothesis, then this fact constitutes valid evidence in support of the hypothesis. If no exceptions to this rule appear and a sufficient number of agreeing cases are adduced the hypothesis is regarded as demonstrated. The number of cases necessary to constitute a proof is a purely individual matter. What one person will consider sufficient to establish proof another will not.

Now in the case of fecundity in fowls, Pearl and Surface\(^5\) first established that the Barred Plymouth Rock stock at the Maine Experiment Station was not homozygous in respect of winter egg production, but that it contained, with frequent occurrence, individuals of high fecundity, and also individuals of low fecundity. The race not being homozygous with respect to fecundity, it was possible to test the Mendelian inheritance of this

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character within the race, without crossing, by the above scheme.

The next step was the definition of the categories of the character winter egg production. From long study of the character I concluded that the natural categories in this strain were (a) zero winter production, (b) winter production between zero and 30 eggs, and (c) a winter production of over 30 eggs. These were chosen as working categories. If any one will turn to p. 719 of Professor Castle's paper and examine Fig. 1, which is there printed, they will find that even he chooses categories of the character with which he is working. Nowhere have these ever been quantitatively defined; nowhere has he ever presented any evidence that the step from his rat grade +1 (for example) to his grade +2 represents a more or less inclusive category than a difference in winter production of from 0 to 30 eggs. Professor Castle reads us a beautiful little homily about Mendel's peas. But I am not clear that either Mendel or Castle has shown that the amount of variation within the category "yellow" is less than the amount of variation within my fecundity category of "under 30." From the only study which has ever been made of the matter, Weldon's, I should certainly conclude that the category "under 30" in winter egg production carries within itself distinctly less variation than the category "yellowness" in peas. Castle's assertion about my fecundity categories ill becomes one whose work in genetics has dealt almost without a single exception with non-quantitatively defined Mendelian categories. Of course, as a matter of fact, he knows, I know, and everybody knows that the variations within the Mendelizing category are of no significance so far as the Mendelian result is concerned. I happen to have observed, for example, that there are at least four genetically distinct rose combs in poultry. Yet they are all rose; any of them crossed with single gives a 3:1 ratio in F2.

Having chosen these categories of the character fecundity because they appeared to represent natural divisions, I proceeded to show for hundreds of matings the distribution of the progeny when individual females whose performance fell into one or another of the categories were mated to particular males. This was done both for the pure bred Barred Rocks and for crosses. The results at once showed that definite ratios were appearing with regularity and constancy. Further analysis showed that a Mendelian hypothesis which postulated two factors, one sex-linked and the other not, accounted for all the facts.

If all this does not conform to the classic canons of Mendelian experimentation, I am sure I do not know what does.

V

Castle charges me with suppressing data. There are just two things which I wish to say regarding this charge. The first is that I shall publish the complete raw data of my work on the inheritance of fecundity when I have finished my own study of these, and not sooner. I am using this material for the study of various problems. There appears to be no reason why I should make valuable original records public property until such time as I have finished my own analysis of them. If Professor Castle will examine my published papers he will find that in lines of work which I am finishing and leaving, complete raw data are published (cf. for example "A Biometrical Study of Egg Production in the Domestic Fowl," Parts I to III).

In the second place I wish to say that so far as any question of concealment is concerned Professor Castle, or any of his students, will be very welcome to come to the laboratory at any time, for as long as they like, and make any examination of the original record books in connection with published results and conclusions.

There is one further point which needs consideration concerning the charge of suppression of pertinent facts. An important reason, I think, why Professor Castle's own interpretation of his rat selection experiment has not been
freely and universally accepted by workers in genetics lies in the fact that he has never presented his results in such a form that any other interpretation of the data could by any chance be tested. There is, from the methodological standpoint, only one way in which an adequate test can be made as to whether any observed change in the composition of a population is the result of a sorting, or of true germinal change, or an adequate idea gained of how the change came about. This is the method of individual pedigree analysis. Only one extensive mass selection experiment has ever been analyzed in this way, and that is in Surface’s discussion of the Illinois corn results. The Hagedoorn’s called Castle’s attention two years ago to the necessity of individual pedigrees before any just opinion could be formed as to the meaning of the data. To paraphrase Castle’s damming indictment of the present writer I may be permitted to call attention to the fact that, so far as concerns the individual pedigree of his rats, “information is denied us” by Castle.

In bringing to a close this part of the discussion I wish to emphasize that, in spite of Castle’s assertion to the contrary, any unprejudiced person who will take the trouble to examine the facts will find that, so far as concerns methods of dealing with the data and presenting them for publication, the method of their Mendelian analysis, the method of presenting the results of selection experiments by a series of averages, and other matters of method, my work with fecundity in fowls exactly parallels at every point Castle’s work with hooded rats, and is in every way, so far as I am able to judge, exactly as critical as his. His experiments are more extensive in scope than mine, and the character fecundity is a more difficult one to deal with, but so far as methodology is concerned the two researches stand on precisely the same footing. I have not

lumped the data any more, nor have I "suppressed" data any more than he has. On the contrary I have published a great deal of exact data, in a series of papers from this laboratory, regarding the character fecundity, its normal variation, etc.

VI

The next point which Castle makes is that the changes which occurred in mean flock production during the sixteen years, for which figures were given in the paper which he criticizes, were probably due to environmental, or at least to non-genetic effects. In making this point he calmly disregards all that I have ever published about the experiments, the means taken to be sure that environmental effects were not mistaken for genetic, etc., and proceeds in his discussion as though all my work on the subject had been absolutely uncritical and that I had never given a thought to checking the correctness of the results. In the first place he notes the changes in the numbers of birds on which the average in different years are based, and points out that these numbers change in a roughly inverse direction to the means. He then says:

Has not the better environment and lessened competition of small numbers possibly something to do with the result?

They have not. Had Professor Castle been less eager to demolish these fecundity results he might have noted that I have repeatedly stated that since 1908 all birds in these experiments have been kept in flocks of the same size, namely 125 birds per flock. The number of such flocks has at times varied, but not the number in each flock except by very small numbers, such as resulted from losses by death, the necessity occasionally of putting a few extra birds in a pen for a brief period and similar very minor

9 To prevent any mental strain in reconciling the above statement with the third column of Table I, p. 599, in my Naturalist paper, let me hasten to say that the pens were filled out, if the number of Barred Rocks in the selection experiments did not just equal multiples of 125, with birds from other experiments.
fluctuations. In the first four years (1899–1900, 1900–1901, 1901–1902, 1902–1903) of the old experiment the birds were kept in 50-bird flocks. During the five years following (i.e., to 1908–1909) they were kept in 50, 100, and 150 bird flocks. Just precisely how much (or really how little) difference the size of flock made in average egg production has been fully and minutely analyzed biometrically and published by Pearl and Surface\(^{10}\) some six years ago. It seems reasonable to suggest that before indulging in fast and loose criticism on such a simple point of fact as this it would become Professor Castle to read the literature respecting the work he is attacking.

Since this material seems to have been forgotten it may be well to repeat here that the results showed (Pearl and Surface loc. cit, p. 115) that in general there was no significant difference in winter production between 50, 100, and 150 bird flocks. In later months of the laying year differences appeared but only in the last month of the winter period (February) was there any significant excess of even 50-bird flocks over the others. Furthermore, besides the material which has already been published regarding the possible influence of environmental factors on the results of these experiments, I have carried out a number of special investigations on different phases of this general question which have not yet been published. For example, I have minutely analyzed the data regarding date of hatching to see whether that might not enter as a significant factor in the interpretation of the results. The data on this question are being prepared for publication now, but it may be said in advance that the results show that date of hatching can not possibly have had anything to do with the rise in average flock production which has occurred between 1908 and 1915.

Turning now to the general problem of selection there are certain fundamental matters which it seems to me are in danger of being lost sight of in the rapid shiftings of view point which are an essential part of any general controversial campaign, such as Professor Castle’s writings of the last few years would indicate that he engaged in. These are:

1. The pure-line concept has certainly been one of the most useful working tools in the practical breeding of plants and animals that has ever appeared. Particularly in plant breeding the pioneer work at Svalöf, which has been repeated and duplicated on a most extensive scale in plant breeding laboratories all over the world, demonstrates in the most complete manner that, whatever may be happening in the germ-plasm of rats, certainly the germ-plasm of our common cereal crops is in such a state or condition that selection within the pure line is without effect. This is a fact, real and definite. It lies definitely at the basis of very extensive commercial seed breeding operations in various different countries. To any one familiar with the extent and stability of the practical applications of the pure-line concept in cereal breeding operations, some of our current discussions of the selection problem seem very academic indeed. Even the justly celebrated magnitude of Castle’s rat experiments is scarcely of the same order as the combined and accordant experience of expert cereal breeders throughout the world. Before any one makes up his mind finally about the problem of the efficiency of selection within the pure line it will be well to remember that besides Johannsen’s famous, if now in certain quarters somewhat distrusted, beans, there are all the Svalöf oats, wheats, etc., to be reckoned with.

2. No one has ever disputed the power of systematic selection to alter populations, which were not pure-lines. Such alteration may extend the range of variation very greatly beyond what it was in the original population.
From a methodological standpoint, however, it is necessary to have a very different sort of evidence from that afforded by changing general population means, such as Castle gives for his rats, and I for fecundity, to prove that the process of selection has been the cause of a change in the absolute somatic equivalent of a particular gene or hereditary determinant.

3. It is just in connection with this last point that there seems to me to be a good deal of unclear thinking and arguing at cross-purposes about the selection problem. Let us examine the logic of the matter symbolically.

Let there be a character $A$, whose somatic variation in the general population is given by a frequency distribution of area $Z \geq A_{41} A$, where $Z$ is the frequency of occurrence of the somatic state or condition $A_1$, and so on to $Z_n$ and $A_n$. Now suppose that selection is practised for the somatic condition $A_{40}$, but that in the original population $A_{38}$ is the most extreme variation in that direction found to exist. Then for $A_{40}$, $Z_{40} = 0$, and for $A_{38}$, $Z_{38}$ is very small. Let it be further supposed that the somatic difference between the $A_{38}$ and $A_{39}$ condition may be of any determinate magnitude $R$. It makes no difference to the logic of the case whether $R$ is large or is extremely minute. Now suppose, as a limiting case, that we assume a gametesoma correlation of 1, i.e., perfect. Then in the gonads of an individual somatically $A_{38}$, all the germ cells will bear the factor $a_{38}$. If two such individuals are bred together the progeny will be somatically $A_{38}$.\(^{11}\) Suppose that for $m$ generations the matings are of $A_{38} \times A_{38}$. This is continued selection. Then suppose in the $m + 1$th generation, $A_{38} \times A_{38}$ parents, appears an $A_{39}$ individual.

Concretely this represents a step in advance in the direction of selection. Let us analyze the possible ways in which this may have happened.

\(^{11}\)This is precisely the condition which prevails in a pure line of oats, except for purely phenotypic variation, superimposed by environmental factors.
First we may assume that $A_{38m}$ and $A_{38n}$, the parents of this $A_{39m+1}$, instead of having $a_{38}$ gametes had $a_{39}$ gametes. This would correspond to what is called a mutation. The gamete-soma correlation has been broken by the appearance of a new kind of gamete different from the parental gametes. There has been a sudden definite change in the germ plasm, such that an $a_{38}$ germ plasm has changed to an $a_{39}$ germ plasm.

Or, we may assume that $A_{39m+1}$ was produced by the union of two $a_{38}$ gametes, but that these gametes dedevelop a 39 soma instead of a 38 soma. This assumption leads logically straight to genetic indeterminism, a conclusion which, I think, is repugnant to all that is known regarding the physiology of the hereditary process.

Embracing alternative (a) then, we may next inquire as to the possible cause of this sudden change of the germ plasm, by an amount of which the somatic equivalent is $R$, from $a_{38}$ to $a_{39}$. If we say that this change has been caused by a selection, we can only conclude that the fact that $A_{381}, A_{382} \ldots A_{38m}$ have been placed in particular cages or apartments to breed, for this is the only physical thing that selection means in this case, has been the cause of the germinal change. For by hypothesis there has been no mixing of germ-plasms. We have been practising straight selection of the most extreme somatic individuals, all by hypothesis $A_{38}$, and each homozygous. It seems to me a misuse of terms to say in such a case as that postulated that selection has caused the appearance of the variation which it selects, unless we are prepared to say that the physical act of the selection of the individuals for mating physiologically effects the germ plasm. Such an assumption we are all agreed would be nonsense. What has happened in the postulated case is precisely this: a new heritable variation in the direction of selection has appeared while selection was in progress. If we say any more than this we are going beyond our facts. If the selectionist would state his results in this form, and
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not incessantly harp on the string that "selection caused" his results, he would be on logically solid ground and would receive a more respectful hearing from those who place a high value upon clear thinking and sound logic in scientific matters.

Now up to this point in the argument there has been no biological point involved, so far as I can see, to which anybody, whether of the pure line or the selection faith can take exception. Certainly I am perfectly willing to admit that germ-plasm changes do sometimes occur, of all magnitudes from the most minute up. Further no one, I take it, will deny that, having appeared, these variations may be seized upon and preserved by selection. I do desire to emphasize, however, that there is no evidence, as yet, that the selection causes the variations.

It may be objected that the postulated case is too simple and leaves out of account too many factors. All this, however, will not affect the logic of the case. Generalized, that logic is as follows: A heritable difference between two individuals or races implies a difference in the germ plasm. The difference in the germ plasm must have made its initial appearance at a definite point of time. At that time the germ plasm changed from its previous condition. The cause of that change can not be conceived to be the selection for breeding purposes of the parents bearing the unchanged germ plasm. To assert that the new variation is a result of amphimixis due to mating unlike parents would be, in the present state of genetic knowledge, a ridiculous begging of the question, because, in the first place, by hypothesis in any selection experiment individuals genetically as nearly alike as possible are always mated together, and in the second place, as selection continues homozygosity automatically increases.

The whole fact of the matter is that the assertion that selection per se causes changes in the germ plasm, is a wholly new addition to the classic Darwinian selection theory, tacked on quite inadvertently, I believe, by some of the modern exponents of that theory. Darwin never
supposed that selection was a cause of favorable variation. Instead he repeatedly pointed out that the fundamental problem behind natural selection was that of the cause of the variations which selection preserved. That problem remains to-day practically in the same condition that it was left by Darwin. We are no nearer, essentially, now than we were then to knowing the cause of new variations. The assertion that new variations are caused by selection is the rankest kind of mysticism plus bad logic.

But if selection of the parents can not be supposed the cause of new variations in the individual, then clearly what selection does, and all it can do, is to change the germinal constitution of a race or population by preserving those individuals in which new variations have appeared, and multiplying them. This is exactly what has been done in the hooded rat experiment, it seems to me on Castle's interpretation of the case. In that experiment every favorable variation in the many thousands of rats has been preserved and the individuals bearing it have been multiplied. Others have been thrown away. The range of the character in the direction of selection has been extended far beyond the original range. But would it have been so extended, or could it have been, if the favorable variations had not appeared for selection, or if, having appeared, they had not been heritable? Suppose one started such an experiment with a character which was in a stable condition and not varying. Take, for example, the single comb of fowls, and attempt by selection from a pure single-combed race to produce a stable rose-combed race by selection alone. Prophecy is dangerous business, but I do fancy one would be a very long time on that job! Characters, so far as I can see, will be altered following selection just in proportion as they are varying genotypically. The cause of the alteration is to be sought in the cause of the variations, not in the selection only.

I have for some time felt that probably the differences in opinion between the selectionists, as represented by
Castle, and the advocates of the pure-line concept, reduces itself finally very largely to a dispute over the use of words, if both are discussing the same objective facts or experiments. It is repugnant to the logical faculties of the pure-linists to be told that selection is a cause of new variations. On the other hand, I suspect that this particular use of words, which is offensive to our camp, would not be deemed absolutely essential to the making of their case by Castle and his followers. Castle’s special bête noir appears to be that the pure-linists seem to him to deny the possibility of germinal variation, except it be large in amount (a proper De Vriesian mutation). Now I am in no wise authorized to speak for the pure-line advocates, but I can say for myself, and I venture to think others would agree, that this contention forms no part of the real, genuine pure-line body of doctrine. The followers of the pure-line merely have observed in fact that it is not so easy to change all things by a process of selective breeding as it has been to change the pattern of Castle’s rats, or the egg production of my fowls. Many characters, and many organisms, when got into a homozygous condition exhibit any germinal variation so rarely as to make any change by the selection of such variation impossible within the limits of finite experimentation. Neither Johannsen nor any followers of his, so far as I am aware, have ever attempted to set any limitations on how big or how little a germinal variation could be.