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MENDEL'S LAWS AND THEIR PROBABLE RELATIONS TO INTRA-RACIAL HEREDITY.

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THE two volumes[1] that are the immediate occasion of these articles have more in common, besides their authorship, than appears from their respective titles; both deal solely with Mendel's Laws of Hybridisation, to which so much attention has been recently directed. The "Report to the Evolution Committee" of the Royal Society contains an account of experiments, begun apparently with other objects, but continued with a view of further testing the scope and validity of the laws. These experiments include crosses between species or varieties of *Lychnis*, *Atropa*, *Datura* and *Matthiola*, together with some observations on poultry. Generally speaking the results are in accordance with Mendel's rules, although Mr. Bateson and Miss Saunders, like other observers, found some difficulties and exceptions, notably in the case of the *Matthiola* hybrids and in the experiments with poultry. The Report on these experiments is preceded by a short account of the work of Mendel himself, and that of Correns, De Vries, and Tschermak, and is followed by some forty pages on "The Facts of Heredity in the light of Mendel's discovery." The second volume on "Mendel's Principles of Heredity" consists of two parts, the first containing a translation of Mendel's papers, with an introduction; the second, with the sub-title "A Defence of Mendel's Principles of Heredity," being a reply to Professor Weldon's article "On Mendel's Laws of Alternative Inheritance in Peas," which appeared in the January number of "*Biometrika*."

[1] (1) Royal Society. Reports to the Evolution Committee. Report I. Experiments undertaken by W. Bateson, F.R.S., and Miss E. R. Saunders. 1902.

(2) Mendel's Principles of Heredity: a Defence; by W. Bateson, F.R.S. Cambridge: at the University Press, 1902.

The facts collected by Mr. Bateson and Miss Saunders in the "Report," have considerably extended the area of application of the highly remarkable laws discovered by Mendel, as well as to a minor extent the list of exceptions; they do not, however, appear to throw much fresh light on the fundamental nature of the laws themselves. The sections of the two volumes which do appear to call for criticism and review are those relating to the bearing of Mendel's results on the conceptions of heredity in general, and on the work of Mr. Francis Galton and Professor Pearson in particular. Mr. Bateson devotes many words to these questions, but one cannot help feeling that his speculations would have had more value had he kept his emotions under better control; the style and method of the religious revivalist are ill-suited to scientific controversy. It is difficult to speak with patience either of the turgid and bombastic preface to "Mendel's Principles," with its reference to Scribes and Pharisees, and its Carlylean inversions of sentence, or of the grossly and gratuitously offensive reply to Professor Weldon and the almost equally offensive adulation of Mr. Galton and Professor Pearson. A writer who indulges himself in displays of this kind loses his right to be treated either as an impartial critic or as a sober speculator. Mr. Bateson is welcome to dissent from Professor Weldon's opinions, but it would have been well if he had imitated the studied moderation and courtesy of his article.

Mr. Bateson may no doubt congratulate himself on a *succès de scandale*, but it is difficult to see that his "Defence" attains any worthier goal. Apart altogether from the question of good manners, the entire history of scientific and philosophical controversy would have taught a more judicious disputant that personal polemic is the very worst method of arriving at truth; an attack of this kind can do nothing but distract attention from the scientific question and concentrate it upon ephemeral personalities. If Mendel's laws are of the importance that Mr. Bateson claims, the general acknowledgment of that importance is bound to come, whether one writer or another is sceptical or not. Nor does it appear that the responsible advisers of the Cambridge Press can be acquitted of a certain failure to appreciate the dignity that should belong to a University Press, in allowing the publication of a volume containing insinuations such as those Mr. Bateson has permitted himself to put on paper.

The fact that I am inclined to agree with Mr. Bateson as to the *possibly* very high importance in practice and theory of Mendelian

phenomena only makes me regret the more the defects of his style and manner of treatment. The assumed separation of characters in the germ cells of the hybrid on which Mendel based his explanation of the results he had observed, *may*, as Mr. Bateson suggests, very possibly be proved in the future to hold good over a much wider field than has yet been experimentally tested. Mendel's phenomena *may* bring us to revise fundamentally some of our conceptions of heredity, they may suggest new directions in which to seek for solutions of some problems of the cell, they may throw fresh light on the process of fertilization in general, and on the nature of variation. But it must be remembered that at present Mendel's Laws are only known to hold for cases of hybridisation, and do not appear to hold invariably then; in the present state of our knowledge it is speculation—legitimate, even desirable, but still speculation, pure and simple,—to postulate the existence of similar phenomena when breeding only with a pure strain. It is well that such a possibility should be borne in mind, and that the whole case should be fully and impartially discussed and considered. Yet Mr. Bateson has taken the very action most effectually calculated to render calm criticism and unbiassed judgment impossible, as he may by now have realized. The language of unbridled enthusiasm and lavish abuse creates nothing but mistrust. It is most regrettable that this convenient translation of Mendel's papers should appear in so disadvantageous a context. Many of the conclusions at which Mr. Bateson arrives seem so entirely due to his misunderstandings of various passages in the writings of Mr. Galton and Professor Pearson that I propose, in the first place, to deal not with Mendel's work at all, but with that of the statistical or biometrical school. Having cleared the ground in that direction, it will be easier to institute comparisons between the results obtained by the two schools, and to discuss the bearing of the two classes of observations on each other.

There has always been a good deal of misunderstanding between biologists in general and those who have done pioneer work in the use of statistical methods, due in great part, I believe, to the fact that the two do not use such terms as *heredity*, *variation*, *variable*, *variability*, in precisely the same signification. The employment of quantitative methods necessarily leads to the use of such expressions in a more precise signification, and hence to a greater or less amount of divergence from the older and more popular usage. "Heredity" is, for instance, most usually defined by biologists as

referring generally to all phenonema covered by the aphorism "like begets like." In this sense it denotes *inter alia* the phenomenon of the constancy of specific or racial types and of sexual characters; a character may be said to be *inherited* when it always, in one generation after another, is one of the characters of the species, of the race, or of the one sex of the race, as distinct from the other. The species, race or sex, so to speak, "begets its like" as a whole. But then a further question remains; even if the type of the race is constant, do *individual* types within the race beget their like? In so far as any *individual* diverges in character from the mean of the race do his offspring tend to diverge in the same direction, or not? It is to this question that statisticians have confined themselves, and they speak of a character being "inherited" or not according as the answer to the question is yes or no—they deal solely with what we may term "*individual* heredity."

The quantitative procedure is in its essence extremely simple, though the actual work may often be rather lengthy. A series of measurements is made of some one variable character, *e.g.* a length, in parents and in their offspring, noting the individual families (the more the better) and not merely measuring the first generation as a whole and then their offspring as a whole. From these measurements an equation is derived, giving, as nearly as may be, the mean character of the offspring in terms of the character of the parent. Supposing X to be the character in the parent, Y the mean character in the offspring, then the simplest form of such equation is:—

$$Y = A + B.\ X. \qquad (1)$$

Where A is a dimension of the same order as X or Y, and B is a number that will vary from case to case. We have for instance from the data collected by Mr. Galton for inheritance of stature in man, reduced by Professor Pearson, the equation relating mean stature of sons and stature of father:—

$$Y = 31{\cdot}10 + {\cdot}45\ X. \qquad (2)$$

i.e. the mean stature of sons is 31·1 inches, together with nine-twentieths of the stature of the father (also in inches of course). The father's stature is thus some guide to the stature of his offspring; it enables us to form a closer estimate of their stature than we could from a mere knowledge of the mean characters of the race, and we may therefore say that stature is an *inherited* character. The sons do diverge from the race-mean in the same

direction as their parent. Quite generally, the statistician speaks of a character as *inherited* whenever the number or "constant" B is greater than zero; if it does not differ sensibly from zero the character is held to be non-heritable, quite apart from the question whether the mean is more or less constant from one generation to the next, a consideration which does not affect the conception of *individual* heredity.

It is important that the biologist should realise this distinction between individual heredity and race-heredity, or as I should prefer to term it, constancy of type; for, although the two phenomena must be in some way related through the processes of reproduction and growth, approximate constancy of type is not only logically, but, I believe, actually quite compatible with very slight individual inheritance, the type being maintained the same by the constancy of external conditions and the action of selection. Moreover, and this is the most important point, the maxim "like begets like" does not hold in the same rigid sense for the individual and the race. One generation of a race is (approximately speaking) the same as the preceding; apart from such changes as are hardly revealed except by measurement, the mean of the offspring is that of the parents. It would seem natural perhaps to assume that the same law holds for individual types within the race—why should an isolated group of individuals behave differently from the race as a whole? Yet the assumption would be false; the offspring of any abnormal individual, any individual differing from the mean of the race, *are always, on the average, more mediocre than himself.* In the terms of our "estimating equation" (1), this means that the constant B is always less than unity—always a fraction, If the offspring simply centred round the parental type we should have

$$Y = X$$

always. For stature in man the value of B is about 0·3 to 0·5; for a certain character in *Daphnia* Warren found B = 0·6, for other characters in an *Aphis*, 0·54, 0·5, and 0·36, the reproduction being parthenogenetic in both these cases; for vegetative reproduction in *Lemna minor* I find, so far as the results are reduced, B = 0·25 to 0·60, roughly speaking. This phenomenon of the relapse of the offspring from the parental type towards mediocrity is termed *regression.* Regression and not constancy of type is for the statistician, the fundamental phenomenon of heredity and the prime fact to be explained by any physical theory. The absolute lack of any mention of the subject in most biological theories makes them seem, to him, in so far, curiously unreal.

It is not essential for the application of such "estimating equations" as (1) or (2) to the study of heredity that the variation of the character concerned should be strictly continuous as in the case of stature in man. The method is equally applicable to any form of variation in a scalar series, even when such variation proceeds by a series of discrete steps. The variation of petals, sepals, and other floral parts, and of numbers of offspring, may be quoted as familiar instances. Even in such cases as stature, discontinuity is, in point of fact, nearly always introduced by the observations being grouped, or the measurements being only taken to some considerable unit of the scale, *e.g.* to the nearest half-inch or quarter-inch. Between real continuity—a continuity that appears to be unbroken with the most careful measurements possible—and the discontinuity of a scalar series proceeding by successive equal units there is therefore no important distinction. The same method and conceptions apply, the same law of regression must hold.

A distinction arises, however, if no scalar series exists, but the race is simply divided into two exclusive classes, the one possessing some attribute, the other not; as one may divide a race of men into deaf-mutes and normals, sane and insane. In such a case the statistician again speaks of the attribute as being *inherited*, if the character of the parent enables one to estimate the character of the offspring more accurately than would be possible from a mere knowledge of the general characters of the race. If the two classes be termed A's and a's, then the attribute is *inherited* if the per- of A's amongst the offspring of A's is larger than the per centage amongst the offspring of a's. This is, as before, *individual* heredity. When the biologist speaks of the transmission of an attribute common to all the members of a race as *heredity* (as the flowers of one race of plants may be white, those of another pink; the stems of one glabrous, those of another hairy), he is dealing with a quite distinct aspect of the phenonemon. I do not, of course, object to such an accepted use of the term, but wish to emphasise the distinction,

Whether, in fact, we deal with continuous variables or attributes, all the individuals observed, in any one case, must be members of one race, or else the two phenomena of race-heredity and individual heredity are superposed and confused. If, for instance, stature-measurements on a tall race and a short race were mixed, it is conceivable that there should be no individual heredity

for either race taken separately, and yet the mixture would exhibit an apparent heredity due simply to the constancy of type for each race. It would be true for the mixture that the taller parents had the taller offspring, but not true for either race separately. The same thing holds good in the case of attributes. If two races be mixed, of which a large per-centage of individuals in the one case and only a small per-centage in the other, possess some attribute, it will be true for the mixture that the offspring of *A*'s exhibit a larger proportion of *A*'s than the offspring of *a*'s; but this will not necessarily be true for either race separately. The two classes *A* and *a* belong, I take it, to one race when pure matings of of *A's* with *A's* may give rise to *a*'s and vice-versâ. In the case of a continuously-variable character, all the individuals may be held to belong to one race if they cannot be divided into two classes such that pure matings between members of the one class never give rise to offspring that would be assigned to the other. The distinctions between continuity and discontinuity of variation, between inheritance of attributes and of variables do not seem to me to be of *necessary* importance for the theory of heredity; successive discontinuities may be so slight as to be undiscoverable by the most careful and repeated measurements. The real and important distinction seems to lie between the phenomena of *heredity* within the race, and the phenomena of *hybridisation* that occur on crossing two races admittedly distinct. Several of the investigations of Mr. Galton relate to the inheritance of attributes (*e g.* of temper, of artistic faculty in man, of colour in Basset-hounds), none of them (so far as I am aware) to hybridisation. It does not seem probable that either he or Professor Pearson intended the term heredity to cover such cases; I can certainly say for myself that in stating any rule to be a "law of heredity," I should not dream of implying thereby that it was a law of hybridisation.

Mr. Bateson fails to make any distinctions whatever. Mr. Galton's researches on Basset-hounds and the Galton-Pearson work on eye-colour in man (both referring to individual heredity within the race) are classed, under the general heading of "discontinuous variation," with Mendel's work on crossing distinct races of peas and Mr. Bateson's own on hybridisation of flowers and races of poultry, as if they referred to comparable matter. This does not tend to clearness. Mr. Bateson further adduces the work of *hybridisers* to rebut the generality of the Galton-Pearson law of *heredity*:—

"Not to mention moderns, these high hopes" (of the generality of the law) "had been finally disposed of by the work of the experimental breeders such as Kölreuter, Knight, Herbert, Gärtner Wichura, Godron, Naudin, and many more. To have treated as non-existent the work of this group of naturalists, who alone have attempted to solve the problems of heredity and species—Evolution as we should now say—by the only sound method—*experimental breeding*—to leave out of consideration almost the whole block of evidence collected in *Animals and Plants*—Darwin's finest legacy as I venture to declare—was unfortunate on the part of any exponent of Heredity, and in the writings of a professed naturalist would have been unpardonable." (Mendel's Principles, pp. 112-13).

The "experimental breeders" referred to never touched the questions of intra-racial individual heredity at all. The work of Darwin bears chiefly on racial heredity, and on hybridisation; the relatively small portions on intra-racial heredity do not give information in any form which enables one to apply it to the criticism of statistical laws. In the present state of our knowledge it is impossible to confuse the subjects in so loose a fashion. Laws of hybridisation cannot be admitted as general principles of heredity until they have been proved to hold as such—the title of Mr. Bateson's volume begs the whole question—nor can laws of heredity be in general, or necessarily, expected to hold good in cases of hybridisation. "Experimental breeding" is certainly a sound method for the study of heredity: it is not the *only* sound method, for equally good material may be obtained by simple observation, as in the case of man. But if by "experimental breeding" is meant solely hybridisation (*i.e.* crossing of different races, varieties, species or genera in general), then I join issue with Mr. Bateson altogether. Experiments on crossing can give nothing but laws of crossing; it may be possible that some of these laws are applicable to the breeding of pure races, but this cannot be decided without definite trial. The work of the whole of the "group of naturalists" he mentions is valueless for the branch of work on which the biometrical school has been engaged.

So far we have dealt solely with the direct heredity between parent and offspring; now let us consider the question of inheritance from the remoter ancestry. Supposing a series of grandparents and their grandchildren to be measured as before, it is evident that we could construct an "estimating equation" just like (1), but giving the mean character of the *grand*children in terms of the character

of the *grand*parent. The only difference in result would probably be, that the constant B would be somewhat reduced as compared with its value for the parental heredity. If an individual have a given abnormality of character, his offspring will *probably* (or on the average) be abnormal, but rather less so, his grand offspring again divergent, but less abnormal still. This phenomenon, that the character of the grandparent (like that of the parent) enables one to estimate the mean character of the offspring more accurately than would be possible from a mere knowledge of the characters of the race, is, in a sense, "ancestral heredity." It is not, however, what the statistician generally means by that term. In the above, the parent's character is supposed either unknown or neglected; we deal solely with grandparent and grandchildren. But supposing the character of the parent known, so that one datum for estimating the mean character of offspring is already given, a wholly new question arises, *viz.* will a knowledge of the grandparent's character enable one to increase the accuracy of estimate? If the answer to the question be in the affirmative, as it is in every case without exception which has yet been tried, then there is what may be termed a *partial* heredity from grandparent as well as parent, and it is to the existence of such *partial* heredity that statistical writers generally refer when they speak of "ancestral heredity." If X_1 and X_2 be the parental and grandparental characters, Y the mean character of the offspring, then all the experience that we have shews that if an equation be formed giving Y as nearly as may be in terms of both X_1 and X_2, *e.g.*

$$Y = A + B_1 . X_1 + B_2 . X_2. \qquad (3)$$

the term B_2 has a very sensible value—*i.e.* the grandparent's character very sensibly increases the accuracy of estimate. This law of partial heredity from the grandparent is known to hold for fertility, length of life, and eye-colour in man, for coat-colour in horses, for one character in a *Daphnia*, three characters in an *Aphis*, and I may add, from some recent work of my own, two or three characters in common duckweed (*Lemna minor*). The list is not a long one certainly, but the characters and the genera are so extraordinarily diverse that the law must be one of very great generality.

Nor, of course, need we suppose investigations to cease with the grandparent. If a knowledge of the grandparental character increases the accuracy of estimate of the mean character of offspring, it is natural to assume that the further knowledge of the great-grandparental, great-great-grandparental, etc., characters would

increase it still more, though probably in a diminishing ratio. It would be very unlikely, to say the least, that an absolute discontinuity should come in at any one generation, and that the nth ancestor should sensibly increase the accuracy of estimate while the $(n + 1)$ th should not. In the absence of such discontinuity the law of heredity (of *intra-racial individual heredity*) must be of the form

$$Y = A + B_1 . X_1 + B_2 . X_2 + B_3 . X_3 + B_4 . X_4 + \ldots \quad (4)$$

where X_1 X_2 X_3, etc. are the characters of the successive ancestry and B_1 B_2 B_3, etc. a series of diminishing fractions. No investigations, so far as I am aware, have been published, that give the values of B's beyond the grandparents, but my own experiments on a small scale on *Lemna minor* give sensible positive values up to B_4—as far as I could carry the observations. Moreover such a form of law is in obvious accordance with the practice of breeders of pure stock, who judge the value of an animal for breeding purposes not by its own characters alone, nor by that and the characters of its immediate parents, but by its whole pedigree. The pedigree would be completely valueless if there were no "partial heredity" from ancestry; if B_2 B_3, etc. were all zero two animals with equally desirable values of X_1 would be equally likely to produce good offspring, even if the one had bad or mediocre ancestry and the other a good pedigree. It is difficult to suppose that the weight attached to pedigree is based on nothing but illusion, yet it is only reconcilable with a law of partial ancestral heredity such as (4). This law then, that *the mean character of the offspring can be calculated with the more exactness, the more extensive our knowledge of the corresponding characters of the ancestry*, may be termed the Law of Ancestral Heredity.

Little work has yet been done on the intra-racial inheritance of attributes, but the form which the law of ancestral heredity would take in such a case is fairly obvious. It might be written in the form (to use as nearly as possible the same words)—*the percentage of A's and a's amongst the offspring can be calculated with the more exactness, the more extensive our knowledge of the corresponding characters in the ancestry*. For instance, one may presumably take it for a fact that the percentage of insane amongst the offspring of the insane is larger than amongst the offspring of normal individuals *i.e.* insanity is inherited. Does the law of "ancestral heredity" hold? If we assume that it does, we are assuming that the percentage of insane amongst the ultimate offspring is greater and greater the more of the ancestry were insane; if we assume on

the contrary that it does not, we are supposing that it is completely indifferent whether the ancestry beyond the *immediate* parents were normal healthy individuals or raving lunatics. For the working out of an actual case the reader may be referred to Mr. Galton's paper on the inheritance of colour in Basset-hounds.

It may be as well to point out that the law of ancestral heredity by no means implies that if the characters of all the ancestry are known, the estimate of the character of the offspring becomes an exact determination, That can never be the case, for the offspring of one individual, or of one pair, exhibit a range of variation as a rule only fractionally less than that of the race, although they all have the same ancestry. In point of fact the accuracy of estimate will sensibly, though not rigidly speaking, reach a limit after relatively few of the ancestry (say to the sixth generation or so) are known. Further it should be noted that the law of ancestral heredity is quite distinct from the alleged law of the prepotency of the phylogenetically older character. Apart from the fact that the latter is usually applied to cases of the crossing of distinct races with which the former has nothing to do, and to cases where the relative ages of the characters are measurable by geological time, while the former gives *sensible* weights only to the recent ancestry, the law of ancestral heredity is not a law of prepotency or dominance at all. The whole of a man's ancestry on the female side might be sane; only the last six generations on the male side insane. The law does not state that the offspring will probably be sane, sanity being the phylogenetically older character. Such a statement would be absurd. All that it affirms is that the more of the ancestry are insane, the greater is the chance of insanity appearing in the offspring—a very different thing. I can see no justification whatever for the confusion of the two laws by Mr. Bateson. Professor Weldon, in the *Biometrika* article referred to (January, 1902), states the law of ancestral heredity in these terms, "The degree to which a parental character affects offspring depends not only upon its development in the individual parent, but on its degree of development in the ancestors of that parent." Apart from Professor Weldon's use of the word "affects" which to some extent implies a direct physical influence, and for which I would prefer to substitute some such phrase as "indicates" or "serves as a basis for estimating the character of," this law is, beyond question, as we have seen, of very general application. Yet Mr. Bateson comments on it in the following terms: "Having rehearsed this pro-

fession of an older faith Professor Weldon proceeds to stultify it in his very next paragraph. For here he once again reminds us that *Telephone*, the mongrel pea of recent origin, which does not breed true to seed characters, has yet manifested the peculiar power of stamping the recessive characters on its cross-bred offspring, though pure and stable varieties that have exhibited the same characters in a high degree for generations have *not* that power." I quote the passage in full as a characteristic example of Mr. Bateson's method. Mr. Bateson endeavours first, *more suo*, to discredit the statement of the law by referring to it as a "profession of faith," and then remarks that it is "stultified" by another fact with which it has nothing to do. The strength of Mr. Bateson's reasoning is hardly equal to that of his language.

I have, in the preceding passages, used the term "Law of Ancestral Heredity" in a sense somewhat wider than that given by Professor Pearson. Mr. Francis Galton, some years since, put forward a formula, subsequently very considerably modified by Professor Pearson, suggesting or implying certain fixed values for the constants B_1 B_2 B_3, etc. (equation 4) or at least fixed relations between them. This law, Galton's Law, or one of its modifications, has been frequently referred to by Professor Pearson as "*The* Law of Ancestral Heredity." I have ventured to drop that signification, as I do not think the facts indicate any fixity of formula even for intra-racial heredity, a point in which I agree with Mr. Bateson, though all his evidence adduced from hybridisation seems to me quite beside the mark. Being unable to accept Mr. Galton's law as *a* law of heredity, *à fortiori* I cannot accept it as *the* law, and have therefore applied the phrase to a more general statement. Mr. Galton states his law in the form that "the two parents contribute between them on the average one half or 0·5 of the total heritage of the offspring, the four grandparents, one quarter, or $(0{\cdot}5)^2$; the eight great-grandparents, one eighth, or $(0{\cdot}5)^3$ and so on." "The theory" says Mr. Bateson, after some other comments with which I do not deal only on account of their less importance, "further demands—and by the analogy of what we know otherwise not only of animals and plants, but of physical or chemical laws, perhaps this is the most serious assumption of all—that the structure of the gametes shall admit of their being capable of transmitting any character varying from zero to totality with equal ease . . . ." The comment does not hold good at all. Mr. Galton's law is only stated as an average or statistical law, and the "one quarter" contributed

by the grandparents *on the average* might be made up by some contributing one half, and others contributing nothing; the average of a series of quantities may exhibit sensible continuity of variation, even though the quantities averaged vary by discrete steps. There is no difficulty for instance in applying the general law of the form (4) to such a case as inheritance of number of petals in flowers, although the coefficients B_1 B_2 B_3, etc. are all fractional; Y only gives the *average* number of petals in the offspring which of course will not be in general a whole number. To the bearing of the analogy of physical and chemical laws I will return later.

A real difficulty in the acceptance of Galton's law as stated in this form lies in the conception of the "heritage," and of ancestry "contributing" thereto, two conceptions of which I find it difficult to grasp the exact meaning. As I know that others have felt a similar difficulty, it may be as well if I point out that the law of regression (the shifting of the offspring from the parental type towards mediocrity) and the law of ancestral heredity are both susceptible of a very simple physical explanation on totally different lines. Both laws, it should be remarked, are known to hold for a sexual reproduction, so that any explanation founded solely on the hypothesis of gamogenesis is necessarily inadequate. I will therefore only consider in this rough indication the first and simplest case. Two assumptions only are necessary. (i.) The continuity of the germplasm, or the central idea of that theory, *i.e.* the conception of the soma and germ cells as separate out-growths from the fertilised ovum; the germ cells and soma not being so intimately related that an alteration in any one cell or group of cells in the soma can produce so specific a change in the germ cells that they tend to produce offspring with a corresponding alteration. (ii.) The assumption that the characters—structure or whatever we please to term it—of the germ cell cannot *rigidly* determine the characters of the resulting soma, owing not merely to the variations in definite and assignable external circumstances, but to that residuum of unanalysable variations which we term chance. I do not now propose to justify these assumptions, if they need justification, but merely to point out their consequences. It follows directly from the second assumption that a series of absolutely identical germ cells will not produce a series of absolutely identical individuals, but a group or *array* of individuals differing more or less *inter se*. The somatic character of an individual is not therefore an absolute guide to the character of the ovum from which he sprang nor, *à fortiori*, to the

mean character of the germ cells which he produces. If we conceive a number of individuals of the same somatic type, some will be abnormal developments of mediocre germ cells (*i.e.* cells producing on the average mediocre types); others, but these will be fewer, will have sprung from germ cells producing on the average, or usually, more abnormal types. The odds are, therefore, that a given abnormal somatic type is an abnormal development of a mediocre germ cell rather than a mediocre (or subnormal) development of an abnormal cell. But the somatic characters of offspring will follow the germ cell characters and not the somatic characters of the parent; therefore *regression* occurs. Further, as the line of germ plasm is handed on from parent to offspring and the processes of nutrition and growth, if continued unchanged, need not be supposed to alter the *average* character thereof, any one individual in a line is an index to the character of the plasm in that line. Hence if ancestry as well as parents be abnormal it is more probable that the parents are an average development of a really abnormal type of germ cell, and hence more probable that the offspring will follow, and not regress from, the parental type, *i.e.* we have "ancestral heredity."

I have put the whole matter as briefly as possible, without discussing details, because I wish at present simply to emphasise the fact that although the theory of ancestral contributions to a heritage implies the law of ancestral heredity, the converse is not true: the law of ancestral heredity need not in any way imply actual physical contributions of the ancestry to the offspring. The ancestry of an individual may serve as guides to the most probable character of his offspring simply because they serve as indices to the character of his germplasm as distinct from his somatic characters.

The same line of argument applies in the case of attributes. The germ cells cannot in general be treated as if they rigidly determined whether the individual should be an A or an a, the one type of cell merely produces a *majority* of A's, the other a majority of a's—as a loaded die tends to fall the more often on the one face than on the others; only in the extreme case the loading might be so heavy that the die would always rest on the one face. In general then one could not be certain that the germ cells of an A individual (reproducing asexually, to avoid complications) were of the A-type, *i.e.* the type producing a majority of A's; they might be or might not. If, however, the parent of the A-individual were also

of the *A*-type, and *à fortiori* if the grandparent were also, and so forth, the germ cells of the *A*-individual would much more certainly be of the type his somatic characters would lead one to expect.

These considerations will, I believe, be useful in considering Mendel's Laws themselves, and their relation to the Law of Ancestral Heredity. I have, I hope, said enough to shew the reader that however the two may be related they cannot at least be "absolutely inconsistent" with each other, as Mr. Bateson contends. The law of ancestral heredity is certainly a law of nature of wide generality which cannot be dismissed in such a fashion. Mendel's Laws I assume to be true also. The problem is to delimit their respective spheres, and shew in what way the one type of law may pass into the other, or the two even coexist.

(to be continued.)

MENDEL'S LAWS AND THEIR PROBABLE RELATIONS TO INTRA-RACIAL HEREDITY.

By G. Udny Yule.

(Continued from page 207.)

On passing from the Law of Ancestral Heredity to Mendel's Laws, we are passing from a law of intra-racial individual heredity to a series of laws based solely on hybridisation-experiments, and clearly stated by their discoverer as laws of hybridisation only. The experimental plants must, Mendel states (I quote from Mr. Bateson's translation, "Mendel's Principles" p. 42) "possess constant differentiating characters." After trying thirty-four varieties of peas he found that while one exhibited some aberrant individuals, "all the other varieties yielded perfectly constant and similar offspring; at any rate, no essential difference was observed during two trial years." The thirty-three "constant" varieties being obtained "for fertilisation, twenty-two of these were selected and cultivated during the

whole period of the experiments. They remained constant without any exception." The races for crossing were thus chosen with the greatest care and patience so as to be absolutely distinct; an *A* individual mated with an *A* never producing *a*'s, nor vice-versâ, for, as I understand, the whole period of ten years (two years of preliminary trials and eight years of experiment.)

Mendel's observations, as most of the readers of these articles will be aware, revealed several distinct uniformities, the verbal description of any one of which might be legitimately entitled a "Mendel's Law." When the two parent races *A* and *a* were crossed, their mongrel or hybrid offspring resembled uniformly either the *A*'s or the *a*'s—say the former, in which case *A* was termed the dominant character, *a* the recessive. When these first uniform hybrids were crossed *inter se*, the resulting offspring were no longer uniform but broke up again into two classes resembling the parent races—approximately three-fourths exhibiting the dominant character and one-fourth the recessive. The whole of these "extracted" recessives—to use Mr. Bateson's convenient terminology—breed pure, *i.e.* never give rise again to the dominant form. Of the dominants, however, only one-third breed pure, the remainder giving rise again to dominants and recessives in the proportion of three to one. These remarkable results Mendel explained by a hypothesis which certainly appears, for the case of a *single* character, of most attractive simplicity. He supposed that a sorting process takes place during the formation of the germ cells of the *Aa* hybrids, of such a nature that each finally formed gamete, without exception, only contains the germinal representative of either the *A*-character or the *a*-character, and that conjugation between *A*-gametes and *a*-gametes takes place at random. The result is that the zygotes *AA*, *Aa*, *aA*, and *aa* are all formed with equal frequency. But the heterozygotes (to use Mr. Bateson's terminology again) both give rise to individuals resembling the pure dominant form. The offspring therefore exhibit three dominant forms (one pure and two hybrids) to one recessive (necessarily pure).

This law of disjunction, or "segregation," Mendel tested also for pairs and triplets of characters. The results obtained for one character alone leave it open to question whether the group of characters (represented by the germ-plasm, chromatin or whatever it may be) derived from the one parent, has separated bodily from that derived from the other, or whether any more

complex process has taken place. The latter Mendel found to be the case; two races, *ABC* and *abc* being crossed, and the hybrids bred *inter se*, the resulting offspring exhibited not merely the parent forms but all the eight possible combinations of somatic characters—*ABC*, *aBC*, *AbC*, *ABc*, *abC*, *aBc*, *Abc*, and *abc*, some being pure and some hybrid forms, as before. These re-combinations of characters and the approximate proportions in which they occurred Mendel held to indicate that each somatic character observed has some distinct and separable germinal representative—"Anlage," determinant or physiological unit, as we might term it—and that the formative divisions of the germ cells always take place in such a way that the finally formed gametes are homogeneous with respect to each character of a pair—no cell containing any *pair* of determinants like *Aa* or *Bb*—but otherwise at random. He experimented on one pair of characters in *Pisum*, form of seed (round or angular) with cotyledon-colour (yellow or green), and on one triplet, seed-coat colour (grey-brown or white) in addition to the above, and the law held in both cases with reasonable accuracy. Mr. Bateson and Miss Saunders give in the *Report* particulars of a few experiments on pairs of characters, *viz.*, colour of flower (violet or white) with character of fruit (smooth or prickly) in *Datura*, character of comb (rose or single) with extra toe in crosses of Leghorn with Dorking fowls, and colour of plumage with form of comb (pea or single) in crosses of Leghorns with Indian Game Fowls. The numerical proportions obtained by the experimenters in these cases diverge more or less (most notably in the second case) from those to be expected on Mendel's hypothesis, but there is no denying the re-combinations of characters that have taken place; starting for instance with breeds of poultry exhibiting rose comb with extra toe and single comb without extra toe, respectively, two new breeds were obtained exhibiting *single* comb with extra toe and *rose* comb without.

Mendel's hypothesis is so ingenious and remarkable, and possibly of such far-reaching importance, that one can understand Mr. Bateson speaking of it as the "essential part" of his discovery, to the complete exclusion of the law of dominance and the various laws of numerical proportions which summarised the *facts* observed. These last two laws obviously enough do not hold in many cases. If they are excluded as not "essential," the "top-hamper," to use Mr. Bateson's nautical metaphor applied to another matter, "is cut

down and the vessel altogether more manageable; indeed she looks trimmed for most weathers."

These then are the facts and the hypothesis the relations of which to the phenomena of intra-racial heredity have to be considered and discussed. The first question to be asked in such a discussion, is one that does not seem to have occurred to any of Mendel's followers, *viz.:* what, exactly, happens if the two races *A* and *a* are left to themselves to inter-cross freely *as if they were one race?* It must be remembered that it is only the knowledge given by preliminary trials like those carried out by Mendel which enables us to state that the races *are* distinct; a man who was merely given a sample of seed from the dominant forms occurring after hybridisation had taken place, would conclude that they were not so—he would find that *A*'s crossed *inter se* gave rise to some *a*'s, though not indeed the reverse, and would therefore class the two forms as springing from the same stock. Now when *A*'s and *a*'s are first inter-crossed we get the series of *uniform* hybrids; when these are inter-bred we get the series of three dominant forms (two hybrids, one pure) to one recessive. If all these are again intercrossed at random the composition remains unaltered. "Dominant" and "recessive" gametes are equally frequent, and consequently conjugation of a "dominant" gamete will take place with a "recessive" as frequently as with another "dominant" gamete. Consider then the successive generations of posterity of the dominant forms, starting, say, with 300 of which 100 are pure. The 100 pure individuals will give rise to dominant forms in the proportion of 50 pure to 50 hybrids; the 200 hybrids may, as segregation takes place, be considered as 100 pure dominants and 100 pure recessives, the former giving rise to 50 pure dominants and 50 hybrids, the latter to 50 hybrid dominants and 50 *pure recessives*. The 300 parent dominants, therefore, give rise to offspring in the proportion of 250 dominant forms to 50 recessive, *i.e.*, the chance of a dominant parent producing a dominant form as offspring is $\frac{5}{6}$. Now consider these 250 dominants whose parents were dominants also, 100 of them being pure and 150 hybrids. The 100 pure dominants will give rise as before to 50 pure and 50 hybrid forms, the 150 hybrid forms to pure dominants, hybrid dominants and pure recessives in the proportion of 37·5 : 75 : 37·5. Five hundred dominants whose parents were dominants should therefore produce 425 dominant offspring to 75 recessives, *i.e.*, the chance

of their producing a dominant form would be $\frac{425}{500} = \frac{17}{20} = \frac{51}{60}$, while the chance of a dominant of unknown parentage producing a dominant form is only $\frac{50}{60}$. But this is precisely a case of the law of ancestral heredity! It is not difficult to continue the calculations on the same simple lines, but the work may be abbreviated by the following considerations. Let T_n denote the total number of dominants in the nth generation all of whose ancestors in one line are also dominants, and let p_n of the T_n be pure, i_n impure or hybrids. Then quite generally one-half of the pure dominants and one-quarter of the hybrids of any generation give rise to pure dominants as offspring, while the remaining half of the pure dominants and one-half of the impure give rise to hybrid forms. That is in symbols

$$p_{n+1} = \tfrac{1}{2} p_n + \tfrac{1}{4} i_n \qquad (5)$$

$$i_{n+1} = \tfrac{1}{2} p_n + \tfrac{1}{2} i_n = \tfrac{1}{2} T_n \qquad (6)$$

Adding the two equations together

$$T_{n+1} = T_n - \tfrac{1}{4} i_n \qquad (7)$$

or by equation (6)

$$T_{n+1} = T_n - \tfrac{1}{8} T_{n-1} \qquad (8)$$

Dividing out on both sides of this equation by T_n and writing

$$C_n = \frac{T_{n+1}}{T_n} \qquad (9)$$

where C_n is the chance of a dominant form of the nth generation producing dominant offspring, we have finally

$$C_n = 1 - \frac{1}{8\, C_{n-1}} \qquad (10)$$

an equation which enables us to calculate the remaining chances very easily, given that $C_1 = \frac{5}{6}$. I find

$$C_1 = \cdot 83333$$
$$C_2 = \cdot 85000$$
$$C_3 = \cdot 85294$$
$$C_4 = \cdot 85345$$
$$C_5 = \cdot 85354$$

where, as is found by equating C_n to C_{n-1} in equation (10) C tends towards the limiting value ·85355339... The figures illustrate as nicely as could be desired the two chief properties of Ancestral Heredity—(i.) the chance of an *A* producing an *A* is increased if the ancestry be also *A's*. (ii.) it is not of much use to take into account more than the first few generations of ancestry (cf. p. 203 supra), for the chance C rapidly approaches a limiting value.

Mendel's Laws, so far from being in any way inconsistent with the

Law of Ancestral Heredity, lead then directly to a special case of that law, for the *dominant* attribute at least. For the *recessive* attribute it does not hold, the chance of a recessive producing a recessive offspring is, on the above hypothesis of random mating, one-half, whatever the parentage may be. Nor is it difficult to see why the law applies in the former case. *Ex hypothesi* pure dominant and hybrid zygotes produce dominant forms indistinguishable the one from the other, so that the somatic characters of the individual are not an absolute guide to the character of his germ cells—they may or may not be of the pure dominant type even though his soma be of the dominant form. If, however, the parent, grandparent, etc., be of the dominant form also, the absence of recessive individuals in the ancestry gives a stronger and stronger presumption that the germ cells are of the type which the somatic characters of the individual would lead one to expect. It is equally easy to see why the law does *not* hold in the case of the recessive attribute. *Ex hypothesi* again, recessive forms can only be produced by pure recessive germ zygotes; it is therefore *certain* without any further witness that the germ cells produced by recessive individuals must be themselves recessive—knowledge of the ancestry is useless for predicting the nature of the germ cells of such an individual, and therefore equally useless for predicting the nature of his offspring. Mendel's Laws, in implying "that the cross-breeding of parents *need* not diminish the purity of their germ cells or consequently the purity of their offspring" (Mendel's Principles, p. 114), do *not* assert, as stated by Mr. Bateson, "a proposition absolutely at variance with all the laws of ancestral heredity *however formulated*" (my italics). Purity of germ cells may very well subsist for a proportion of the individuals of a race without in any way invalidating the principle of the Law of Ancestral Heredity, in the sense defined; it is a law applying to aggregates and predicates nothing concerning the individual. The value of the work of Mendel and his successors lies not in discovering a phenomenon inconsistent with that law, but in shewing that a process, consistent with it, though neither suggested nor postulated by it, might actually occur.

The form of the law of ancestral heredity to which Mendel's principles have led us is, however, clearly a special case, and the next question to be asked is therefore this:—in what way may the special conditions under which Mendel's Laws hold good be broadened so as to permit of a generalisation of the results? Two of these conditions suggest themselves at once as being in all probability somewhat exceptional in character, *viz:* (i.) the necessity

for *dominance*, or, to define it in a somewhat more general sense than that used by Mendel, the condition that the hybrid zygote *Aa* should always behave, as regards the production of the attribute noted, as if it were a pure zygote of the one race, say *A*; (ii.) the necessity for the given somatic attributes being rigidly *predetermined* by the characters of the gametes, and not liable to such fluctuations owing to the variations of circumstance or otherwise that an individual of the pure race *A* might be classed as an *a* or vice versâ. As pointed out already (p. 206) this condition cannot universally hold good. To take an example from the inheritance of disease, the chances of an individual dying of phthisis depends not only on the phthisical character of his ancestry, but also very largely on his habits, nurture, and occupation. If, however, either dominance fail, or the rigid predetermination of the somatic attributes by the germ cell, or both, Mendel's Laws will cease to hold, but the Law of Ancestral Heredity will still apply.

Suppose first that dominance fails, and, to take a rather interesting case, suppose the failure to be complete, *i.e.* assume the heterozygotes on development exhibit *A* characters and *a*-characters with equal frequency. Then when the two forms are first crossed the resulting offspring, hybrid without exception, will exhibit both attributes with equal frequency. When these hybrids are crossed *inter se*, the offspring will again exhibit both characters with equal frequency, but one-half of both forms will be pure, the other half hybrids. All succeeding generations after this will be the same.

Consider then the offspring of say 400 *A*'s. Mating being random, as before, conjugations of an *A*-gamete with an *A*-gamete and with an *a*-gamete will be equally frequent. Of the 200 pure *A*'s, 100 mate with *A*'s and produce 100 pure *A*'s; 100 mate with *a*'s and produce 50 hybrid *A*'s and 50 hybrid *a*'s. The two hundred hybrid *A*'s may be treated separately as 100 pure *A*'s and 100 pure *a*'s. The former give rise to 50 pure *A*'s, 25 hybrid *A*'s and 25 hybrid *a*'s, the latter to 25 hybrid *A*'s, 25 hybrid *a*'s and 50 pure *a*'s. Adding up, the four hundred *A*'s give rise on the whole to 250 *A*-forms (150 pure, 100 hybrid), and 150 *a*-forms (100 hybrids, 50 pure). The chance of an *A* producing an *A* is therefore $\frac{250}{400} = \frac{5}{8}$. The chances of an *A* whose parent, parent and grand-parent, and so on are *A*'s producing an *A*-form as offspring are most easily calculated by the method of equations (5) — (10). To use the symbols of those equations let T_n denote the total number of *A*-individuals of the *n*th generation, all of whose ancestors in one line are also *A*'s, and let p_n of these be pure, i_n impure. Then we have in the present case:—

$$p_{n+1} = \tfrac{1}{2}\, p_n + \tfrac{1}{4}\, i_n \qquad (11)$$

$$i_{n+1} = \tfrac{1}{4}\, p_n + \tfrac{1}{4}\, i_n = \tfrac{1}{4}\, T_n \qquad (12)$$

whence, as before, if C_n be the chance of an A-individual, all of whose n ancestors in one line are A's, producing an A-form as offspring

$$C_n = \tfrac{3}{4} - \frac{1}{16.\ C_{n-1}} \qquad (13)$$

This equation gives the following figures for the successive chances :—

$C_1 = \cdot62500$
$C_2 = \cdot65000$
$C_3 = \cdot65385$
$C_4 = \cdot65441$
$C_5 = \cdot65449$

where again the value of C tends towards a fixed limit ·6545085 ... obtained by writing C_n for C_{n-1} in equation (13) and solving. As we have assumed the complete absence of dominance in the above case, the values of C given will apply to both a's and A's. Had we assumed only a partial failure of dominance, supposing, *e.g.* the heterozygote to give rise to 80% of A's and 20% of a's, the law of ancestral heredity would still have applied to both forms, but the two series of chances would have been different. The failure of dominance thus generalises the forms of the law of ancestral heredity derivable from Mendel's principles in two different ways (i.) by rendering it possible to obtain any arbitrary value for C_1, (ii.) by rendering the law applicable to a's as well as A's.

I have, of course, in the preceding, assumed random mating merely to simplify the work. If the mating were homogamic, A's only mating with A's, and a's with a's, as would generally be the case if the breeder wished to obtain as nearly as possible pure races of A's and a's, the law of ancestral heredity would still apply. The working would, however, be a good deal complicated, for the proportion of pure forms and hybrids amongst the A's would vary from one generation to the next.

Next suppose the absolute predetermination of the somatic attributes by the germ-cell to fail, pure zygotes of the one type producing not only A's, but also a proportion of a's, and pure zygotes of the other type, not only a's, but also a proportion of A's. At the same time, to keep the case fairly simple, let dominance in a generalised form still hold good, the heterozygote behaving, as regards the proportions of the two forms produced, precisely as if it were a pure homozygote of one or other type. Let the two classes of gametes be, say, B's and b's, pure B-zygotes producing 70% of

A's, pure *b*-zygotes 35 % of *A*'s, and let the *B*-gamete be dominant over the *b*-gamete in the sense that the *Bb* zygote produces 70% of *A*'s just as if it were a pure *BB*-zygote. Now, the assumption of the break-down of "predetermination" brings in a new element; we have not only to consider *A*'s developed from the dominant *BB*-zygotes and hybrid *A*'s developed from the heterozygotes but also *A*'s that have developed *from the pure recessive bb-zygotes*, "aberrant recessives" as they may be termed. In assuming the race, as before, to have reached the steady state, in the second generation after the first cross there will again be pure *B*'s, *Bb*-heterozygotes and pure *b*'s in the proportion of 1 : 2: 1. But the pure *B*'s and the *Bb*-zygotes both develope 70°/₀ of *A*-forms and 30°/₀ of *a*'s; the *b*'s develope 35°/₀ of *A*-forms and 65°/₀ of *a*'s; therefore the proportion of *A*-forms in the race will be

$$\frac{3 \times 70 + 35}{4} = \frac{245}{4} = 61{\cdot}25\%$$

while of these A forms $\frac{70}{245}$ or 28·57°/₀ are pure, $\frac{140}{245}$ or 57·14°/₀ hybrids, and $\frac{35}{245}$ or 14·29 °/₀ aberrant recessives. As before, let T_n be the whole number of dominant forms, all of whose ancestors in one line are also dominants, in any one generation, and of these let p_n be pure forms, i_n impure or hybrids, and e_n aberrant recessives. Then I find that the following relations, corresponding to (11) and (12), or (5) and (6) hold good:—

$$p_{n+1} = \cdot 35\, p_n + \cdot 175\, i_n \qquad (14)$$

$$i_{n+1} = \cdot 35\, p_n + \cdot 35\, i_n + \cdot 35\, e_n \qquad (15)$$

$$e_{n+1} = \cdot 0875\, i_n + \cdot 175\, e_n \qquad (16)$$

where of course

$$T_n = p_n + i_n + e_n \qquad (17)$$

These equations do not appear to give readily an expression relating the chance C_n to C_{n-1} as in equations (10) and (13), the aberrant recessives forming a troublesome element, but they enable the values of T_2, T_3, etc. to be calculated directly from the figures given above, say $T_1 = 245$, $p_1 = 70$, $i_1 = 140$, $e_1 = 35$ (the absolute figures do not matter). Then C_n is given at once by the ratio of T_{n+1} to T_n. Using this process I find

$$C_1 = \cdot 62500$$
$$C_2 = \cdot 63000$$
$$C_3 = \cdot 63194$$
$$C_4 = \cdot 63269$$
$$C_5 = \cdot 63298$$

The figures were deliberately chosen so as to make the value of C_1, the same as in the last case, *but the values of the remaining chances are totally different.* The differences between successive chances run as follows in the two series—

Failure of dominance.	Failure of predetermination.
·02500	·00500
·00385	·00194
·00056	·00075
·00008	·00029

In the first case the knowledge of the grand-parent makes a marked difference in the expectation as to the attributes of the offspring, but the higher ancestry are of very rapidly diminishing importance; in the second case the difference introduced by the knowledge of the grand-parent is small, but the higher ancestry are of greater relative importance. Moreover one could find a whole series of pairs of values for the proportions of A's contributed by B-cells and b-cells (instead of ·7 and ·3), such that the chance of an A of unknown parentage producing an A-form as offspring was 0·625, but for each of these pairs the remainder of the series of chances would be different. This is a greater generalisation than could be obtained by the former assumption of the failure of dominance alone. Given that the heterozygotes produce any fixed proportion of A's, whether 100, 80, 60 or 50 %, the *whole* of the series of ancestral chances is, in that case, determined, so that no two different series can be obtained starting from the same value.

The preceding considerations should suggest to the "Mendelian" that it is a little futile to deny the fact of ancestral heredity when its existence is predicated by his own results, and to the biometrical school that they on their side should be rather cautious in drawing conclusions as to the processes that are or are not consistent with, and still more implied by, the existence of that phenomenon. I gave on pp. 205-6 supra, one theory accounting for the occurrence of ancestral heredity, *viz.*: the failure of "predetermination," and may confess that I had not then remarked that the application of Mendel's Laws, without any modification, would lead to the same result; both theories implying—and this seems the one thing needful—the development of *similar* somatic characters from germ cells of *different* characters. *One* case of ancestral heredity arises directly from Mendel's Laws, and a whole series of cases of a very general character indeed may be derived by supposing either dominance or predetermination of the somatic attributes to fail; the case of *both* failing would be more

general still. In any case then where it is only possible to deal with *attributes*, and not measurably variable characters, it seems impossible to disprove the existence of segregation; it may occur (as Mr. Bateson seems to consider probable) or may not. Nor have we exhausted the ways in which segregation may be masked, apart from any question of its partial failure such as might be invoked to account for some of the divergent results obtained by Mr. Bateson and others. In supposing dominance to fail, we have still assumed the inheritance to be exclusive; this is a logical necessity if predetermination hold, but, if predetermination fail, as well as dominance, the inheritance may become *blended*, *i.e.* the heterozygote may produce, on the average, a per-centage of A's characteristic neither of the B-cells nor the b-cells (in our previous notation), but intermediate between the two; such blending would give rise to yet another series of forms of ancestral heredity.

So far, however, we have dealt solely with cases of inheritance of attributes, without considering the individual variations to which the attribute may be subject within the race; but the relation of these individual variations and their laws of inheritance to the phenomena considered by Mendel is obviously a question of first-class importance. They present two features not directly treated by Mendel at all, but noticed at some length by Mr. Bateson—a sensible continuity of variation in the first place, and, so far as our experience goes, *blended* inheritance in the second, the offspring of two widely different parental forms shewing no tendency to revert to such forms, but resembling the offspring of an intermediate type.

There can be no doubt, of course, as to the *existence* of such individual variations in many of the characters dealt with by Mendel and his followers—length of stem in peas may be cited as an instance where the variations are not merely conspicuous, and, one would imagine, susceptible of easy quantitative measurement—but they are necessarily neglected by hybridisers, who unfortunately rely on their unaided judgments (no sarcasm is intended). The attitude of Mr. Bateson towards these individual variations, and his views on the bearings of Mendelian phenomena on the conceptions of variation in general are rather difficult to follow. He believes, as I gather, that the origin of races or of species is due solely to large and marked variations, and that small variations are of no importance, and speaks of Mendel's discovery as "that discovery which, once and for all, ratifies and consolidates the conception of discontinous variation" (Mendel's Principles, p. 116). It

is not easy to see in what way. As he states elsewhere (*Report*, p. 150) "Mendel's discovery.... applies only to the manner of transmission of a character already existing. It makes no suggestion as to the manner in which such a character came into existence," and the question of the origin of varieties stands, therefore, precisely where it did. Mendel's work has only ratified the conclusion of the biometric school, that, beyond question, large variations are rare, for as we have already pointed out, Mendel's Laws cannot hold in any case where *A*'s give rise to *a*'s during the course of the experiments, and Mendel himself states that no such large variations were remarked in thirty-three varieties of peas during ten years. I do not wish to imply that such variations do not occur at all—on the contrary I think that, *e.g.*, the chemical properties of the soma necessarily imply discontinuity of variation in some respects, a discontinuous origin of colour varieties such as those of the Iceland Poppy (*Papaver nudicaule*) being highly probable—but all the evidence we have goes to indicate that large and sudden variations are most exceptional, the highly divergent individuals forming a vanishingly small proportion of any race.

Mr. Bateson reaches his conclusion as to the importance of Mendelian phenomena for the theory of discontinous variation only, I fancy, by tacitly assuming that the germinal determinant of a character is a structure—or whatever one may term it—incapable of small variations, and as fixed and stable as the ideal molecule of a chemical compound; this I take to be the meaning of his reference "to physical and chemical laws," as rendering continuous variation in the germ cell improbable (p. 22), in the sentence already quoted from Mendel's Principles But this assumption is in no way justified by the results of Mendel's work; all that is predicated by his hypothesis is the existence of some sort of separable determinant for each character for which the law of segregation holds; concerning the variability of that determinant nothing is postulated but the fact that it shall not be so large as to render the boundary between the two races obscure. Surely the very fact that the germplasm gives rise to a long and complex ontogeny indicates that its molecules differ in some way from the simple molecules of water, salt, or sulphuric acid? A dozen or two of atoms may be susceptible of only a few stable groupings, but can the same assumption be made as regards a molecule built up of some thousands? Richter's *Lexikon der organischen Verbindungen* gives for instance as the formula for Haemocyanin $C_{867}\ H_{1363}\ O_{258}\ N_{223}\ S_4\ Cu$

for Haemoglobin, $C_{759}\ H_{1263}\ O_{218}\ N_{195}\ S_3\ Fe$, the first containing 2716 atoms, the second 2378. I confess I speak without a knowledge of modern chemistry, but I can see no physical reason why the loss of an atom or two of hydrogen, or of carbon, in any such case should entail so great an alteration of structure in the molecule as to *alter completely* its physical and chemical characters. It does not seem wholly absurd to suggest that one might obtain *within limits* a sensibly continuous variation of properties with such a compound, even supposing it possible to isolate "pure cultures" of identically similar molecules. "Continuous variation" would then correspond to such minor alterations as did not destroy the stability of the general structure, "discontinuous" or abnormally large variations to such alterations as caused the whole structure to slide over, so to speak, into a new position of equilibrium. I write with a picture in my mind of Mr. Galton's model:—an irregularly polygonal prism which is stable about its position of rest on one face for small oscillations, but will fall into a fresh position if the oscillation be too great (Bibliography, 2). So far then as speculative possibilities go, the occurrence of *sensibly* continuous variation in the properties of an isolated "determinant" seems a hypothesis by no means to be excluded, and surely it can be put to the test. Mr. Bateson appears to accept, and Mendel's hypothesis almost to imply, the truth of the theory of the continuity of the germ plasm as distinct from a theory of pangenesis. If then the individual variations of a character are heritable, it follows that they are, in part at least, due to germinal variation and not wholly to circumstance; if the character is one obeying Mendel's Laws, it also follows, with a high degree of probability, that it is represented by a single determinant, and therefore if the individual variations are heritable, variations in the single determinant are possible. Such an experiment is surely necessary to clear up the facts.

But further, *all* characters cannot be simple units. To take the simplest possible case of compounding, let $x_1\ x'_1$ be a pair of corresponding lengths (say) in two races, these lengths obeying the laws of dominance and segregation; and let $x_2\ x'_2$ be another pair pair of corresponding lengths also obeying the laws. Then clearly Mendel's Laws cannot hold for the "compound characters"

$$X = x_1 + x_2$$
$$X' = x'_1 + x'_2$$

If x_1 and x_2 be both dominant with respect to x'_1 and x'_2 then the

first generation of offspring will exhibit X as the dominant character; but if x_1 and x'_2 or x_2 and x'_1 are the dominant forms then the first generation may exhibit a character that is a "blend" or intermediate between the two parent races, or a character greater or less than either. In the second generation again the parental forms will not be the only ones to appear; the new forms $x_1 + x'_2$ and $x'_1 + x_2$ will also present themselves. Two doubly-compound characters will therefore give rise after crossing to *four* somatic forms; two triply-compound characters to *eight*; two characters compounded of n such units to 2^n forms. But how great is n likely to be in such a case as *stature*, assuming that it *can* be analysed into a set of Mendelian units? As Mr. Galton has remarked ((2) p. 83) "... human stature is not a simple element, but a sum of the accumulated lengths or thicknesses of more than a hundred bodily parts, each so distinct from the rest as to have earned a name by which it can be specified. The list includes about fifty separate bones, situated in the skull, the spine, the pelvis, the two legs, and in the two ankles and feet." Surely it would be a very moderate estimate that the number of units could not be less than 50? Yet this would suffice to give, on the simplest Mendelian assumption that each unit can only exhibit two types, not some mere ten thousand different values of stature, the run of which would be quite indistinguishable from strictly continuous variation, but *over a thousand-million million different types!* Even then if the variations of "units" do take place by discrete steps *only* (which is unproven), discontinuous variation must merge insensibly into continuous variation simply owing to the compound nature of the majority of characters with which one deals. There does not seem any escape from this conclusion. Continuous variation, in the present state of our knowledge, we can only say *may* be due to continuous variation of the elements of the germ cell (determinants or what not), or may be due to the compounding in some way of the discontinuous variations of a number of such elements.

Precisely similar considerations hold good for the case of blending. It is quite possible that characters behaving in other respects as Mendel's Laws would lead one to expect *i.e.*, "unit characters," may in some cases give a blended form for the individuals developed from the heterozygote. But in any case compound characters must blend—more or less. This is obvious if dominance be absent; two pure forms $x_1 + x_2$ and $x'_1 + x'_2$ would then produce with equal frequency offspring of the somatic types $x_1 + x_2$,

$x_1 + x'_2$, $x'_1 + x_2$, $x'_1 + x'_2$, the mean of which is precisely $\frac{1}{2}$ $(x_1 + x'_1 + x_2 + x'_2)$ *i.e.* the mean of the two parent forms. If dominance still hold good the result is not quite so obvious, but it seems correct to state that so long as the greater and the lesser values of the pairs like $x_1\ x'_1$ are the dominants with about equal frequency, the inheritance will be blended, while otherwise blends will only occur in a proportion of the results.

This possible mode of the occurrence of blending was noted long ago by Mr. Galton. He found that the inheritance of stature was strictly blended, as tested by the fact that the offspring of a tall mother with a short father, or *vice versâ*, shewed no more divergence *inter se* than the offspring of two mediocre parents. The inheritance of eye-colour on the other hand he found to be *exclusive*, the offspring generally resembling the one parent or the other, and intermediate tints being rare. "The blending in Stature," he remarks (2, p. 139) "is due to its being the aggregate of the quasi-independent inheritances of many separate parts, while Eye-colour appears to be much less various in its origin." Blending then *must* occur with compound characters, it *may* occur for all we know in some cases of unit characters. It would be easier to gauge the probabilities, if Mendel's followers had made some experiments with a view to elucidating the nature of "exclusive" inheritance in general, and of the very curious phenomenon of "dominance" in particular.

I must apologise for the length to which these remarks have extended, but the subject is a large one, and even as it is I have been compelled to pass over many subsidiary points. It is, however, essential, if progress is to be made, that biologists—statistical or otherwise—should recognise that Mendel's Laws and the Law of Ancestral Heredity are not necessarily contradictory statements, one or other of which must be mythical in character, but are perfectly consistent the one with the other and may quite well form parts of one homogeneous theory of heredity. To make my own position clear, let me repeat with regret, that I cannot include under the same heading the special laws as to the operation of Ancestral Heredity which were formulated by Galton and Pearson. These laws have, beyond question, been of service in suggesting lines of research and possible methods of study, but the *fixity* of the numerical constants involved, which they imply, has not stood the test of time. Selective mating, natural selection, reproductive selection, the effect of circumstance, had all in turn to be recognised

as causes affecting the values of the constants of heredity, until now any statement as to numerical fixity seems reduced to the truism that the constants will always be the same unless for some reason they are different. What is required from a physical theory of heredity is that it should assign a meaning to the variations in the constants that do occur, enabling one, given the law of ancestral heredity for an organ, to state the relative influences thereon of the different agencies concerned—selection, in all forms, circumstance, and so forth. That an ideally complete theory cannot come yet, may be conceded at once; that it is impossible in the present state of biology to form a quantitative theory, founded on clear and definite physical conceptions, which will carry one some steps on the way, I do not believe.

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