VII. *Mathematical Contributions to the Theory of Evolution.*—III. *Regression, Heredity, and Panmixia.*

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(1.) *Introductory.*

There are few branches of the Theory of Evolution which appear to the mathematical statistician so much in need of exact treatment as those of Regression, Heredity, and Panmixia. Round the notion of panmixia much obscurity has accumulated, owing to the want of precise definition and quantitative measurement. The problems of regression and heredity have been dealt with by Mr. FRANCIS GALTON in his epoch-making work on 'Natural Inheritance,' but, although he has shown exact methods of dealing, both experimentally and mathematically, with the problems of inheritance, it does not appear that mathematicians have hitherto developed his treatment, or that

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biologists and medical men have yet fully appreciated that he has really shown how many of the problems which perplex them may receive at any rate a partial answer. A considerable portion of the present memoir will be devoted to the expansion and fuller development of Mr. GALTON's ideas, particularly their application to the problem of *bi-parental inheritance*. At the same time I shall endeavour to point out how the results apply to some current biological and medical problems. In the first place, we must definitely free our minds, in the present state of our knowledge of the mechanism of inheritance and reproduction, of any hope of reaching a mathematical relation expressing the degree of correlation between individual parent and individual offspring.* The causes in any individual case of inheritance are far too complex to admit of exact treatment; and up to the present the classification of the circumstances under which greater or less degrees of correlation between special groups of parents and offspring may be expected has made but little progress. This is largely owing to a certain prevalence of almost metaphysical speculation as to the causes of heredity, which has usurped the place of that careful collection and elaborate experiment by which alone sufficient data might have been accumulated, with a view to ultimately narrowing and specialising the circumstances under which correlation was measured. We must proceed from inheritance in the mass to inheritance in narrower and narrwoer classes, rather than attempt to build up general rules on the observation of individual instances. Shortly, we must proceed by the method of statistics, rather than by the consideration of typical cases. It may seem discouraging to the medical practitioner, with the problem before him of inheritance in a particular family, to be told that nothing but averages, means, and probabilities with regard to large classes can as yet be scientifically dealt with; but the very nature of the distribution of variation, whether healthy or morbid, seems to indicate that we are dealing with that sphere of indefinitely numerous small causes, which in so many other instances has shown itself only amenable to the calculus of chance, and not to any analysis of the individual instance. On the other hand, the mathematical theory will be of assistance to the medical man by answering, *inter alia*, in its discussion of regression the problem as to the average effect upon the offspring of given degrees of morbid variation in the parents. It may enable the physician, in many cases, to state a belief based on a high degree of probability, if it offers no ground for dogma in individual cases.

One of the most noteworthy results of Mr. FRANCIS GALTON's researches is his discovery of the mode in which a population actually reproduces itself by regression and fraternal variation. It is with some expansion and fuller mathematical treatment of these ideas that this memoir commences.

* The physical and arithmetical statements of WEISMANN's "Theory of Germ Plasm" offer, so far as I have been able to interpret them, no sound basis for a quantitative theory of hered ty in the mathematician's sense.

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(2.) *Definitions.*

It is necessary to give definitions to several current biological conceptions, in order to introduce them into our mathematical analysis.

(*a.*) *Variation.*—If a curve be constructed, of which the ordinate y is such that $y\,\delta x$ measures the frequency with which an organ lying in size between x and $x + \delta x$, occurs in a considerable population (500 to 1000 or more), the constants which, for any particular organ for any particular animal determine the form of this curve, are termed the *constants of variation*, or more briefly, the variation of the given organ.

The assumption is made that the frequency is continuous, or that we really reach a curve. In the great majority of cases, where real statistical methods have been used, continuous curves (or, practically, polygons) have been found, and we shall assume this continuity to hold in all cases to which our formulæ are applied.

The size of the organ (x) which corresponds to the ordinate (y) through the centroid of the frequency curve, is termed the *mean;* the size of the organ, which corresponds to the ordinate bisecting the area of the frequency curve, is termed the *median;* the size of the organ corresponding to maximum frequency is termed the *mode.*

We assume, what may be considered as fairly established, that variation curves in zoometry, and more especially anthropometry, approximate closely to probability curves. When the variation curve has more than one mode, it may, as a rule, be resolved into simple probability curves, each with a single mode, and it may be even heterogeneous and require resolution, when only one mode is apparent.* These probability curves may be skew, and in this case the treatment of the problem of heredity involves a discussion of skew-correlation,† but in a very great range of cases the frequency is sufficiently closely given by the normal probability curve. Here the variation is defined by a single constant,‡ the standard deviation σ, and the equation to the curve is given by

$$y = \frac{N}{\sqrt{2\pi}\sigma} e^{-x^2/(2\sigma^2)},$$

and we shall confine our attention to such variation in the present memoir. The following assumption, therefore, lies at the basis of our present treatment of heredity. The variation of any organ in a sufficiently large population—which may be selected in any manner other than by this organ itself from a still larger population—is closely defined by a normal probability curve.

(*b.*) *Correlation.*—Two organs in the same individual, or in a connected pair of

* On resolution and skew variation, see 'Contributions to the Mathematical Theory of Evolution,' Memoirs I. and II., 'Phil. Trans.,' vols. 185 and 186.

† Dealt with in a memoir not yet published.

‡ Inheritance can be treated by single-constant variation in the case of most organs in human adults, but it could not be dealt with in like manner in the of case pedigree buttercups, see De Vries: 'Berichte der Deutschen Botanischen Gesellschaft,' 1894 and 1895.

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individuals, are said to be correlated, when a series of the first organ of a definite size being selected, the mean of the sizes of the corresponding second organs is found to be a function of the size of the selected first organ. If the mean is independent of this size, the organs are said to be non-correlated. Correlation is defined mathematically by any constant, or series of constants, which determine the above function.

The word "organ" in the above definitions of variation and correlation must be understood to cover any measurable characteristic of an organism, and the word "size," its quantitative value.

(*c.*) *Natural Selection.*—This is of two kinds: *Secular Natural Selection* is measured by the changes due solely to mortality, in the mean and standard deviation of the variation-curve as we pass from one adult generation to the next. In statistical observations on man it is by no means easy—as we shall indicate later—to differentiate it from the effects of sexual selection, and of altered sanitary conditions.

Periodic Natural Selection may leave no trace of itself in the adult variation-curves of successive generations; it is measured by the changes due solely to mortality in the mean and standard deviation of the variation curves at successive stages of the *same* generation—*due allowance being made for the changes of the variation-constants due to growth.* In other words, if we watched a generation from birth to the adult stage, carefully preserving it from any form of selective mortality, such as arises from the struggle for existence, we should still find changes in the variation-constants due to the law of growth. If now the same generation be subjected to the struggle for existence, *i.e.*, placed in its natural surroundings, the variation-constants will differ from their values at the corresponding stages of the unselected growth. This difference is due to the selective mortality, *i.e.*, to natural selection. But this selective mortality may go on and still leave the variation-constants of the adult stage of each generation the same. In this case we speak of it as periodic natural selection. It repeats itself in each generation, but produces no secular change. It maintains an adult standard, but is not a factor of progressive evolution.

No estimate of periodic natural selection can be formed until the law of growth has been accurately ascertained by a series of observations on individuals. The influence of secular natural selection will be allowed for in our investigations by supposing the means and standard deviations of successive adult variation-curves to be not necessarily the same.*

(*d.*) *Sexual Selection.*—Sexual Selection† is of two kinds, due respectively to what

* Variation-curves for non-adult populations appear to be frequently skew. I propose in another paper to discuss the general law of selection on the basis of skew curves and with any arbitrary law of growth.

† I think DARWIN's view would be of the following kind. Let A be the most attractive female, *a* the most efficient male, Z the least attractive female and *z* the least efficient male. Then supposing only these four, *a* and *z* would both desire A with the result that (1) *a* would drive away *z* or (2) kill him. In the first case *z* would be free to mate with Z, but if he did so they would tend to produce a miserable

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may be spoken of as individual and tribal taste. Tribal taste manifests itself in the preference of one sex as a whole for mating with members of the other sex having special characteristics, or to the rejection as mates by one sex of members of the other having special characteristics. The preference and rejection being in neither case absolute, but relative. This type of sexual selection, which may be spoken of as *preferential mating*, is measured by the differences in mean and standard deviation between the variation-curves for the whole adult population of one sex, and for the mated portion of it. For example, the mean height and mean variation in height of women generally are not identical, or are not necessarily identical with the mean height and mean variation in height of wives. Preferential mating may have reference to any organ or measurable characteristic of either sex.

Individual taste on the other hand does not denote the exclusion from mating of any section of the population of either sex. It is due to the preference of individuals with an organ or characteristic of given size for mates with the same or another organ or characteristic of a size, the average of which differs from the whole population average. This type of sexual selection which may be spoken of as *assortative mating* is measured mathematically by the coefficient of correlation between the two organs or characteristics in mated pairs.

It will be obvious that preferential mating and assortative mating are fundamental ideas to be quantitatively allowed for in any theory of heredity. Their action may often be in entirely opposite directions.*

(*e.*) *Reproductive Selection.*—One pair may produce more offspring than another, and in this manner give through heredity greater weight to their own characteristics. For example, the mean height of mothers is not identical, or is not necessarily identical with the mean height of wives, nor is the standard-deviation of fathers identical or necessarily identical with the standard-deviation of husbands. Further, the means and standard-deviations of mothers or fathers of sons may be different from those of mothers or fathers of daughters. The quantitative measure of reproductive selection is the correlation between the size of any selected organ in either male and female and their reproductivity, the reproductivity being measured by the number of their offspring in either sex or both sexes.

offspring fated to die out. Of course a might in certain cases after (1) mate with both A and Z. None of these possibilities corresponds exactly to what is described in this section as assortative mating, which in no way necessitates the exclusion from mating of z. a and z are not indeed competitors, but seeking different qualities in their mates. Thus, in man for example, the intellectual and non-intellectual might, and possibly do, sort themselves out in pairs, *i.e.*, there is a correlation between intellectual capacity of husband and wife.

* For example, preferential mating might lead in a highly social community to the rejection of consumptive mates, while assortative mating might, through localisation or community of habit, lead to considerable consumptive correlation. Thus sexual selection as a whole may influence in diverse ways the inheritance of the consumptive taint.

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The importance of determining whether there is any correlation between reproductivity and a given organ of either parent appears to be great. For, if there be, it is not easy to understand how, even in the absence of both natural and sexual selection, a population can remain in a stable state. For example, suppose the mean father or the mean mother or both to be taller than the mean man or the mean woman or both, then this reproductive selection would appear to involve a gradual increase of height in the population in the same manner as selective breeding of animals by man might do. It is probable, therefore, that if reproductive selection be demonstrated by a finite value of the correlation constants, the instability of the population which results is partially or completely screened by natural selection.*

(*f.*) *Heredity.*—Given any organ in a parent and the same or any other organ in its offspring, the mathematical measure of heredity is the correlation of these organs for pairs of parent and offspring. If the organs be the same for parent and offspring, the heredity may be spoken of as *direct*, if they be different as *cross*. The word organ here must be taken to include any characteristic which can be quantitatively measured.

If the organs are not those of parent and offspring, but of any two individuals with a given degree of blood relationship, the correlation of the two organs will still be the proper measure of the strength of heredity for the given degree of relationship. *Cf.* § 6.

(*g.*) *Regression.*—Regression is a term which has been hitherto used to mark the amount of abnormality which falls on the average to the lot of offspring of parents of a given degree of abnormality. The mathematical measure of this special regression is the ratio of the mean deviation of offspring of selected parents from the mean of all offspring to the deviation of the selected parents from the mean of all parents. This may be further elucidated as follows:—Let parents, having an organ or characteristic of given deviation from the average or normal, be termed a "parentage," let the offspring of a parentage be termed a "fraternity." Then the *coefficient of regression* may be defined as the ratio of the mean deviation of the fraternity from the mean offspring to the deviation of the parentage from the mean parent. Both parentage and fraternity may be either male or female. It will be noted that we have so framed our definition of regression, that it marks the deviation of the fraternity from the filial and not the parental mean. We are thus able to allow for secular natural selection and reproductive selection. We shall see in the sequel that the coefficient of regression is a function of the variations in parents and offspring, and further of the correlations which define parental heredity and assortative mating. Further, as in heredity, the deviation or abnormality in parentage and fraternity may be measured with respect to the same or different organs; we have thus *direct* and *cross* regression.

From this special definition of regression in relation to parents and offspring, we

* I hope shortly to publish a paper on "Reproductive Selection in Man," and show how completely it appears to screen Natural Selection in the case of *civilised* man.

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may pass to a general conception of regression. Let A and B be two correlated organs (variables or measurable characteristics) in the same or different individuals, and let the sub-group of organs B, corresponding to a sub-group of A with a definite value a, be extracted. Let the first of these sub-groups be termed an *array*, and the second a *type*. Then we define the coefficient of regression of the array on the type to be the ratio of the mean-deviation of the array from the mean B-organ to the deviation of the type a from the mean A-organ. The following are illustrations of types and arrays :—

Type.	Array.
Organ of given magnitude in—	Distribution of the correlated organs in—
Parent	Fraternity.
Offspring	Parentage.
Wife.	Male matage.
Husband	Female matage.
Given value of—	Distribution of correlated—
Height	Spans.
Cephalic index	Alveolar indices.
Barometric height	Heights at second station.
Local wages	Local pauper percentages.
Etc.	Etc.

It will be seen in the sequel that for the same pair of correlated organs or characteristics, the coefficient of regression is, if the law of frequency be the normal law, the same for the arrays corresponding to all types. But the coefficient is not the same when the type and array organs are interchanged, *e.g.*, the regression of husbands (male matage) on wives is not the same as the regression of wives (female matage) on husbands.

(*h.*) *Panmixia.*—Suppose that starting from a population of given mean and variation for any particular organ, secular natural selection of definite amount takes place for p generations and produces a population of another definite mean and variation for this same organ. Now suppose natural selection, whether periodic or secular, to be suspended for q generations, and sexual selection to be non-extant or negligible, then those members of the general population which were formerly weeded out, will now mix with all the other members of the population, and the results of interbreeding are spoken of as *panmixia*. The mathematical measure of the result on the given organ of panmixia acting for q generations is the change in mean and variation of the population with regard to that organ during these q generations. Should the mean and variation of the population tend with increase of q to approach the mean and variation of the population $p + q$ generations previously, panmixia may be said to reverse natural selection.

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We have now defined the chief factors which will be dealt with in the present memoir, and shown how they are to be quantitatively measured. We shall now proceed to their mathematical analysis on the fundamental assumption that the variations with which we are about to deal obey the normal law of frequency.

(3.) *Correlation with special reference to the Problem of Heredity.*

(*a.*) *Historical.*—The fundamental theorems of correlation were for the first time and almost exhaustively discussed by BRAVAIS ('Analyse mathématique sur les probabilités des erreurs de situation d'un point.' Mémoires par divers Savans, T. IX., Paris, 1846, pp. 255-332) nearly half a century ago. He deals completely with the correlation of two and three variables. Forty years later Mr. J. D. HAMILTON DICKSON ('Proc. Roy. Soc.,' 1886, p. 63) dealt with a special problem proposed to him by Mr. GALTON, and reached on a somewhat narrow basis* some of BRAVAIS' results for correlation of two variables. Mr. GALTON at the same time introduced an improved notation which may be summed up in the 'GALTON function' or coefficient of correlation. This indeed appears in BRAVAIS' work, but a single symbol is not used for it. It will be found of great value in the present discussion. In 1892 Professor EDGEWORTH, also unconscious of BRAVAIS' memoir, dealt in a paper on 'Correlated Averages' with correlation for three variables ('Phil. Mag.' vol. 34, 1892, pp. 194-204.) He obtained results identical with BRAVAIS', although expressed in terms of 'GALTON's functions.' He indicates also how the method may be extended to higher degrees of correlation. He starts by assuming a general form for the frequency of any complex of n organs each of given size. This form has been deduced on more or less legitimate assumptions by various writers. Several other authors, notably SCHOLS, DE FOREST and CZUBER, have dealt with the same topic, although little of first-class importance has been added to the researches of BRAVAIS. To Mr. GALTON alone is due the idea of applying these results—usually spoken of as "the laws of error in the position of a point in space"—to the problem of correlation in the theory of evolution.

The investigation of correlation which will now be given does not profess, except at certain stated points, to reach novel results. It endeavours, however, to reach the necessary fundamental formulæ with a clear statement of *what assumptions are really made*, and with special reference to what seems legitimate in the case of heredity.

(*b.*) *Theory of Correlation.*—Let $\eta_1, \eta_2, \eta_3 \ldots \eta_n$ be the deviations from their respective means of a complex of organs or measurable characteristics. These organs may be in the same or in different individuals, or partly belong to one and partly to another individual. The complex may be constituted by a natural or artificial tie

* The coefficient of correlation was assumed to be the same for the arrays of all types, a result which really flows from the normal law of frequency.

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of any kind, but the tie is to remain the same for every complex, whether it be the result of mating or parentage, or flow from any physiological or social relation, &c.

We shall now assume that the sizes of this complex of organs are determined by a great variety of *independent* contributory causes, for example, magnitudes of other organs not in the complex, variations in environment, climate, nourishment, physical training, various ancestral influences, and innumerable other causes, which cannot be individually observed or their effects measured. Let these causes be m in number, m being generally much greater than n, and let their deviations from their mean intensities be $\epsilon_1, \epsilon_2, \epsilon_3, \ldots \epsilon_m$, then $\eta_1, \eta_2, \eta_3, \ldots \eta_n$ will be functions of $\epsilon_1, \epsilon_2, \epsilon_3, \ldots \epsilon_m$. Further, certain of the ϵ's will appear only in certain of the η's, and the ϵ's will not be fully determined for a given η complex.

We shall in the next place assume that the variations in intensity of the contributory causes are small as compared with their absolute intensity, and that these variations follow the normal law of distribution.* The mean complex being reached with the mean intensities of contributory causes, we have by the principle of the superposition of small quantities :

$$\left.\begin{aligned} \eta_1 &= \alpha_{11}\epsilon_1 + \alpha_{12}\epsilon_2 + \alpha_{13}\epsilon_3 + \ldots + \alpha_{1m}\epsilon_m, \\ \eta_2 &= \alpha_{21}\epsilon_1 + \alpha_{22}\epsilon_2 + \alpha_{23}\epsilon_3 + \ldots + \alpha_{2m}\epsilon_m, \\ &\ldots\ldots \\ \eta_n &= \alpha_{n1}\epsilon_1 + \alpha_{n2}\epsilon_2 + \alpha_{n2}\epsilon_3 + \ldots + \alpha_{nm}\epsilon_m, \end{aligned}\right\} \quad \ldots\ldots \quad \text{(i).}$$

Here any of the system of α's may be zero.

Further, the chance that we have a conjunction of contributory causes lying between ϵ_1 and $\epsilon_1 + \delta\epsilon_1$, ϵ_2 and $\epsilon_2 + \delta\epsilon_2 \ldots e_m$ and $\epsilon_m + \delta\epsilon_m$ will be given by

$$\mathrm{P} = \mathrm{C}e^{-\left(\frac{\epsilon_1^2}{2\kappa_1^2} + \frac{\epsilon_2^2}{2\kappa_2^2} + \frac{\epsilon_3^2}{2\kappa_3^2} + \ldots + \frac{\epsilon_m^2}{2\kappa_m^2}\right)} \times \delta\epsilon_1\delta\epsilon_2\delta\epsilon_3, \ldots \delta_m, \quad \ldots\ldots \quad \text{(ii)}$$

where the standard deviations of the variation distributions for $\epsilon_1, \epsilon_2, \epsilon_3, \ldots \epsilon_n$ are respectively $\kappa_1, \kappa_2, \kappa_3, \ldots \kappa_n$, and C is a constant.

Now by aid of the equations (i.) let n of the variables ϵ, say, the first n, be replaced by the variables η, then the probability that we have a complex with organs lying between η_1 and $\eta_1 + \delta\eta_1$, η_2 and $\eta_2 + \delta\eta_2 \ldots \eta_n$ and $\eta_n + \delta\eta_n$, together with a series of contributory causes lying between ϵ_{n+1} and $\epsilon_{n+1} + \delta\epsilon_{n+1}$, ϵ_{n+2} and $\epsilon_{n+2} + \delta\epsilon_{n+2} \ldots \epsilon_m$ and $\epsilon_m + \delta\epsilon_m$ will be

$$\mathrm{P}' = \mathrm{C}'e^{-\frac{1}{2}\phi^2}\,\delta\eta_1\,\delta\eta_2 \ldots \delta\eta_n\,\delta\epsilon_{n+1}\,\delta\epsilon_{n+2} \ldots \delta\epsilon_m$$

* This may be taken at any rate as a first approximation. It is at this point that the theory of skew-correlation diverges from our present treatment.

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where C′ is a constant, a function of C and the α's, and ϕ^2 consists of the following parts :—

(i.) A quadratic function of the η's from η_1 to η_n,
(ii.) A quadratic function of the ϵ's from ϵ_{n+1} to ϵ_m,
(iii.) A series of functions of the type :

$$\begin{aligned}
&\epsilon_{n+1}\,(b_{1,\,n+1}\eta_1 + b_{2,\,n+1}\eta_2 + \ldots + b_{n,\,n+1}\eta_n),\\
&\epsilon_{n+2}\,(b_{1,\,n+2}\eta_1 + b_{2,\,n+2}\eta_2 + \ldots + b_{n,\,n+2}\eta_n),\\
&\ldots\ldots\ldots\ldots\ldots\ldots\\
&\epsilon_m\,\;(b_{1,\,m}\eta_1 \;\; + b_{2,\,m}\eta_2 \;\; + \ldots + b_{n,\,m}\eta_n),
\end{aligned}$$

where some of the b's may be zero.

Now if P′ be integrated for the values from $-\infty$ to $+\infty$ of all the contributory causes $\epsilon_{n+1}, \epsilon_{n+2} \ldots \epsilon_m$, we shall have the whole chance of a complex with organs falling between η_1 and $\eta_1 + \delta\eta_1$, η_2 and $\eta_2 + \delta\eta_2 \ldots \eta_n$ and $\eta_n + \delta\eta_n$. But every time we integrate with regard to an ϵ, ϵ_{n+1}, say, we alter the constants of each contributory part of ϕ^2, but do not alter the triple constitution of ϕ^2, except to cause one ϵ to disappear from its (ii.) and (iii.) constituents. At the same time we alter C′ without introducing into it any terms in η. Thus, finally, after $m - n$ integrations, ϕ^2 is reduced to its first constituent, or we conclude that the chance of a complex of organs between η_1 and $\eta_1 + \delta\eta_1$, η_2 and $\eta_2 + \delta\eta_2 \ldots \eta_n$ and $\eta_n + \delta\eta_n$ occurring is given by

$$\mathrm{P} = \mathrm{C}e^{-\frac{1}{2}\chi^2}\,\delta\eta_1\,\delta\eta_2 \ldots \delta\eta_n \quad \ldots\ldots\ldots \quad \text{(iii.)}$$

where χ^2 is a quadratic function of the η's. This is the law of frequency for the complex.

Now our deduction of (iii.) seems to have considerable justification in case of heredity. We allow for an indefinite number of quite inappreciable and unascertainable independent contributory causes. We suppose that some of these causes are common to parent and offspring; how many and in what degree we make no pretence at saying. We assume, however, that the action of these causes does not differ very widely in intensity throughout the special range of organisms from which our complexes are drawn, and further, that the variation in intensity of any contributory cause follows that law of frequency, which we know to be at any rate approximately true, for distributions of physical and organic variation similar in character to those in which we may with a high degree of probability suppose the phenomena of heredity to ultimately have their origin.

Having thus deduced, with special reference to our particular topic, BRAVAIS' law of frequency, I propose to consider its characteristics in two special cases, as it is needful to deduce for our present purposes one or two, I believe, novel results.

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(4.) *Special Case of Two Correlated Organs.*

(*a.*) *Theory.*—Let x and y be the deviations of a pair of organs (or measurable characteristics) from their respective means. Let σ_1 and σ_2 be the standard deviations of x and y, treated as independent variations. Let N be the total number of pairs and $z \times \delta x\, \delta y$ the frequency of a pair falling between x and $x + \delta x$, y and $y + \delta y$, then, by BRAVAIS' form,

$$z = \mathrm{C} \times e^{-(g_1 x^2 + 2hxy + g_2 y^2)}$$

where g_1, g_2, and h are constants.

Integrate z for all values of y from $-\alpha$ to $+\alpha$, and we must have the normal curve of x-variation, hence

$$\frac{1}{2\sigma_1^2} = g_1\,(1 - h^2/g_1 g_2).$$

Similarly integrating z for all values of x, we have

$$\frac{1}{2\sigma_2^2} = g_2\,(1 - h^2/g_1 g_2).$$

Now integrate z for all values of x and y to obtain the total frequency, and we have

$$\mathrm{N} = \mathrm{C}\pi/\sqrt{g_1 g_2 - h^2}.$$

If we now write r for $-h/\sqrt{g_1 g_2}$, we can throw z into the form

$$z = \frac{\mathrm{N}}{2\pi\sigma_1\sigma_2}\,\frac{1}{\sqrt{1 - r^2}}\,e^{-\frac{1}{2}\left\{\frac{x^2}{\sigma_1^2(1-r^2)} - \frac{2xyr}{\sigma_1\sigma_2(1-r^2)} + \frac{y^2}{\sigma_2^2(1-r^2)}\right\}}.$$

(*b.*) *On the best Value of the Correlation Coefficient.*—This is the well-known Galtonian form of the frequency for two correlated variables, and r is the GALTON function or coefficient of correlation. The question now arises as to what is *practically* the best method of determining r. I do not feel satisfied that the method used by Mr. GALTON and Professor WELDON will give the best results. The problem is similar to that of determining σ for a variation-curve, it may be found from the mean error or the median, but, as we know, the error of mean square gives the theoretically best results.

Let the n pairs of organs be $x_1, y_1, x_2, y_2, x_3, y_3$, &c. . . . then the chance of the observed series for a given value of r varies as

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$$\frac{1}{(1-r^2)^{\frac{1}{2}n}} e^{-\frac{1}{2}\left\{\frac{x_1^2}{\sigma_1^2(1-r^2)} - \frac{2x_1y_1r}{\sigma_1\sigma_2(1-r^2)} + \frac{y_1^2}{\sigma_2^2(1-r^2)}\right\}}$$
$$\times e^{-\frac{1}{2}\left\{\frac{x_2^2}{\sigma_1^2(1-r^2)} - \frac{2x_2y_2r}{\sigma_1\sigma_2(1-r^2)} + \frac{y_2^2}{\sigma_2^2(1-r^2)}\right\}}$$
$$\times e^{-\frac{1}{2}\left\{\frac{x_3^2}{\sigma_1^2(1-r^2)} - \frac{2x_3y_3r}{\sigma_1\sigma_2(1-r^2)} + \frac{y_3^2}{\sigma_2^2(1-r^2)}\right\}}$$
$$\times \ldots\ldots,$$

or, S denoting summation, since $\sigma_1^2 = S(x^2)/n$, $\sigma_2^2 = S(y^2)/n$, the chance varies as

$$\frac{1}{(1-r^2)^{\frac{1}{2}n}} e^{-n\left\{\frac{1-\lambda r}{1-r^2}\right\}},$$

where λ is written for $S(xy)/(n\sigma_1\sigma_2)$, and $S(xy)$ corresponds to the product-moment of dynamics, as $S(x^2)$ to the moment of inertia.

Now, assume r to differ by ρ from the value previously selected, and expand by TAYLOR's theorem, after expressing the function, in the following manner :—

$$u_r = \frac{1}{(1-r^2)^{\frac{1}{2}n}} e^{-n\left\{\frac{1-\lambda r}{1-r^2}\right\}} = e^{n\left\{\frac{1}{2}\log(1-r^2) - \frac{1-\lambda r}{1-r^2}\right\}}.$$

We have

$$\frac{1}{n}\log u_{r+\rho} = \frac{1}{n}\log u_r + \frac{(1+r^2)(\lambda - r)}{(1-r^2)^2}\rho + \frac{1}{2}\frac{\lambda(2r^3+6r) - 1 - 6r^2 - r^4}{(1-r^2)^3}\rho^2$$
$$+ \frac{1}{6}\frac{\lambda(6+36r^2+6r^4) + 4r^5 - 6r^4 - 28r^3 - 18r}{(1-r^2)^4}\rho^3 + \&c.$$

Hence $\log u_r$ and therefore u_r is a maximum when $r = \lambda$, for the coefficient of ρ^2 is then negative. Thus, it appears that the observed result is the most probable, when r is given the value $S(xy)/(n\sigma_1\sigma_2)$. This value presents no practical difficulty in calculation, and therefore we shall adopt it. It is the value given by BRAVAIS, but he does not show that it is the best.*

(c.) *Probable Error of the Correlation Coefficients.*—Assuming that r has this value, we may put $\lambda = r$ in the above result, and we find

$$u_{r+\rho} = u_r e^{-\frac{n(1+r^2)}{(1-r^2)^2}\frac{\rho^2}{2} - \frac{2nr(r^2+3)}{(1-r^2)^3}\frac{\rho^3}{3} - \&c.}$$

Now $u_{r+\rho}$ is the chance of the observed series on the assumption that the coefficient

* It seems desirable to draw special attention to this best value of the correlation coefficient, as it has hitherto been frequently calculated by methods of somewhat arbitrary character, involving only a portion of the observations.

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of correlation r is $r + \rho$ instead of r. Hence the above is the law of distribution of variation in the coefficient of correlation. If the second term be negligible as compared with the first, we see that ρ follows the normal law of distribution. Thus we may say that with sufficient accuracy for most cases the standard deviation of a coefficient of correlation is

$$\frac{1 - r^2}{\sqrt{n\,(1 + r^2)}},$$

or its probable error $= \cdot 674506 \dfrac{1 - r^2}{\sqrt{n\,(1 + r^2)}}$.

The ratio of the first term neglected to the term retained

$$= \tfrac{4}{3} \frac{r\,(r^2 + 3)}{(r^2 + 1)\,(1 - r^2)} \rho,$$

or to determine the order, giving ρ its probable value on a first approximation, we have

$$\text{ratio} = \tfrac{4}{3} \frac{1}{\sqrt{n}} \frac{r\,(r^2 + 3)}{(r^2 + 1)^{\frac{3}{2}}} \times \cdot 674506.$$

This may be shown to be a maximum for $r^2 = 1$, and the ratio then takes the value $\dfrac{1 \cdot 272}{\sqrt{n}}$, or the second term in this most unfavourable case will only be about 4 per cent. of the first when $n = 1000$. For $r = \cdot 5$, the ratio takes the value $1 \cdot 046/\sqrt{n}$ or for $n = 1000$ is about 3·3 per cent.

It will be sufficient, therefore, for most practical purposes to assume that the probable error of a coefficient of correlation

$$= \cdot 674506 \frac{1 - r^2}{\sqrt{n\,(1 + r^2)}}.$$

(*d.*) *Constancy of Correlation Coefficients for Local Races.*—This result is not only of importance in dealing with the problem of heredity, it is crucial for determining whether constancy of correlation is characteristic of all races of the same species. Mr. GALTON has suggested that the coefficient of correlation might be found to be constant for any pair of organs in different families of the same race. Professor WELDON has determined a series of coefficients of correlation for shrimps and crabs, which he thinks justify him in assuming "as at least an empirical working rule that GALTON's function has the same value in all local races. The question whether the empirical rule is rigidly true will have to be determined by fuller investigation, based on larger samples."*

* 'Roy. Soc. Proc.,' vol. 54, p. 329, 1893.

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Now whether the sample be large enough or not seems to depend on the just determined value of the probable error, and in Professor WELDON's case the probable error is so small, as compared with the value determined for GALTON's function, that I think we may safely draw conclusions from his results.

Taking the case of shrimps, we have for the most reliable determination of r, that for total length of carapace and length of post-spinous portion* :—

	n.	r.	*p.e.* of r.
Plymouth	1000	·81	·0057
Southport	800	·85	·0050

Thus the difference between the r's is not very large, but still between five and six times the probable error (·0075) of their difference.

Taking two cases from Professor WELDON's results for crabs,† with r's of considerably different order, we have :—

		n.	r.	*p.e.* of r.
Breadth, frontal, and . . . . R. antero-lateral margin . . .	Naples	1000	·29	·0187
	Plymouth	1000	·24	·0196
R. antero-lateral margin, and . L. dentary margin	Naples	1000	·60	·0117
	Plymouth	1000	·70	·0089

With these probable errors the identity of the first pair of r's is unlikely ; the identity of the second excessively improbable.

The conclusions therefore to be drawn from our results are these :—The samples taken were sufficiently large to determine r with close practical accuracy. Hence, therefore, unless there were large errors of measurement, or in the determination of r, the evidence of these observations is against the constancy of GALTON's function for local races of the same species. If the differences in the values of r be attributable not to deviation in the sample from the mean, but to experimental error or to methods of calculation, then it would appear that the methods adopted or the measurements are not sufficiently close to supply an answer to the problem proposed, it being an essential condition of the requisite observations that the experimental, or the arithmetic error shall be less than the probable error of the sample. It seems to me extremely improbable that the divergence should be due to errors of measurement, and Professor WELDON's papers, I venture to think, illustrate not the constancy of

* 'Roy. Soc. Proc.,' vol. 51, p. 2, 1892.

† 'Roy. Soc. Proc.,' vol. 54, p. 327, 1893.

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correlation in species, but the equally interesting point of the extent and manner of its variation in local races.

(5.) *Regression, Uniparental Inheritance, and Assortative Mating.*

(*a.*) *General Formulæ.*—On the basis of the above discussion we can obtain the formulæ requisite for calculating scientific measures of uniparental inheritance and assortative mating.

Let male or female parents solely be kept in view, and let male or female parents be considered which have an organ or measurable characteristic differing h from that of the general population of male or female parents. Then the frequency of a variation x in the same or any other organ of the offspring is given by

$$z = \frac{N}{2\pi\sigma_1\sigma_2} \frac{1}{\sqrt{(1-r^2)}} e^{-\frac{1}{2}\left\{\frac{x^2}{\sigma_1^2(1-r^2)} - \frac{2xhr}{\sigma_1\sigma_2(1-r^2)} + \frac{h^2}{\sigma_2^2(1-r^2)}\right\}}.$$

The offspring, therefore, have variation following a normal distribution about the mean

$$x_0 = r\frac{\sigma_1}{\sigma_2}h,$$

and with standard deviation $\sigma_1\sqrt{(1-r^2)}$.

Hence, by our definition, the coefficient of regression $= x_0/h = r\sigma_1/\sigma_2$, and the variability of the offspring of the selected parents is reduced from that of the general population of offspring in the ratio of $\sqrt{(1-r^2)}$ to 1. We thus have a measure of the manner in which selection of parents reduces the variability in offspring, *i.e.*, tends to make the latter closer to a definite type. This result is achieved even with promiscuity in the case of one parent, if there be selection in the case of the other. The greater closeness of approach to type when both parents are selected will be dealt with under biparental inheritance.

We note that the coefficient of regression and the restriction of variability are the same whatever type of parent be adopted, or the closeness with which selection leads to a given type of offspring is independent of the parent adopted and the type of offspring which results from this parent.*

* This is, of course, true of the regression and variability of the array corresponding to any type whatever, when frequency follows the normal law. Mr. G. U. Yule points out to me that if the coefficient of regression be constant for the arrays of all types, then it follows that *whatever be the law of frequency*, the coefficient of regression must $= r\sigma_1/\sigma_2$, where $r = S(xy)/(n\sigma_1\sigma_2)$. This much generalises the formula. At the same time, in the case of skew-correlation, the coefficient of regression usually varies with the type, and the fundamental problem is to determine what function it is of the type. Let bridegrooms of age differing by p years from the mean age of all bridegrooms have an array of brides with a mean age differing q years from the mean age of all brides; then p/q is *not* constant for all values of p.

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These results have been reached by Mr. GALTON in his work on 'Natural Inheritance.' He, however, supposes the population to be stable, and makes the mean and variation of successive generations the same, *i.e.*, x_0 is measured from the mean of the general population of parents, and σ_1 taken equal to σ_2. It seems better to keep our formulæ perfectly general, and allow for possible natural selection of the secular kind as well as for possible reproductive selection.

(*b.*) *Special case of Stature in Man.*—In order to get some idea of the nature of direct and cross inheritance, of assortative mating, &c., in man, I have, in conjunction with Professor W. F. R. WELDON, issued a circular and card appealing for help in collecting family measurements. We hope eventually to procure 1000–2000 families with data of height, span, and arm-length, but it may be many months, or even years, before sufficient material has been accumulated to allow of fairly definite statements being made. Meanwhile, Mr. GALTON, with his accustomed generosity, has placed at my disposal the family data on which his work on 'Natural Inheritance' was based. These data contain statistics with regard to one organ, *height*, for about 200 families. The number is not sufficiently great to make the probable error of quite small enough dimensions in several cases, and so allow of definite conclusions. The data do not offer, as those we are collecting, material for the treatment of cross as well as direct inheritance. Nevertheless, the drift of Mr. GALTON's statistics is in many cases obvious enough, and even in other cases, where the weight of the numerical results is not great, the conversion of our formulæ into numbers will still assist the reader to understand their significance, and serve to some extent for comparison when wider series of statistics are forthcoming.* Hence, in the numerical results of this paper, I wish more to draw attention to method than emphasise general laws. Mr. GALTON's families appear to have been drawn from the upper middle classes, and therefore any conclusions formed must not be hastily extended to the whole community.

* Only those who have attempted to get the measurements of, say 20 families, will appreciate the difficulty of the task of completing even 200 for one organ. Parents and children must be alive and fall within suitable limits of age; and what is more, their interest must be aroused.

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The following tables give the chief results :—

TABLE I.—Variation.

Class.	Number.	Mean Height in inches.	Probable Error of M.H.	S.D. in inches.	Probable Error of S.D.
Males	683	69·215	·066	2·592	·047
Husbands	200	69·136	·126	2·628	·089
Sons	483	69·247	·081	2·617	·057
Fathers in general	935	69·175	·055	2·501	·039
Fathers of sons	483	69·106	·071	2·325	·050
Fathers of daughters	452	69·248	·086	2·731	·061
Females	652	64·043	·061	2·325	·043
Wives	200	63·839	·110	2·303	·078
Daughters	452	64·118	·075	2·347	·053
Mothers in general	935	64·099	·051	2·308	·036
Mothers of sons	483	64·054	·072	2·334	·051
Mothers of daughters	452	64·147	·072	2·274	·051

TABLE II.—Correlation.

Class.	Coefficient r.	Probable Error of r.
Husbands and wives	·0931	·0473
Fathers and sons	·3959	·0241
Fathers and daughters	·3603	·0260
Mothers and sons	·3018	·0267
Mothers and daughters	·2841	·0281

TABLE III.—Regression.

Class.	Coefficient of Regression.
Assortative Mating :—	
Husbands on wives	·1062
Wives on husbands	·0816
Inheritance :—	
Fathers on sons	·3517
Sons on fathers	·4456
Fathers on daughters	·4192
Daughters on fathers	·3096
Mothers on sons	·2692
Sons on mothers	·3384
Mothers on Daughters	·2753
Daughters on mothers	·2932

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TABLE IV.—Variation in Selected Groups.

Class of Selected.	S.D. in inches.	S.D. in inches.	Unselected.
Matage:—			
Wives of selected husbands	2·293	2·303	All wives
Husbands of selected wives	2·617	2·628	All husbands
Parentage:—			
Fathers of selected sons	2·135	2·325	All fathers of sons
Fathers of selected daughters . . .	2·548	2·731	All fathers of daughters
Mothers of selected sons	2·225	2·334	All mothers of sons
Mothers of selected daughters . . .	2·180	2·274	All mothers of daughters
Fraternity:—			
Sons of selected fathers	2·403	2·617	All sons
Daughters of selected fathers . . .	2·189	2·347	All daughters
Sons of selected mothers	2·495	2·617	All sons
Daughters of selected mothers . . .	2·250	2·347	All daughters

TABLE V.—Sexual Ratio.

Class.	Ratio of Means.	Ratio of S. D.'s.	Ratio of V.'s.*
Husbands to wives	1·082	1·141	1·055
Males to females	1·081	1·115	1·032
Sons to daughters	1·080	1·115	1·032
Fathers to mothers . . . , .	1·079	1·084	1·005

* V = the "coefficient of variation" or percentage of variation in organ = 100 S. D. ÷ (mean). See below.

N.B.—Mr. GALTON excluded from his calculations the larger families, but it seems to me that large families form an essential feature of the community. Two brothers are more likely to be two brothers of a large than of a small family, and, accordingly, large families ought to be given their proportionate weight. The whole problem, indeed, of reproductive selection turns upon the inclusion of large families.

Explanation of the Tables.—These tables were calculated in the following manner: Table I. A father or mother appears once for each child in this Table. The mean heights of each group were then calculated, as well as their standard deviations (S.D.) or deviations of mean square. The probable errors of the means and standard deviations were then found by means of the formulæ

$$p.e. \text{ of M.H.} = \cdot 674506 \times \text{S.D.}/\sqrt{n},$$

$$p.e. \text{ of S.D.} = \cdot 674506 \times \text{S.D.}/\sqrt{2n},$$

where n is the number of cases recorded in the second column of the Table.

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To obtain Table II., tables of double entry* were formed for the class enumerated in the first column, *e.g.*, height of husband and height of wife as the variables x and y, and frequency of each pair of heights as z. From this table S (xy) was calculated by very laborious but straightforward arithmetic. This product moment was reduced to parallel axes (x', y') through the centroid of the system and r determined from the formula $r = \mathrm{S}\,(x'y')/n\sigma_1\sigma_2$ (see p. 265). The *p.e.* of r was then found from the formula on p. 266.

The coefficients of regression, in Table III., have the value $r\sigma_1/\sigma_2$, given on p. 267, where, if σ_1 be the standard deviation of A, and σ_2 of B, $r\sigma_1/\sigma_2$ is the regression of an A array on a B type, and $r\sigma_2/\sigma_1$, the regression of a B array on an A type.

In Table IV., the array is first stated and then the type; *e.g.*, in the first line the type is the husband of given height, the array the distribution of all wives of husbands of this height. The first S.D. is that of the array obtained from the formula S.D. $= \sigma_1\sqrt{1 - r^2}$, of p. 267, σ_1 being the second S.D. of Table IV., or the S.D. of the whole group from which the array has been extracted by selecting a particular value of the correlated group.

Table V. gives the ratio for corresponding groups of the two sexes of the constants given in Table I.

Now, a consideration of the probable errors recorded in Tables I. and II. shows us that, in several cases, definite conclusions may be drawn, and in certain other cases very probable conclusions. In particular, the probable errors of the correlation coefficients of inheritance are sufficiently small to show that these coefficients give the chief features of heredity in the group and for the characteristic we are dealing with. We may note one or two special features.

(i.) *Natural Selection.*—We are dealing with two adult populations, and, therefore, should only expect to find traces of secular natural selection. The data, however, are not suited, either by their nature or number, to illustrate this point. There is a slight increase in height of sons over height of husbands, and a larger increase in height of daughters over height of mothers. Neither can be definitely asserted to be significant. Even if they were significant they might be accounted for by (*a*) shrinkage due to old age,† and (*b*) increased physical activity and exercise in the middle classes of the younger generation, especially daughters. If we turn from means to S.D.'s we see again an insignificant change in the range of variation of husbands and sons, the sons being slightly less variable than fathers. This result, were it necessary to account for it, would be more likely due to our having taken sons from a less general population than husbands—a point to be borne in mind

* It did not seem necessary to publish these tables, but the corresponding tables will be published when the fuller data for heredity in man, which I am at present collecting, are complete.

† In my own collection of data, several parents state that they are now shorter than they used to be. The shrinkage in the case of fathers of sons cannot be great in Mr. GALTON's statistics, to judge by the means, unless we suppose a sensible regression in sons' stature.

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when statistics of this kind are collected, and more than one son in a family is included. There is a more significant difference in the variation of wives and daughters. It is, however, in the opposite sense to what we may suppose would be produced by natural selection, or by the fact that we have drawn daughters from a less general population than wives. There is no definite evidence as to natural selection to be drawn from these results accordingly.

(ii.) *Sexual Selection.*—(*a.*) *Preferential Mating.*—We have no general populations to compare with those of husbands and wives. If we suppose the population stable, and treat sons and daughters as characteristic of the general unmarried population, husbands are not a significant selection from sons. Possibly the difference between the variation in daughters and wives might be accounted for by a distaste for very tall or very short wives in the middle classes. The difference is, however, not very significant, but it should be borne in mind in dealing with a larger range of statistics.

(*b.*) *Assortative Mating.*—Although the probable error (Table II.) is about half the coefficient of correlation, it is unlikely that the latter can be really zero, and although we must not lay very great stress on the actual value of r, still we are justified in considering that there is a definite amount of assortative mating with regard to height going on in the middle classes. It may be expressed by saying that wives 1″ taller than the mean will have on an average husbands ·11″ taller than the mean, and husbands 1″ taller than the mean, wives on an average ·08″ taller than the mean (Table III.). Table IV. shows us that the variation in matages would hardly be discoverable directly from our present range of statistics.*

(iii.) *Reproductive Selection.*—Although in the matter of means we cannot assert significance between the heights of males in general and fathers in particular, it is quite possible that such will reveal itself in more ample data. On the other hand, we see at once that fathers are definitely less variable than husbands, and fathers of sons remarkably less variable than fathers of daughters. Thus, while the height of a father is less closely related to his chances of having a daughter, any tendency to normality is of service in the chances of having a son. Reproductivity in males seems to be thus essentially correlated to height, and again, height to be potential in the question of male or female offspring.

An endeavour to directly calculate the correlation of reproductivity and height is

* Of course 200 couples give graphically nothing like a surface of correlation, nor can any section of it be taken as a fair normal curve. We assume *à priori* that 1000 couples would give a fair surface. This is practically what I have found for skull-measurements, 900 give an excellent curve, 50 a doubly, or even trebly, peaked polygon. None the less, sets of 50 skulls give means and S.D.'s in close accord. For example, in Professor FLINDERS PETRIE'S newly discovered race, 50 male crania from T. and Q. graves give for cephalic index: Mean, 72·96, S.D., 2·82; while 53 male crania from General and B. graves give: Mean, 72·92, S.D., 2·95. The 103 crania together give: Mean, 72·938, S.D., 2·885, with a probable error of S.D. = ·29. The variation *curves* would not suggest any such close agreement at all. The constants, however, suffice to show the homogeneous character of the two sets of excavations.

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frustrated by the obvious fact that size of adult families does not follow any approach to a normal distribution. Thus, I find in 205 adult families the following frequency:—

Title.	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
(1) Sons and daughters	..	32	22	23	31	23	19	18	18	8	5	5	..	..	..	1
(2) Sons only . .	25	43	46	42	30	10	5	3	..	..	1					
(3) Daughters only	25	56	44	34	21	13	5	3	3	1						

This Table shows the number of those families in which (1) the number of sons and daughters, (2) the number of sons only, (3) the number of daughters only correspond, with the title in the top line.

Now, although, as I propose to show later, the quantity, $r = S(xy)/(n\sigma_1\sigma_2)$, is really a significant characteristic of correlation, just as σ_1 and σ_2 are significant for variation even in the case of skew frequency, still there is little to be gained by working it out in this particular case, where, the statistics being insufficient to accurately determine the skew law of frequency, we shall not be able to find what we want—the law of regression.*

But several points as to paternal reproductivity may be learnt from these families. In the first place, of the 25 families with no sons, the father in 5 cases only was below the mean, in 20 cases above the mean height. The mean height of fathers in general is 5′ 9″·17, but of sonless fathers is 5′ 11″·03. Of the 25 daughterless fathers, 14 are below and 11 above the mean height; the mean height of the daughterless father being 5′ 8″·71. Or, the same point may be emphasised in this way: If short fathers be taken as those below 5′ 6″·5, and tall fathers as those above 5′ 11″·5, short fathers have 65 sons and tall fathers 67 sons. We should accordingly, with our proportion of sons and daughters, expect 61 daughters to short fathers and 63 to tall fathers, but we find short fathers with 73 and tall fathers with 81. This point in reproductive selection, that mediocre fathers have more tendency to sons and exceptional fathers to daughters, seems of considerable importance in relation to the prepotency of paternal inheritance. A similar point, but less emphatically significant, may be noted in the case of mothers. Mothers of daughters are less variable than mothers of sons. Without laying too great stress on statistics of so small a range and of one characteristic only, we may still suggest that it might be worth while to investigate whether the offspring of a mediocre parent and an abnormal parent do not tend to follow the sex of the mediocre parent.

* Much more complete statistics of size in families have recently been sent to me by Mr. F. Howard Collins. They give a remarkably smooth skew frequency distribution, thus demonstrating the need of the theory of skew correlation when we are dealing with reproductive selection. I propose to illustrate this in a memoir on skew correlation.

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Finally, it is impossible to more than *hazard* suggestions as to reproductive selection in relation to mothers' height. It will be noticed that both mothers of sons and mothers of daughters are taller than wives, and, further, daughters, while taller than wives, are not so tall as mothers of daughters. Hence, while the difference in height of daughters and wives might be due to natural selection or improved physical training, it might also be accounted for by greater reproductivity as to daughters in tall women, *i.e.*, mothers of daughters taller than wives, and this tallness being transmitted in a lesser extent to daughters. This would be a case of secular change due to reproductive selection. The statistics are, however, too few to make the differences in the mean heights of wives, daughters and mothers, very definitely significant.

(iv.) *Inheritance.*—Mr. GALTON has concluded from his data that the coefficient of regression is ·3333 from father to son or from son to father, and by the assumption of the "midparent" has practically given the mother an equal prepotency with the father in heredity. The fuller theory developed in this paper does not seem in entire agreement with these conclusions. In the first place, the theory of uni-parental inheritance shows us that it is not the constancy of variation in two successive generations with which we have to deal, but the question whether sons have the same degree of variability as the "fathers of sons," and this must be definitely answered in the negative. Table II. shows us that there are undoubtedly significant differences in the coefficients of correlation, which may be summed up in the words *prepotency in heredity of the father*. It must be remembered that this is only for one characteristic, *height*, but in this characteristic both sons and daughters, on the average, take very considerably more after their father than after their mother. Turning to Table V., we see that the ratio of the mean heights of the two sexes, considered in three different classes, is practically the same, *i.e.*, 1·08, or 13 to 12, as Mr. GALTON has expressed it. Now, in Table III. we see that the coefficients of regression in paternal inheritance are not only sensibly greater than those of maternal inheritance, but, as these coefficients have to be multiplied by the *absolute* deviations of father or mother from their means to obtain the absolute deviations of offspring, and as these absolute deviations will be in the ratio of 13 to 12, there is a considerable further reduction to be made in comparing the strength of maternal with that of paternal heredity.

Thus it may be said that paternal heredity is to maternal heredity, in the case of sons, as ·4456 to ·3384 $\times \frac{12}{13}$ or to ·3124, and in the case of daughters, ·3096 $\times \frac{13}{12}$ or ·3354 to ·2932. Thus, while daughters inherit less from both their parents on the average than sons, both—and sons especially—take more after their father than their mother. The inferior inheritance of daughters may, to some extent, be counterbalanced by the law already noticed, that exceptional fathers have more often daughters than sons.

We may illustrate this by two examples—the regression of grandson on grandfather, and of great-grandson on great-grandfather when the inheritance is respectively through the male and female lines.

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	Male line.	Female line.
Grandson on grandfather	·1986	·0885
Great-grandson on great-grandfather . .	·1048	·0307

In the first case, the strength of inheritance is more than double through the male; in the second, more than triple through the male what it is through the female line. Were this law of inheritance true, not only of stature, but of other physical, and especially of mental characteristics, some justification might be found for confining hereditary peerages initially given for merit to the male line. Meanwhile, it cannot be too strongly emphasized that the present results apply only to one organ, are based on comparatively few families drawn from a special class of the community, and thus stand in need of careful criticism in the light of ampler statistical material.

Another point already briefly referred to, which seems of significance, is the inequality of regression in the case of ascent and descent in the direct line. It may seem paradoxical to assert that sons are more like fathers than fathers are like sons, but the solution is bound up in the statement that fathers of sons are less variable than sons, or, in another form, that every son is not to the same degree a potential father. Similarly, the opposite paradox that fathers are, on the average, more like their daughters than daughters are like their fathers, finds its solution in the relatively great variability of fathers of daughters.

In Table IV. are tabulated alongside, in each case, the standard deviation for the corresponding general population, the standard deviations for inheritance from selected classes. Here again we see a general law for height, which deserves to be investigated for other organs, and for a variety of animals, namely, we breed "truer to the type," have less variability in offspring, if we breed from selected males rather than from selected females. We shall see later the effect of selecting both parents.

(*c.*) *On Further Relations between Correlation, Regression, and Variability.* (i.) *The Coefficient of Variation V.*—In dealing with the comparative variation of men and women (or, indeed, very often of the two sexes of any animal), we have constantly to bear in mind that relative size influences not only the means but the deviations from the means. When dealing with absolute measurements, it is, of course, idle to compare the variation of the larger male organ directly with the variation of the smaller female organ. The same remark applies also to the comparison of large and small built races.

If the absolute measurements* have in the case of man to be on the average altered in the ratio of 13 to 12 to obtain those of the woman, if Mr. Galton has gone so far as to replace any woman by an equivalent man on this basis, then, clearly, to compare

* The ratio 13 to 12 is not only true of stature, but approximately of several other organs, weight, brain-capacity, &c., &c.

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deviations in man and woman, we must alter the deviations in the same ratio. Freeing ourselves from this particular ratio, we may take as a measure of variation the ratio of standard deviation to mean, or what is more convenient, this quantity multiplied by 100. We shall, accordingly, define V, the coefficient of variation, as the percentage variation in the mean, the standard deviation being treated as the total variation in the mean; since the p.e. = ·674,506 × S.D., V multiplied by ·674,506 may be called the "probable percentage variation." Of course, it does not follow because we have defined in this manner our "coefficient of variation," that this coefficient is really a significant quantity in the comparison of various races; it may be only a convenient mathematical expression, but I believe there is evidence to show that it is a more reliable test of "efficiency" in a race* than absolute variation. At present, however, we will merely adopt it as a convenient expression for a certain function, and proceed to examine its relation to correlation.

Let m_1, m_2 be the means of two correlated organs; σ_1, σ_2 their standard deviations; r their coefficient of correlation; V_1, V_2 their coefficients of variation; and R_1, R_2 the respective regressions for deviations d_2 and d_1 of the two organs.

Now

$$R_1 = r\,\frac{\sigma_1}{\sigma_2}\,d_2 = r\,\frac{V_1}{V_2} \times \frac{m_1\,d_2}{m_2},$$

or

$$\frac{R_1}{m_1} = r\,\frac{V_1}{V_2} \times \frac{d_2}{m_2},$$

and similarly

$$\frac{R_2}{m_2} = r\,\frac{V_2}{V_1} \times \frac{d_1}{m_1}.$$

But we see that the amounts d_2/m_2 and d_1/m_1 are equally significant deviations in the case of the second and first organ, while the amounts R_1/m_1 and R_2/m_2 are equally significant regressions in the case of the first and second organ.†

It follows, therefore, that *the significances of the mutual regressions of the two organs are as the squares of their coefficients of variation.*

Hence inequality of coefficients of variation marks inequality of mutual regressions. Now coefficients of variation are rarely, if ever, equal for the same organ in corresponding classes of men and women. In dealing with male and female skull measurements for a great variety of races, this inequality is often very marked, and, therefore, differences of variation tell, especially in mutual regression in the case of sexual selection and inheritance from the opposite sex. They are sufficient, I think, to preclude Mr. GALTON's theory of the mid-parent from being considered as more than a

* By "race efficiency," I would denote stability, combined with capacity to play a part in the history of civilization. I hope later to publish details of variation, especially in skull measurements of different races of man, the data of which I have been for some years reducing.

† For example, 1″ and $\frac{12}{13}$″ I term equally significant deviations or regressions in the stature of man and woman, and 1″ and $\frac{13}{12}$″ in the stature of woman and man.

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first approximation. Turning to Table V., we see that variation in height is greater for males than females; but while very sensible for husbands and wives, and sons and daughters, it is insignificant for fathers and mothers. This superiority of male to female variation, as measured by the coefficient of variation, is in accordance with the usual belief that the male is more variable than the female, but it is entirely out of accordance with the great bulk of the statistics I have so far reduced. The belief seems to have arisen from a very loose notion of how variation is to be estimated. These stature statistics of the English middle classes seem to some extent anomalous. For example, I find from statistics of stature in the German working classes:—

Male coefficient of variation . . .	= 4·0245,
Female ,, ,, . . .	= 4·2582.

Ratio of female to male coefficient = 1·058, thus more than reversing the highest English ratio, that of husbands and wives. It is noteworthy that, while the variation is thus reversed, the ratio of the mean heights equals 1·078, and remains practically the same. These remarks are introduced in order to prevent any too hasty generalisation as to the nature of male and female correlation based on a current belief in the greater intensity of male variation.

(ii.) *Coefficient of Correlation and Coefficients of Variation.*—Let x and y be two correlated organs, and let ξ and η be corresponding deviations from the mean values m_1 and m_2. We shall suppose that ζ and η are so small that the squares of the ratios ξ/m_1 and η/m_2 are negligible as compared with the first powers. Let r be the coefficient of correlation of x and y, σ_1, σ_2 their standard deviations, v_1, v_2 their coefficients of variation, and let z be any function $f(x, y)$ of x and y with a deviation ζ, corresponding to ξ and η, and a standard deviation, mean and coefficient of variation respectively Σ, M, and V.

Differentiating $z = f(x, y)$ and remembering our hypothesis as to the smallness of the variations, we have:

$$\zeta = f_x\xi + f_y\eta.$$

Squaring:

$$\zeta^2 = f_x^2\xi^2 + f_y^2\eta^2 + 2f_xf_y\xi\eta.$$

Summing for every possible value of ξ and η, and dividing by n the total number of correlated pairs:

$$\frac{S(\zeta^2)}{n} = f_x^2\frac{S(\xi^2)}{n} + f_y^2\frac{S(\eta^2)}{n} + 2f_xf_y\frac{S(\xi\eta)}{n},$$

or,

$$\Sigma^2 = f_x^2\sigma_1^2 + f_y^2\sigma_2^2 + 2f_xf_y\sigma_1\sigma_2 \times \frac{S(\xi\eta)}{n\sigma_1\sigma_2}.$$

Now, if there were no correlation, we should have: $\Sigma^2 = f_x^2\sigma_1^2 + f_y^2\sigma_2^2$; hence

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any law of frequency whatever which causes $S(\xi\eta) = 0$,—for example, if it be equally likely that η occurs with an equal negative or positive value of ξ,—will show that x and y are independent variations. Hence, if we define $r = S(\xi\eta)/(n\sigma_1\sigma_2)$ as the coefficient of correlation, we see that it has a significance extending much further than the normal law of error. Just as σ_1, σ_2 are radii of gyration (and independent of any special law of error), so $S(\xi\eta)$ is a product moment, and its vanishing marks the absence of correlation, or directions of independent variation.

We see, then, that the coefficient of correlation may be found from

$$r = \frac{\Sigma^2 - f_x^2\sigma_1^2 - f_y^2\sigma_2^2}{2f_xf_y\sigma_1\sigma_2},$$

or by calculating standard deviations.

The question naturally arises as to what is the best value of $f(x, y)$. This will often be already answered by the data themselves. A common case is that in which the variations in x and y are given, and the variation in their ratio or the index x/y is calculated. In this particular instance $f_x = M/m_1$ and $f_y = -M/m_2$. Hence

$$r = \frac{v_1^2 + v_2^2 - V^2}{2v_1v_2}$$

We thus throw back the determination of correlation on ascertaining three coefficients of variation.

This formula, while less general than the one previously given, in that we have neglected squares of small quantities, is more general in that we have not limited ourselves to any special law of frequency.

(iii.) *Example.*—The formula may be illustrated by the following statistics taken from a not yet published paper on variation in man. r = coefficient of correlation between length and breadth.

Adult Male Crania.

Professor Flinders Petrie's newly discovered race.*

Length of skull . . .	$m_1 = 185{\cdot}2777$,	$\sigma_1 = 5{\cdot}7783$,	$v_1 = 3{\cdot}1187$
Breadth of skull . . .	$m_2 = 135{\cdot}0194$,	$\sigma_2 = 4{\cdot}4076$,	$v_2 = 3{\cdot}4183$
Cephalic index, B/L .	$M = 72{\cdot}9379$,	$\Sigma = 2{\cdot}8848$,	$V = 3{\cdot}9551$

$$r = {\cdot}2705.$$

* Professor Flinders Petrie kindly replied to my request for 100 skulls of a homogeneous race, 3,000 to 4,000 years old, by bringing back to England the finest anthropological collection—skeletons as well as crania—known to me. The collection was packed and brought to England at the charge of Mr. A. B. Pearson-Gee. Mr. Herbert Thompson has made a series of measurements on 301 skulls, ♂ and ♀, details of which will be published later, and the above constants are calculated from his measurements. The date of the new race is about 3000 B.C.

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MODERN German (Bavarian Peasants*).

Length of skull . . .	$m_1 = 180{\cdot}58$,	$\sigma_1 = 5{\cdot}8441$,	$v_1 = 3{\cdot}2363$
Breadth of skull . . .	$m_2 = 150{\cdot}47$,	$\sigma_2 = 5{\cdot}8488$,	$v_2 = 3{\cdot}8871$
Cephalic index, B/L .	$M = 83{\cdot}41$,	$\Sigma = 3{\cdot}5794$,	$V = 4{\cdot}2913$

$r = {\cdot}2849.$

MODERN French (Parisians†).

Length of skull . . .	$m_1 = 181{\cdot}85$,	$\sigma_1 = 5{\cdot}9420$,	$v_1 = 3{\cdot}2675$
Breadth of skull . . .	$m_2 = 144{\cdot}93$,	$\sigma_2 = 5{\cdot}2139$,	$v_2 = 3{\cdot}5975$
Cephalic index, B/L .	$M = 79{\cdot}82$,	$\Sigma = 3{\cdot}7865$,	$V = 4{\cdot}7438$

$r = {\cdot}0474.$

The probable error of r in all three cases lies between ·06 and ·07. Now it is clear that had we only dealt with the race from Egypt and the Bavarians, we might easily have concluded that the coefficient of correlation was constant for local races of man, and had remained so for nearly 5,000 years. The French numbers completely upset this view. In order to test my French results I give another series from the Anthropological Collection at Munich; the skulls are those of French soldiers who died at Munich during the Franco-German war.

MODERN French (Peasants).

Length of skull . . .	$m_1 = 179{\cdot}93$,	$\sigma_1 = 6{\cdot}2987$,	$v_1 = 3{\cdot}5006$
Breadth of skull . . .	$m_2 = 143{\cdot}51$,	$\sigma_2 = 5{\cdot}4208$,	$v_2 = 3{\cdot}7772$
Cephalic index, B/L .	$M = 79{\cdot}7857$,	$\Sigma = 3{\cdot}8410$,	$V = 4{\cdot}8141$

$r = {\cdot}1265.$

This collection numbers only 57 crania, and the probable error of r is about ·09, but clearly we have the same general features as in the previous French series. In particular the closeness in the line for the cephalic index constants is remarkable. The value of r might possibly be the same as for the Parisians; it is highly improbable that it should agree with the value of r for the Germans or the race from Egypt. We are compelled to conclude, therefore, that it is very unlikely that "GALTON's function" is constant for all local races of man.

* Calculated from measurements given by Professor J. RANKE: 'Beiträge zur physischen Anthropologie der Bayern,' Bd. 1, S. 88, Kapitel VI., I may take this opportunity of acknowledging the extreme kindness of Professor RANKE in helping me in a variety of ways.

† Calculated from measurements extracted from the manuscripts of M. PAUL BROCA, which I owe to the courtesy of M. MANOUVRIER. He has responded to my request by forwarding to me copies of a great variety of measurements, which will be largely used in a paper on variation in man.

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An examination of the above numbers brings out a fact which I am not sure has been noted before: namely, the alteration from dolicocephaly to brachycephaly appears to chiefly depend upon an alteration in the breadth and not in the length of the skull. We see too that, if variation be judged, not by standard-deviations, but by the coefficients of variation advocated in this paper, the breadth of skull is in all cases a sensibly more variable quantity than the length, and, further—a point to which I shall return on another occasion—that the more civilised races are the more variable. Both of these results have, I believe, very important bearing on the mathematico-statistical theory of evolution. On the present occasion the above example is only given to illustrate the relation of variation to correlation.

(6.) *Collateral Heredity.*

(*a.*) *Stature in Man.*—The whole theory of correlation as applied to uniparental inheritance may be at once applied to correlation between brothers, sisters and brothers and sisters. To illustrate the theory I give the following tables, again based on Mr. GALTON's statistics.

In the pairs sister-sister and brother-brother the elder sister or the elder brother has been taken first in order to ascertain the effect of earlier birth on correlation. In the pairs sister-brother, I had no data as to relative age.

TABLE VI.—Variation.

Class.	Number.	Mean Height in inches.	Probable error of M.H.	S.D. in inches.	Probable error of S.D.
Elder sisters of sisters	595	63·869	·0617	2·2303	·0436
Younger sisters of sisters . . .	595	64·199	·0695	2·5119	·0491
Elder brothers of brothers . .	605	69·0174	·0715	2·6080	·0506
Younger brothers of brothers .	605	69·0814	·0725	2·6434	·0513
Sisters of brothers	1181	63·9274	·0440	2·2430	·0311
Brothers of sisters	1181	69·0963	·0533	2·7164	·0377

TABLE VII.—Correlation.

Class.	Coefficient r.	Probable error of r.
Sister-sister	·4436	·0203
Brother-brother	·3913	·0216
Sister-brother	·3754	·0158

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TABLE VIII.—Regression.

Class.	Coefficient of Regression.
Younger sister on elder sister	·4996
Elder sister on younger sister	·3939
Younger brother on elder brother . . .	·3966
Elder brother on younger brother . . .	·3860
Sister on brother.	·3100
Brother on brother	·4547

TABLE IX.—Variation in Selected Groups.

Selected fraternity.	S.D. in inches.	S.D. in inches.	Unselected.
Younger sisters of selected elder .	2·2512	2·5119	All younger sisters of sisters
Elder sisters of selected younger .	1·9989	2·2303	All elder sisters of sisters
Younger brothers of selected elder .	2·4327	2·6434	All younger brothers of brothers
Elder brothers of selected younger .	2·4000	2·6080	All elder brothers of brothers
Sisters of selected brother . . .	2·0789	2·2430	All sisters of brothers
Brothers of selected sister	2·5178	2·7164	All brothers of sisters

These tables have been calculated in precisely the same manner as the previous series.

(*b.*) *Conclusions.*—Now these results seem at several points of very great suggestiveness. In the first place, with regard to variation, we see that elder sisters are significantly more mediocre than younger sisters; younger sisters are taller and more variable. The same difference appears in the case of elder and younger brothers, but the probable errors do not allow us in this case to assert that the difference is certainly significant. To illustrate this conclusion we give the constants for pairs of sisters, no respect being paid to relative age.

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TABLE X.—Sisters of Sisters.

Mean height	64·0″454
Probable error of mean height	·0655
S.D.	2·3668
Probable error of S.D.	·0463
Coefficient of correlation r	·4386
Probable error of r	·0205
Coefficient of regression	·4386
S.D. of array of sisters of selected sister	2·1270

It will be seen from this table that elder and younger sisters of sisters are respectively less and more variable than sisters of sisters in general. It will be noted also that sisters of brothers are, both in stature and variation, nearer akin to elder sisters of sisters than to younger sisters. It deserves accordingly to be investigated whether or not sisters are not on the average older than brothers—on this point I have no data. As sisters of brothers approximate to elder sisters of sisters, so brothers of sisters correspond more closely to younger than to elder brothers of brothers. These are points which require fuller investigation, when ampler statistics are forthcoming. Turning to correlation we note that the coefficients in the case of collateral inheritance are slightly greater than in the case of direct inheritance. It will be remarked at once that the values are much less than those given by Mr. GALTON, "Natural Inheritance," p. 133, who has himself drawn attention to the considerable difference between the constants for collateral inheritance given by his R.F.F. Data and by his Special Data. Mr. GALTON having kindly allowed me to use his data, I have recalculated from the formula $r = S(xy)/(n\sigma_1\sigma_2)$ the value of r for the Special Data, taking my pairs of brothers precisely as I had done for the Records of Family Faculties. I find $r = \cdot 5990$ with a probable error of $\pm \cdot 0124$. This value is not as high as Mr. GALTON's, but differs very widely from the value ·3913 given above.

In making the calculations, however, I was much struck by the peculiarities presented by a certain portion of the data, which I will speak of as the Essex contribution. The brothers therein were very short and remarkably close together. I therefore went through the calculations again, separating the Essex contribution, and with the following results:—

MR. GALTON'S Special Data.

	Whole population.	Essex contribution.	Remainder.
Mean height	68·0″544	67·0″797	68·0″797
Probable error	·0402	·1013	·0457
r for brothers	·5990	·7175	·5574
Probable error of r	·0124	·0200	·0152

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Now the probable error of the difference of the Essex contribution and the remainder is ·1111″ for height and ·0251 for correlation. Thus difference in height is nine times, and the difference in correlation more than six times the corresponding probable error. It is absolutely necessary therefore to conclude that the Essex contribution differs significantly from the remainder of the data. Now the Essex contribution appears to be drawn from brothers in a volunteer regiment, and I am inclined to think there may be two sources accounting for its peculiarities, (*a*) unconscious selection as to height by those who join the volunteers, (*b*) a greater correlation among the agricultural and working classes than among the middle classes. At any rate the great variation within the family to be found in the R.F.F. data does not repeat itself either in the Essex contribution or in other portions of the special data, which appear also to be drawn from military and working class sources.

I would accordingly suggest that the R.F.F. data and the Special data give different results, because the latter are largely drawn from a different class of the population from the former (and possibly in the case of volunteer regiments by a method which itself tends to emphasise correlation). I should expect that the influence of natural selection is far greater—witness the greater infantile mortality—in the working classes, and that accordingly we should find the variation in a fraternity sensibly less, or the correlation much greater. I believe, then, that difference of variation in different classes of the community will ultimately be found to account for part, if not all, of the difference between the two values given for the correlation of brothers by the Special data and by the R.F.F. data.

Considering the amount by which the elimination of a portion only of the heterogeneity of the Special data reduces r, it does not seem likely that the R.F.F. data are so wide of the mark in the correlation values as might at first be thought. I doubt whether the correlation coefficients for collateral inheritance—at any rate in the middle classes—can be greater than ·5. I have not at present sufficient data of my own to make a trustworthy determination of brother-brother correlation, but I was able to find the correlation of 237 brother-sister pairs from about 160 families. The measurements were taken without boots, and give values for the mean heights of brothers and sisters sensibly over 69″ and 64″ respectively. The families were all middle-class families—mostly those of male and female college students. They thus approximate to Mr. Galton's R.F.F. series. The result was

$$r = \cdot 4703 \pm \cdot 0308$$

The previous result was

$$r = \cdot 3754 \pm \cdot 0158$$

The probable error of the difference therefore $= \cdot 0346$ and the difference ·095, between two and three times the probable error. The two differ, of course, consider-

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ably,* but they are nearer together than to Mr. GALTON's ·67, and being entirely independent series, may be taken to justify the statement made above that the coefficient for the middle classes can hardly exceed ·5. Thus there is not, I think, sufficient ground at present for forming any definite conclusion as to the manner in which lineal is related to collateral heredity. It does not seem to me necessary that the coefficient for the former should be half that for the latter, as supposed by Mr. GALTON.

In some respects, indeed, the Special data verify the conclusions we may draw from the R.F.F. data. Thus R.F.F., Special data, and the two components into which I have divided the latter, all four agree in making the younger brother taller than the elder brother. The variability of both brothers is practically equal in the Special data and slightly greater than that of the R.F.F. data—2·656 as compared with 2·626—a difference not significant, and which, if it were, might be put down to the mixture of classes in the Special data.

Assuming that the regression coefficients in Table VIII. give the relative if not the absolute values for collateral inheritance, we draw from them a few suggestions for further inquiry when the statistics are forthcoming. In the first place, sisters are more like each other than brothers. At any rate, the younger sister is more like the elder sister than brother is like brother. If this appears to contradict the principle that sons are more like their parents than daughters, a solution of the paradox must be sought in the relative variabilities of daughters, elder sisters, and younger sisters. To compare the strength of inheritance in brothers and sisters, we have to consider not ·3100 and ·4547, but these coefficients of regression multiplied and divided respectively by 13/12, or ·3358 and ·4197, whence we see that the brother takes more after the sister than the sister after the brother.

It will be wise, however, to lay no great stress on these results, until a wider series of statistics has been collected.

The following example must be taken only as the roughest approximation, but so far as it goes as confirming the above results.

An exceptional grandmother in Baden† had a length-breadth head index of 90, her 20 grandchildren had a mean head index of 83·55, with a S.D. = 3·025. The mean head index of the general population‡ was 83·15 with S.D. = 3·63. Thus, if r_1 be the regression of offspring on parent, and r_2 of offspring on each other, $r_1^2 \times 6{\cdot}85 = {\cdot}4$, and $\sqrt{(1 - r_2^2)} = 3{\cdot}025/3{\cdot}63$.

Hence, $r_1 = {\cdot}24$ and $r_2 = {\cdot}55$. Considering the large probable error of the S.D. of the fraternity (·32), these results indicate inheritance in head indices of the same order as in stature.

* The difference is to be expected. Mr. GALTON's R.F.F. series allows for due weight being given to the variability in large families. My statistics take only four members at a maximum, and frequently only two out of each family.

† O. AMMON, 'Die natürliche Auslese beim Menschen,' p. 13. Three children were unmeasured, and I have accordingly had to disregard this generation.

‡ Calculated from results for 6748, BADENSER, given by AMMON, p. 67.

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(7.) *Special Case of Three Correlated Organs.*

We need not stay long over the general theory as it has been fully treated by BRAVAIS. We indicate its general outline in a modified form. By p. 263 we have, if x, y, z be the deviations from the means of the three organs, and σ_1, σ_2, σ_3 their standard deviations,

$$P = Ce^{-\frac{1}{2}\left(\lambda_1 \frac{x^2}{\sigma_1^2} + \lambda_2 \frac{y^2}{\sigma_2^2} + \lambda_3 \frac{z^2}{\sigma_3^2} - \frac{2yz}{\sigma_2\sigma_3}\nu_1 - \frac{2zx}{\sigma_3\sigma_1}\nu_2 - \frac{2xy}{\sigma_1\sigma_2}\nu_3\right)} dx\, dy\, dz.$$

This may be written in either the form,

$$P = Ce^{-\frac{1}{2}\lambda_1\left(\frac{x}{\sigma_1} - \frac{\nu_3 y}{\lambda_1\sigma_2} - \frac{\nu_2}{\lambda_1}\frac{z}{\sigma_3}\right)^2} \times e^{-\frac{\lambda_2\lambda_1 - \nu_3^2}{2\lambda_1}\left(\frac{y}{\sigma_2} - \frac{z}{\sigma_3}\frac{\nu_1\lambda_1 + \nu_2\nu_3}{\lambda_2\lambda_1 - \nu_3^2}\right)^2}$$

$$\times e^{-\frac{\lambda_1\lambda_2\lambda_3 - 2\nu_1\nu_2\nu_3 - \lambda_1\nu_1^2 - \lambda_2\nu_2^2 - \lambda_3\nu_3^2}{2(\lambda_1\lambda_2 - \nu_3^2)}\frac{z^2}{\sigma_3^2}} dx\, dy\, dz \quad . \quad . \quad (A),$$

or,

$$P = Ce^{-\frac{1}{2}\lambda_1\left(\frac{x}{\sigma_1} - \frac{\nu_3}{\lambda_1}\frac{y}{\sigma_2} - \frac{\nu_2}{\lambda_1}\frac{z}{\sigma_3}\right)^2}$$

$$\times e^{-\frac{1}{2}\left\{\left(\frac{y}{\sigma_2}\right)^2 \frac{\lambda_2\lambda_1 - \nu_3^2}{\lambda_1} + \left(\frac{z}{\sigma_3}\right)^2 \frac{\lambda_3\lambda_1 - \nu_2^2}{\lambda_1} - \frac{2yz}{\sigma_2\sigma_3}\frac{\nu_1\lambda_1 + \nu_2\nu_3}{\lambda_1}\right\}} dx\, dy\, dz \quad . \quad . \quad . \quad (B).$$

Integrating A for x, y, z successively between $\pm\infty$, we have, if n be the number of correlated triplets, and

$$\chi = \lambda_1\lambda_2\lambda_3 - 2\nu_1\nu_2\nu_3 - \lambda_1\nu_1^2 - \lambda_2\nu_2^2 - \lambda_3\nu_3^2,$$

$$n = C\,.\,(2\pi)^{3/2}\sigma_1\sigma_2\sigma_3/\sqrt{\chi},$$

or,

$$C = n\sqrt{\chi}/((2\pi)^{3/2}\sigma_1\sigma_2\sigma_3).$$

Integrating B for x between $\pm\infty$, we have

$$P' = C'e^{-\frac{1}{2}\left\{\left(\frac{y}{\sigma_2}\right)^2 \frac{\lambda_2\lambda_1 - \nu_3^2}{\lambda_1} + \left(\frac{z}{\sigma_3}\right)^2 \frac{\lambda_3\lambda_1 - \nu_2^2}{\nu_1} - \frac{2yz}{\sigma_2\sigma_3}\frac{\nu_1\lambda_1 + \nu_2\nu_3}{\lambda_1}\right\}} dy\, dz.$$

But this must be the correlation distribution for y and z treated independently of x, or, comparing with p. 264, if r_1, r_2, r_3 be the three correlation coefficients for the pairs yz, zx, xy respectively, we have

$$\lambda_1/(\lambda_1\lambda_2 - \nu_3^2) = 1 - r_1^2 = \lambda_1/(\lambda_3\lambda_1 - \nu_2^2).$$

$$(\nu_1\lambda_1 + \nu_2\nu_3)/(\lambda_2\lambda_1 - \nu_3^2) = r_1.$$

Integrating A for x and y from $\pm\infty$, we must have the distribution for z treated independently, or a normal distribution σ_3; this gives at once

$$\lambda_1\lambda_2 - \nu_3^2 = \chi.$$

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Hence we have by symmetry the equations,

$$\lambda_1 = \chi(1 - r_1^2), \qquad \lambda_2 = \chi(1 - r_2^2), \qquad \lambda_3 = \chi(1 - r_3^2),$$
$$\nu_1\lambda_1 + \nu_2\nu_3 = \chi r_1, \qquad \nu_2\lambda_2 + \nu_3\nu_1 = \chi r_2, \qquad \nu_3\lambda_3 + \nu_1\nu_2 = \chi r_3.$$

We easily deduce

$$\chi^2(r_1 - r_2r_3) = (\lambda_2\lambda_3 - \nu_1^2)(\nu_1\lambda_1 + \nu_2\nu_3) - (\nu_2\lambda_2 + \nu_3\nu_1)(\nu_3\lambda_3 + \nu_1\nu_2) = \nu_1\chi,$$

or,

$$\nu_1 = \chi(r_1 - r_2r_3),$$

and similarly,

$$\nu_2 = \chi(r_2 - r_3r_1), \quad \nu_3 = \chi(r_3 - r_1r_2).$$

Finally,

$$\chi^2\{(r_1 - r_2r_3)(1 - r_1^2) + (r_2 - r_1r_3)(r_3 - r_1r_2)\} = \chi r_1,$$

or,

$$\chi(1 - r_1^2 - r_2^2 - r_3^2 + 2r_1r_2r_3) = 1.$$

Thus all the constants are determined, and we have,

$$P = \frac{n\sqrt{\chi}}{(2\pi)^{3/2}\sigma_1\sigma_2\sigma_3}$$
$$e^{-\frac{1}{2}\chi\left\{\frac{x^2}{\sigma_1^2}(1-r_1^2) + \frac{y^2}{\sigma_2^2}(1-r_2^2) + \frac{z^2}{\sigma_3^2}(1-r_3^2) - 2(r_1 - r_2r_3)\frac{yz}{\sigma_2\sigma_3} - 2(r_2 - r_3r_1)\frac{zx}{\sigma_3\sigma_1} - 2(r_3 - r_1r_2)\frac{xy}{\sigma_1\sigma_2}\right\}} dx\,dy\,dz.$$

This agrees with BRAVAIS' result, except that he writes for r_1, r_2, r_3 the values $\Sigma(yz)/(n\sigma_2\sigma_3)$, etc., which we have shown to be the best values (see *loc. cit.*, p. 267).

Obviously we have the following general results. If Σ_1 be the standard deviation of a group of x-organs selected with regard to values h_2 and h_3 of y and z,

$$\Sigma_1 = \frac{\sigma_1}{\sqrt{\{\chi(1 - r_1^2)\}}} = \sigma_1\sqrt{\frac{1 - r_1^2 - r_2^2 - r_3^2 + 2r_1r_2r_3}{1 - r_1^2}}$$

and if h_1 be the deviation of the mean of the selected x-organs from the x-mean of the whole population

$$h_1 = \frac{r_3 - r_1r_2}{1 - r_1^2}\frac{\sigma_1}{\sigma_2}h_2 + \frac{r_2 - r_1r_3}{1 - r_1^2}\frac{\sigma_1}{\sigma_3}h_3.$$

Expressions of the form $\frac{r_3 - r_1r_2}{1 - r_1^2}$ will be spoken of as coefficients of double correlation, and expressions of the form $\frac{r_3 - r_1r_2}{1 - r_1^2}\frac{\sigma_1}{\sigma_2}$ as coefficients of double regression.*

* [The above values for Σ_1 and h_1 are still true, as Mr. G. U. YULE points out to me, *whatever be the law of frequency*, provided the standard-deviations of all arrays be the same and h_1 be a linear function of h_2 and h_3.]

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(c.) *Bi-parental Inheritance of Stature.*

TABLE XII.—Correlation as Influenced by Assortative Mating.

Class.	Correlation coefficients.	
	Modified by mating.	Direct.
Daughters and fathers	·3368	·3603
,, mothers	·2528	·2841
Sons and fathers	·3710	·3959
,, mothers	·2673	·3018

TABLE XIII.—Regression Coefficients as Influenced by Assortative Mating.

Class.	Regression coefficients.	
	Modified by mating.	Direct.
Daughters on fathers	·2895	·3096
,, mothers	·2609	·2932
Sons on fathers	·4176	·4456
,, mothers	·2997	·3384

Thus we see that both in correlation and regression very sensible differences are made by the introduction of bi-parental formulæ.

TABLE XIV.—Variation in Selected Groups.

Class.	Standard deviations.			
	(i.) All offspring.	(ii.) Offspring of selected mother.	(iii.) Offspring of selected father.	(iv.) Offspring of selected mother and father.
Sons	2·617	2·495	2·403	2·300
Daughters	2·347	2·250	2·189	2·108

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Still one or two remarkable general principles may be noticed. Let us suppose, what is not improbable, that there is a first organ, say in the father, which has no sensible correlation with a second organ in the offspring, but that the latter organ in the mother is closely correlated by assortative mating with the first organ in the father. The formula for regression in the offspring of parents having the deviations h_2 and h_3 in the two organs (or characteristics) will now be

$$h_1 = -\frac{r_1 r_2}{1 - r_1^2}\frac{\sigma_1}{\sigma_2} h_2 + \frac{r_2}{1 - r_1^2}\frac{\sigma_1}{\sigma_3} h_3.$$

This shows us that the possession in any exceptional degree of the first organ by the father will actually reduce the amount of the second organ which the offspring inherits from the mother. Let a special example be used to illustrate this. Suppose the problem to be the inheritance of artistic sense from the mother and (h_1, h_3) be measures of the deviations of this sense in son and mother from the normal. Suppose further that h_2 be a measure of the father's physique, say his girth of chest. Now it is conceivable that artistic sense in the mother may be closely correlated with physique in father. If now we deal with artistic sense of the son as related to physique in father and artistic sense in mother, we conclude that exceptional physique in the father will *reduce* the exceptional artistic sense which the son inherits from his mother. Similarly, the exceptional physique which the son would inherit from his father would be reduced by exceptional artistic sense in his mother. It will be noted that these results have no relation whatever to the coexistence or not of artistic sense with physique in the father or the mother. They depend entirely on the influence of assortative mating. It is remarkable that, given mothers of high artistic sense, then this will be handed down in a greater degree to those offspring whose fathers have a physique below the average, than to those of fathers who have a physique above the average.

The above example is not to be taken as a demonstrated truth, but as an illustration of the effect of assortative mating on cross-heredity. Innumerable similar statements can be made, but it seems desirable to await the collection of definite statistics before discussing them at length.

The only statistics which are at present at my disposal for the consideration of bi-parental inheritance are Mr. GALTON's "Family Records," and to these I now turn.

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(8.) *Double Regression and Biparental Inheritance.*

(*a.*) *General Formulæ and Comparison with the Theory of the Midparent.*—If we apply the results of Section (7) to the problem of inheritance, we obtain some interesting results. Let r_1 = coefficient of correlation for the same or different organs in two parents, *i.e.*, be the measure of assortative mating; r_3 = coefficient of correlation of organs of offspring and male parent, *i.e.*, be the measure of paternal inheritance; r_2 = coefficient of correlation of organs of offspring and female parent, *i.e.*, be the measure of maternal inheritance; then the above formulæ express the chief characteristic of biparental inheritance as modified by assortative mating. If r_1, as probably is frequently the case, be small, then we see that the effect of assortative mating is to reduce the deviation of the offspring. Suppose there were no assortative mating, then the mean deviation of the offspring of selected parents would be

$$h_1 = r_3 \frac{\sigma_1}{\sigma_2} h_2 + r_2 \frac{\sigma_1}{\sigma_2} h_3,$$

and the actual value r_1, being small, is clearly less than this. Again, even admitting the insignificance of the assortative mating in some cases, we see that, unless $r_2 = r_3$, and further special relations hold between the variations of parents and offspring, this formula is not reducible to a mid-parent formula.

For example, in the case of stature, consider the male offspring of two pairs of parents. In the first case, let the father be 4″ and the mother ·923″ above the average; in the second, let the father be 1″ and the mother 3″·692 above the average. In both cases the height of the mid-parent is 2″·5 above the average, and the average male offspring will, on the mid-parent theory, exceed the mean by 1″·67. But in the first case, the bi-parental formula gives 1″·95, and in the second, 1″·52. In the case of the female offspring of the same pairs, the mid-parental formula gives 1″·54 for both pairs, and the bi-parental formula 1″·41 and 1″·25 respectively. These differences are due to the prepotency of paternal inheritance, and to the inequality of the variation in different male and female groups.

These results have, of course, no greater validity than the statistics upon which they are based—a validity which Mr. GALTON has been very careful to weigh ('Natural Inheritance,' pp. 73, 131), but, I think, they suffice to show that the mid-parent theory must be looked upon as only an approximation of a rough kind.

It must further be borne in mind, that the variability of a fraternity with given mid-parent is, if assortative mating be neglected, $\Sigma_1 = \sigma_1 \sqrt{1 - r_2^2 - r_3^2}$; or if r_2 be $= r_3$, it is equal to $\sigma_1 \sqrt{1 - 2r_2^2}$, and *not* $\sigma_1 \sqrt{1 - r^2}$.

(*b.*) *Effect of Assortative Mating on Cross Heredity.*—Our formula of course applies to the problems I have classed as those of *cross heredity.* Unfortunately, I have no statistics at present to give any illustration of the intensity of cross-heredity.

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GENERAL FORMULÆ FOR REGRESSION IN STATURE:—

h_2 = deviation of father; h_3 = deviation of mother.

Sons:—The mean height of array of sons corresponding to fathers of height h_2 and mothers of height h_3 is

$$h_1 = \cdot 4176\, h_2 + \cdot 2997\, h_3,$$

or,

$$= \cdot 4176\, h_2 + \cdot 2766\, (1\cdot 08\, h_3).$$

Daughters:—The mean height of array of daughters corresponding to fathers of height h_2 and mothers of height h_3 is

$$h'_1 = \cdot 2895\, h_2 + \cdot 2609\, h_3.$$
$$= \cdot 3136\, (h_2/1\cdot 08) + \cdot 2609\, h_3.$$

In the second expressions given with both formulæ, the parental heights are exhibited in terms of the equivalent heights of the sex of the offspring.

Explanation of the Tables.—Table XII. gives the value of the correlation coefficient as influenced by assortative mating, *e.g.*, $\frac{r_3 - r_1 r_2}{1 - r_1^2}$. The values of the simple correlation coefficients (r_1, r_2, r_3) are taken from Table II. Against each coefficient is placed the value of the "direct" coefficient, on the supposition that $r_1 = 0$—*e.g.*, r_3—in order to exhibit immediately the influence of assortative mating.

Table XIII. gives the regression coefficients as influenced by assortative mating, *e.g.*, $\frac{r_3 - r_1 r_2}{1 - r_1^2} \frac{\sigma_1}{\sigma_2}$ (see p. 286), and "direct" or uninfluenced by such mating, *e.g.*, $r_3 \frac{\sigma_1}{\sigma_2}$; the former are calculated from the values given in Tables XII. and I., and the latter are reproduced from Table III.

Table XIV. exhibits the decreasing variation in arrays of sons and daughters, when we select (i.) neither father nor mother, (ii.) a mother of given type, (iii.) a father of given type, and (iv.) both mother and father of given types; (i.) is taken from Table I., (ii.) and (iii.) from Table IV., and (iv.) is calculated from the formula for Σ_1 deduced on p. 287.

(*d.*) *Conclusions. Prepotency of Father.*—These tables bring out the essential prepotency of the father in the case of both sons and daughters, the ratio of the contributions being 42 to 28 in the first case and 31 to 26 in the second case. A prepotency of the father* in other characteristics has been noted by Mr. GALTON in his "Hereditary Genius," but it is there attributed to the greater ease with which the male characteristic (genius) makes itself apparent. It deserves, however, to be

* Prepotency of either parent might, I think, be easily tested statistically in the case of morbid inheritance, particularly in tubercular disease. Dr. R. E. THOMPSON ('Family Phthisis,' pp. 89 and 95), indicates a prepotency of the mother in both male and female inheritance of this disease.

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considered whether there is not, at any rate in many characteristics, an actual and not apparent male prepotency. It is, perhaps, needless to point out the sensible, if small, modifications introduced into inheritance by assortative mating.

Lastly, we note in Table XIV. the increasing tendency to "breed truer" as we select (i.) mother, (ii.) father, and (iii.) both mother and father.

(9.) *On Some Points connected with Morbid Inheritance.*

(*a.*) *On the Skipping of Generations.*—It must be carefully borne in mind that the formulæ we have discussed make not the least pretence to explain the mechanism of inheritance. All they attempt is to provide a basis for the quantitative measure of inheritance—a schedule, as it were, for tabulating and appreciating statistics. At the same time we may reasonably ask whether our formulæ are wide enough to embrace certain of the more isolated and remarkable features of heredity. Let the subscripts 1, 2, 3, 4 refer respectively to father, mother, son, daughter. Thus, σ_3 would be the S.D. of the son population, h_2 a deviation of a mother from the mean of mothers, r_{14} the correlation coefficient of fathers and daughters, and so on. Now if we consider the general form for single correlation :

$$z = z_0 e^{-\frac{1}{2}\left(\frac{x^2}{\sigma'^2} - \frac{2rxy}{\sigma'\sigma''} + \frac{y^2}{\sigma''^2}\right)\frac{1}{1-r^2}},$$

we may give any values whatever to σ' and σ'', and any value to r, which is less than unity, and deduce the theoretical results. Let us suppose r to be of finite value, but that σ'' is very small as compared with σ'. Then the regression of y on $x = h'r\sigma''/\sigma'$ will be very small, while the regression of x on $y = h''r\sigma'/\sigma''$ will be large. On the other hand, the deviation in y will never be very remote from its mean. All this is perfectly true whatever be the value of r.

Now let us apply this to some secondary sexual characteristic, say hair on the face. A very small amount of hair on the woman's face, with a very large amount of hair on the man's face, is compatible with a large value of r; a small amount of hair on the woman's face may be accounted for by a low mean and very small standard deviation. The regression from father to daughter will be expressed by

$$h_4 = r_{14}\frac{\sigma_4}{\sigma_1}h,$$

or, since σ_4 is extremely small, the daughter will hardly differ sensibly from the mean small hairiness of women. The regression from daughter to daughter's son will be

$$r_{23}\frac{\sigma_3}{\sigma_2}h_4 = r_{23}r_{14}\frac{\sigma_3}{\sigma_2}\frac{\sigma_4}{\sigma_1}h_1$$

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or, since σ_2 and σ_4 are nearly, if not practically, equal, and σ_3 and σ_1 also, we have—regression from grandfather to grandson through the female line $= r_{23}r_{14}h_1$.

This may be a very sensible quantity, if the correlation coefficients are of considerable magnitude. What we have here, then, is the *skipping of a generation*, the inheritance of an especially male characteristic through the female line. The same reasoning would apply to the inheritance of an especially female characteristic through the male line. The formula, of course, gives no *explanation* of why σ_4 is small and r_{14} finite. It is only suggested that these outlying facts of heredity are not necessarily inconsistent with the formula. It may be argued that this account of skipping a generation would only apply to a characteristic which actually exists in both sexes, even if only in a small degree in one of them, and further, it assumes the distribution of this small degree to be of a normal character. This argument would certainly touch characteristics functionally necessary and peculiar to one sex; it may be doubted how far it would affect the question of secondary sexual characteristics, which may have rudimentary values in the sex of which they are not characteristic. It must further be remembered, however, that our correlation formulæ are perfectly true for *cross* heredity, and accordingly the idea of rudimentary value may be pushed a good way, even to the idea of latency in a second closely-allied organ. The idea of latency here is not to be pressed into any theory of panmixia or of germ plasm. Given that certain bulls get good milkers, we have the problem, what organ or characteristic, rudimentary or not, in bulls has the highest numerical coefficient of correlation with the milk-giving capacity of the cows they beget? We may not be able to ascertain this organ or characteristic, but the problem is really a statistical one, and does not assert anything as to the mechanism of heredity. The skipping of a generation in secondary, or even in primary, sexual characteristics, does not seem accordingly to present anything of a character which our formula fails to cover. In particular, in the case of morbid inheritances, such as gout and colour-blindness, which, while peculiarly male diseases, are yet handed down through the female line, our formula seems to be of considerable suggestiveness. This suggestiveness essentially depends on the independence of the two factors—correlation and variation—which are components of the formula. Thus, while there appears to be no necessary relation between power of transmitting and capacity for developing a disease, the independence of correlation and variation will probably allow us to account for most special cases. The reader must be careful to note that we are not compelled to give r or σ meanings relating directly to the intensity of the disease; they may refer to the size of organs or intensity of characteristics on which the liability to the disease or its intensity directly or indirectly depends. Bearing this in mind, we have only to put r_{13} finite, or vanishingly small, while both σ_1 and σ_3 are finite, to grasp (i.) how gout may be transmitted from grandfather through either son or daughter to grandson, and yet (ii.) how colour-blindness and hæmophilia are transmitted, as a rule, through daughter only to grandson—in both cases the

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daughter generally herself escaping (r_{14} finite and σ_4 very small). The protection of the transmitting sex is due, not to smallness of correlation, but to relative smallness of variation in that sex.

(*b.*) *General Formulæ for Four Correlated Organs.*—Another point—especially important for the problem of morbid inheritance—is the relative ages at which a characteristic appears in parent and offspring. DARWIN has noted how a characteristic appearing at a given age in the parent will reappear at the same age—sometimes indeed earlier—in the offspring. In particular, inherited diseases tend to develop themselves at an earlier date in the offspring than in the parent in proportion to the intensity of the inheritance. This appears to be especially the case in gout, rheumatic fever, diabetes, and phthisis.*

Now, the quantities with which we have to deal here are *four* in number, ages of parent and offspring on appearance of disease and intensities of the disease in the parent and offspring. We require, accordingly, the formulae for triple correlation. Proceeding, as in the earlier discussions, we find, if x_1, x_2, x_3, x_4 be the deviation of the four quantities from their respective means, σ_1, σ_2, σ_3, σ_4 their standard deviations, r_{12}, r_{13}, r_{14}, r_{23}, r_{24}, r_{34}, the six correlation coefficients pair and pair of organs or characteristic $z\,\delta x_1\,\delta x_2\,\delta x_3\,\delta x_4$, the frequency out of a total of n quadruplets of the quadruplets with organs or characteristics between x_1, x_2, x_3, x_4 and $x_1 + \delta x_1$, $x_2 + \delta x_2$, $x_3 + \delta x_3$, $x_4 + \delta x_4$:

$$z = \frac{n\sqrt{\chi}}{4\pi^2\sigma_1\sigma_2\sigma_3\sigma_4}\, e^{-\frac{\chi}{2}\left\{\lambda_1\left(\frac{x_1}{\sigma_1}\right)^2+\lambda_2\left(\frac{x_2}{\sigma_2}\right)^2+\lambda_3\left(\frac{x_3}{\sigma_3}\right)^2+\lambda_4\left(\frac{x_4}{\sigma_4}\right)^2-2\nu_{12}\frac{x_1x_2}{\sigma_1\sigma_2}-2\nu_{13}\frac{x_1x_3}{\sigma_1\sigma_3}-2\nu_{14}\frac{x_1x_4}{\sigma_1\sigma_4}-2\nu_{23}\frac{x_2x_3}{\sigma_2\sigma_3}-2\nu_{24}\frac{x_2x_4}{\sigma_2\sigma_4}-2\nu_{34}\frac{x_3x_4}{\sigma_3\sigma_4}\right\}}$$

where

$$\lambda_1 = 1 - r_{23}^2 - r_{34}^2 - r_{42}^2 + 2r_{23}r_{34}r_{42},$$

$$\nu_{12} = r_{12}(1 - r_{34}^2) - r_{13}r_{23} - r_{14}r_{24} + r_{34}(r_{14}r_{23} + r_{13}r_{24}),$$

and

$$\begin{aligned} 1/\chi = 1 &- r_{12}^2 - r_{13}^2 - r_{14}^2 - r_{23}^2 - r_{24}^2 - r_{34}^2 + r_{12}^2r_{34}^2 + r_{23}^2r_{14}^2 + r_{13}^2r_{24}^2 \\ &+ 2(r_{23}r_{24}r_{34} + r_{34}r_{14}r_{13} + r_{12}r_{14}r_{24} + r_{12}r_{13}r_{23}) \\ &- 2(r_{12}r_{14}r_{23}r_{34} + r_{14}r_{13}r_{23}r_{24} + r_{12}r_{13}r_{24}r_{34}), \end{aligned}$$

while the remaining λ's and ν's may be written down by symmetry from λ_1 and ν_{12}.

Accordingly we have for regression the formula

$$h_1 = \frac{\nu_{12}}{\lambda_1}\frac{\sigma_1}{\sigma_2}h_2 + \frac{\nu_{13}}{\lambda_1}\frac{\sigma_1}{\sigma_3}h_3 + \frac{\nu_{14}}{\lambda_1}\frac{\sigma_1}{\sigma_4}h_4,$$

and for the standard deviation of a group of organs x, corresponding to selected organs h_2, h_3, h_4 (*i.e.*, an array)

$$\Sigma_1 = \sigma_1/\sqrt{\chi\lambda_1}.$$

* Here, as elsewhere, I have to thank my friend, Dr. R. T. RYLE, for the kindness with which he has allowed me to examine the material he has collected with regard to morbid inheritance.

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(c.) *Antedating of Family Diseases.*—We may now apply these results to the case of morbid inheritance, making the following assumptions :—

(a.) The distribution of the disease with regard to both age and intensity will be taken to be the same for any two successive generations, and to be normal.

(b.) The age at which the disease appears and its intensity are both directly inherited, but the age of appearance and intensity of the disease in the parent are not directly correlated with the intensity of the disease and the age of its reappearance in the offspring.

Let ϵ be the coefficient of correlation between the age of appearance of disease in the parent and the age of the offspring at its reappearance; let σ be the standard-deviation for the frequency of the disease at different ages, and M the mean age at which the disease appears in the population.

Let η be the coefficient of correlation between the intensity of the disease in the parent and the intensity of the disease in the offspring; let σ' the standard-deviation of the intensity-frequency and M′ be the mean intensity.*

Let $M + A_1$, $M' + I_1$, be the mean age of the appearance of the disease and its mean intensity for an array of offspring, whose parents exhibited the disease when $M + A_2$ years old with an intensity $M' + I_2$.

Let the subscripts 1, 2, 3, 4 refer respectively to age of offspring, age of parent, and intensity in offspring and intensity in parent. Then, in the formula for triple correlation, we must put:

$$r_{12} = \epsilon, \quad r_{34} = \eta, \quad r_{13} = r_{24} = \kappa, \quad r_{14} = r_{23} = 0.$$

Hence:

$$\lambda_1 = \lambda_2 = 1 - \eta^2 - \kappa^2, \quad \nu_{12} = \epsilon + \eta(\kappa^2 - \epsilon\eta),$$
$$\lambda_3 = \lambda_4 = 1 - \epsilon^2 - \kappa^2, \quad \nu_{34} = \eta + \epsilon(\kappa^2 - \epsilon\eta),$$
$$\nu_{13} = \nu_{24} = \kappa - \kappa(\kappa^2 - \epsilon\eta),$$
$$\nu_{14} = \nu_{23} = -\kappa(\epsilon + \eta),$$
$$1/\chi = 1 - \epsilon^2 - \eta^2 - 2\kappa^2 + (\kappa^2 - \epsilon\eta)^2.$$

Substituting these values in the regression formula, we find:

$$A_1 = \frac{\epsilon + \eta(\kappa^2 - \epsilon\eta)}{1 - \eta^2 - \kappa^2} A_2 + \frac{\kappa - \kappa(\kappa^2 - \epsilon\eta)}{1 - \eta^2 - \kappa^2} \frac{\sigma}{\sigma'} I_1 - \frac{\kappa(\epsilon + \eta)}{1 - \eta^2 - \kappa^2} \frac{\sigma}{\sigma'} I_2.$$

Now as the parents in the group $M + A_2$, $M' + I_2$ are in no way selected by

* It might be difficult to get a mathematical measure of the intensity of a disease. For simple theory as apart from statistical measurements, such is, however, unnecessary. The terms used in medical works, acute, subacute, chronic, &c., sufficiently indicate that the relative intensity of various cases is a fact duly recognized by the trained medical mind, if it cannot always be quantitatively expressed.

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parentage, or influenced by heredity, being general statistics, we shall assume that, on the average, $A_2 = \kappa \frac{\sigma}{\sigma'} I_2$, and hence:

$$A_1 = \frac{\kappa - \kappa(\kappa^2 - \epsilon\eta)}{1 - \eta^2 - \kappa^2} \frac{\sigma}{\sigma'} (I_1 - \eta I_2) = \left\{\kappa + \frac{\kappa\eta(\eta + \epsilon)}{1 - \eta^2 - \kappa^2}\right\} \frac{\sigma}{\sigma'} (I_1 - \eta I_2).$$

Similarly:

$$I_1 = \left\{\kappa + \frac{\kappa\epsilon(\eta + \epsilon)}{1 - \epsilon^2 - \kappa^2}\right\} \frac{\sigma'}{\sigma} (A_1 - \epsilon A_2).$$

These formulæ give the chief influence of age of appearance and intensity of disease in parent upon intensity and age of appearance in the offspring. If we suppose κ *positive, i.e.*, if increased age of appearance means for the diseased population as a whole increased intensity, then intensity of disease in parents tends to lower the age at which the disease appears in the offspring, and this tendency to antedate is the greater, the greater the correlation (η) between intensity of the disease in parent and child, *i.e.*, the stronger the hereditability of the disease. If κ be *negative, i.e.*, increased age of appearance means for the diseased population as a whole decreased intensity, then the opposite result will follow, for A_1 will have a less negative value than if $\eta = 0$, *i.e.*, the age of offspring be raised towards the mean.* It would thus seem possible that the antedating of inheritance in the case of gout and diabetes might correspond to a post-dating in the cases of diseases intenser in youthful incidence.

Our second formula shows that for diseases with increased intensity at increased age of appearance, a late age of appearance in the parent decreases the intensity of appearance in the offspring, while the reverse holds if the disease is intenser for youthful than for senile incidence.

It must be noted that the correlation between intensity and age without regard to heredity is given by:

$$I_1 = \kappa \frac{\sigma'}{\sigma} A_1,$$

so that heredity affects the constant of correlation κ by multiplying it by the quantity:

$$1 + \frac{\epsilon(\eta + \epsilon)}{1 - \epsilon^2 - \kappa^2}.$$

The second part of this expression is by no means necessarily negligible as compared with the first part, if heredity be strong. For example, with the order of correlation we have found between parent and offspring, in the case of stature the

* Generally but not absolutely, for $\eta^2 + \kappa^2$ for some diseases may be > 1, and, if not *very* different, then the second term is the important term.

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second term might be $\frac{1}{3}$ to $\frac{1}{4}$, while, for values of the order ·7 in the correlation coefficients, it would be a much more important term than the first, *i.e.*, heredity would completely obscure the general correlation between intensity and age.

Similar remarks apply, of course, to the formula

$$A_1 = \kappa \frac{\sigma}{\sigma'} I_1,$$

and the modification of its κ by the factor

$$1 + \frac{\eta(\eta + \epsilon)}{1 - \eta^2 - \kappa^2}.$$

While the above discussion has been adapted particularly to the problem of morbid inheritance, it should be noted that the general formulæ for triple correlation apply to a number of interesting problems on the inheritance of two faculties by the offspring from the parent. In particular, the above special formulæ in η, ϵ, and κ apply without modification to any case when (*a*) the two faculties are correlated in like manner (κ) in parent and offspring, (*b*) the two faculties are each directly inherited (η and ϵ), (*c*) there is an insensible or zero amount of cross heredity. I do not stay to develop the formulæ at present, because I hope to return to them when I have more ample statistics to illustrate the properties of cross heredity from.

(*d.*) *On the Skewness of Disease Curves.*—There is one qualifying remark which must, however, be made before we leave the topic of morbid inheritance. We have assumed that the frequency surface for intensity and age of appearance of disease is a normal correlation surface. This, however, is only an approximation. If we add together all the intensities for each age, we shall have a frequency with age curve for the disease, and if the correlation surface were a true normal surface, this would be a true normal curve. In many diseases, possibly in all, it is however, a distinctly skew curve, and this whether we take the case-frequency or the mortality-frequency. This has been illustrated in "Contributions to Mathematical Theory of Evolution, II." ('Phil. Trans.,' vol. 186, A.), Plate 12, for enteric fever.* The following statistics illustrate the same skewness for a disease more distinctly associated with heredity† :—

PHTHISIS : 2000 cases with History of Parental Phthisis.

Age . . .	1	10	15	20	25	30	35	40	45	50	55	60
Frequency	26	100	436	549	392	217	149	65	27	6	9	4

* It is, I think, true for all fevers, some of which, however, have κ positive and others κ negative.

† R. E. THOMSON, 'Family Phthisis,' p. 22, London, 1884.

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It is clear that we have here to deal with a skew curve of the kind discussed in my second memoir, and the intensity-age distribution must be a skew correlation surface to give rise to such a curve. The full treatment, accordingly, of morbid inheritance requires a discussion of skew correlation. I hope to be able to return to it again when dealing with the general theory of disease distributions. Meanwhile, the considerations of this section are based on an approximate theory, which, however, can hardly fail to give the main outlines of the subject, if a more accurate development might be requisite when actual statistics were forthcoming to be dealt with.

(10.) *Natural Selection and Panmixia.*

(*a.*) *Fundamental Theorem in Selection.*—The general theory of correlation shows us that taking $p + 1$ correlated organs, if we select p of them of definite dimensions, the remaining organ will follow a normal law of distribution, of which the standard-deviation and mean can be determined. Now, in the problem of natural selection, we do not select absolutely definite dimensions, and the p organs selected may be specially correlated together in selection, in a manner totally different from their "natural" correlation or correlation of birth. We, therefore, require a generalised investigation of the following kind: Given $p + 1$ normally correlated organs, p out of these organs are selected in the following manner: each organ is selected normally *round a given mean*, and the p selected organs, pair and pair, are correlated in any arbitrary manner. What will be the nature of the distribution of the remaining $(p + 1)^{\text{th}}$ organ?

Geometrically in p-dimensional space we have a correlation surface of the pth order among the p organs, and out of this, with any origin we please, we cut an arbitrary correlation surface of the pth order—of course, of smaller dimensions—the problem is to find the distribution of the $(p + 1)^{\text{th}}$ organ related to this arbitrary surface cut out of what we may term the natural surface.

If the p organs are organs of ancestry—as many as we please—and the $(p + 1)^{\text{th}}$ organ that of a descendant, we have here the general problem of natural selection modified by inheritance.

We will distinguish the two correlation surfaces as the unselected and the selected. Let $\beta_1, \beta_2, \beta_3, \ldots$ be the regression coefficients of the $(p + 1)^{\text{th}}$ organ on the p organs for unselected correlation, then for values of the p organs $h_1, h_2, h_3, \ldots$ from their respective means, the $(p + 1)^{\text{th}}$ organ will have a distribution centering round $\beta_1 h_1 + \beta_2 h_2 + \beta_3 h_3 + \ldots$, and a standard deviation σ given by the general theory of correlation (*i.e.*, the S.D. of the array). Similarly, for values $h_1 + x_1, h_2 + x_2, h_3 + x_3 \ldots$ of the p organs, the $(p + 1)^{\text{th}}$ will have a distribution with standard-deviation σ and centre

$$\beta_1 (h_1 + x_1) + \beta_2 (h_2 + x_2) + \beta_3 (h_3 + x_3) + \ldots = \zeta + \mathrm{S}(\beta_1 x_1), \text{ say.}$$

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Thus a deviation of the pth organ lying between v and $v + dv$ from the mean of these organs will occur with a frequency varying as

$$dv\, e^{-\frac{\{v-\zeta+S(\beta_1 x_1)\}^2}{2\sigma^2}}.$$

Now let the selected correlation surface centering round $h_1, h_2, h_3, \ldots$ be given by

$$z = \text{constant} \times e^{-\frac{1}{2}(a_{11}x_1^2 + a_{22}x_2^2 + \ldots + 2a_{12}x_1x_2 + \ldots)}.$$

Then the total frequency of the pth organ lying between v and $v + dv =$

$$\text{Constant} \times dv \int_{\infty-}^{\infty}\int_{\infty-}^{\infty}\int_{\infty-}^{\infty} \ldots e^{-\frac{\{v-\zeta-S(\beta_1 x_1)\}^2}{2\sigma^2} - \frac{1}{2}\{S(a_{11}x_1^2) + 2S(a_{12}x_1x_2)\}} dx_1 dx_2 dx_3 \ldots$$

To carry out the integrations, let us first transfer the expression in the exponential power to its "centre," writing $v - \zeta = u$, and $x_1', x_2', x_3', \ldots$ as the coordinates of the centre.

To find the "centre" we have the equations:

$$\beta_1(u - S(\beta_1 x_1'))/\sigma^2 = a_{11}x_1' + a_{12}x_2' + a_{13}x_3' + \ldots,$$
$$\beta_2(u - S(\beta_1 x_1'))/\sigma^2 = a_{21}x_1' + a_{22}x_2' + a_{23}x_3' + \ldots,$$
$$\beta_3(u - S(\beta_1 x_1'))/\sigma^2 = a_{31}x_1' + a_{32}x_2' + a_{33}x_3' + \ldots,$$
$$\ldots\ldots\ldots\ldots\ldots\ldots$$

hence

$$\Delta x_1' = (\beta_1 A_{11} + \beta_2 A_{12} + \beta_3 A_{13} + \ldots)(u - S(\beta_1 x_1'))/\sigma^2,$$
$$\Delta x_2' = (\beta_1 A_{21} + \beta_2 A_{22} + \beta_3 A_{23} + \ldots)(u - S(\beta_1 x_1'))/\sigma^2,$$
$$\Delta x_3' = (\beta_1 A_{31} + \beta_2 A_{32} + \beta_3 A_{33} + \ldots)(u - S(\beta_1 x_1'))/\sigma^2,$$
$$\ldots\ldots\ldots\ldots\ldots\ldots$$

where Δ is the determinant of the a's, and the A's are its minors, clearly $a_{ij} = a_{ji}$ and $A_{ij} = A_{ji}$. Multiplying these equations by $\beta_1, \beta_2, \beta_3 \ldots$ respectively, and adding we find

$$\begin{aligned}\sigma^2\Delta S(\beta_1 x_1') &= \{\beta_1^2 A_{11} + \beta_2^2 A_{22} + \beta_3^2 A_{33} + \ldots \\ &\qquad + 2A_{12}\beta_1\beta_2 + 2A_{13}\beta_1\beta_3 + \ldots\}(u - S(\beta_1 x'_1)) \\ &= \{S(\beta_1^2 A_{11}) + 2S(A_{12}\beta_1\beta_2)\}(u - S(\beta_1 x'_1)),\end{aligned}$$

hence

$$S(\beta_1 x_1') = \frac{\chi u}{\sigma^2\Delta + \chi}$$

where

$$\chi = S(\beta_1^2 A_{11}) + 2S(A_{12}\beta_1\beta_2).$$

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We can now transfer the exponential expression to its centre and we find for the frequency

$$\text{Constant} \times du\, e^{-\frac{u^2}{2}\left(\frac{1}{\sigma^2}-\frac{\chi}{\sigma^2\Delta+\chi}\right)} \int_{-\infty}^{\infty}\int_{-\infty}^{\infty}\int_{-\infty}^{\infty} \ldots e^{-\frac{1}{2}\left\{ S\left(x_1^2\left(a_{11}+\frac{\beta_1^2}{\sigma^2}\right)\right)+S\left(2x_1x_2\left(a_{12}-\frac{\beta_1\beta_2}{\sigma^2}\right)\right)\right\}} dx_1 dx_2 dx_3.$$

Where x_1, x_2, $x_3 \ldots$, now denote the coordinates transferred to the new origin. The integrations can then be performed without changing the u factor, and finally the frequency

$$= \text{constant} \times du\, e^{-\frac{1}{2}u^2/\left(\sigma^2+\frac{\chi}{\Delta}\right)}.$$

Hence we notice the following important results:

(a.) The $p+1^{\text{th}}$ organ follows a normal distribution.

(b.) Its standard deviation Σ is given by

$$\Sigma^2 = \sigma^2 + \beta_1^2\frac{A_{11}}{\Delta} + \beta_2^2\frac{A_{22}}{\Delta} + \ldots + 2\beta_1\beta_2\frac{A_{12}}{\Delta} + 2\beta_1\beta_3\frac{A_{13}}{\Delta} + \ldots$$

(c.) Its mean (since $u = v - \zeta$) $= \beta_1 h_1 + \beta_2 h_2 + \beta_3 h_3 + \ldots$

We conclude that

(i.) so long as selection is normal, however complex may be the system of organs selected, and however complex their correlation, the distribution of any single organ remains normal. This possibly accounts for the persistency with which normal grouping reappears in nature.

(ii.) If we select organs *varying* about any means whatever, the mean of the correlated organ resulting from this selection will be identical with the mean we should have obtained by selecting organs actually at the means of selection.

(iii.) The standard deviation of the organ which results from the selection is *not* that of an array (σ) arising from selection of the organs actually at the means, but is (as we might expect) greater. This greater variability is due to the expression

$$\beta_1^2\frac{A_{11}}{\Delta} + \beta_2^2\frac{A_{22}}{\Delta} + \ldots + 2\beta_1\beta_2\frac{A_{12}}{\Delta} + \ldots$$

which admits of the following interpretation.

Consider the selection correlation surface

$$z = \text{constant} \times e^{-\frac{1}{2}(a_{11}x_1^2 + a_{22}x_2^2 + \ldots + 2a_{12}x_1x_2 + \ldots)}$$

and give x_1 and x_2 chosen values η_1 and η_2.

Transfer the remaining variables to the "centre." The equations to do this are

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$$
\begin{aligned}
f + \eta_1 a_{11} + \eta_2 a_{12} &= \eta_1 a_{11} + \eta_2 a_{12} + a_{13} x_3' + a_{14} x_4' + \ldots \\
g + \eta_1 a_{12} + \eta_2 a_{22} &= \eta_1 a_{21} + \eta_2 a_{22} + a_{23} x_3' + a_{24} x_4' + \ldots \\
0 &= \eta_1 a_{31} + \eta_2 a_{32} + a_{33} x_3' + a_{34} x_4' + \ldots \\
0 &= \eta_1 a_{41} + \eta_{42} a_{42} + a_{43} x_3' + a_{44} x_4' + \ldots \\
&\ldots\ldots\ldots\ldots \\
&\ldots\ldots\ldots\ldots
\end{aligned}
$$

where f and g are written for $a_{13}x'_3 + a_{14}x'_4 + \ldots$ and $a_{23}x'_3 + a_{24}x'_4 + \ldots$ respectively. Solving, we find

$$
\begin{aligned}
A_{11}(f + \eta_1 a_{11} + \eta_2 a_{12}) + A_{12}(g + \eta_1 a_{12} + \eta_2 a_{22}) &= \eta_1 \Delta, \\
A_{21}(f + \eta_1 a_{11} + \eta_2 a_{12}) + A_{22}(g + \eta_1 a_{12} + \eta_2 a_{22}) &= \eta_2 \Delta.
\end{aligned}
$$

Hence

$$
\begin{aligned}
f &= \frac{\eta_1 A_{22} - \eta_2 A_{12}}{A_{11}A_{22} - A_{12}^2}\Delta - \eta_1 a_{11} - \eta_2 a_{12}, \\
g &= \frac{\eta_2 A_{11} - \eta_1 A_{12}}{A_{11}A_{22} - A_{12}^2}\Delta - \eta_1 a_{12} - \eta_2 a_{22}.
\end{aligned}
$$

But the exponential expression with its origin changed is given by

$$
\begin{aligned}
z = \text{constant} &\times e^{-\frac{1}{2}(a_{11}\eta_1^2 + 2a_{12}\eta_1\eta_2 + a_{22}\eta_2^2 + f\eta_1 + g\eta_2)} \\
&\times e^{-\frac{1}{2}(a_{33}x_3^2 + a_{44}x_4^2 + \ldots + 2a_{34}x_3x_4 + \ldots)}.
\end{aligned}
$$

Integrating between the limits $\pm\infty$ for all the variables $x_3, x_4, x_5 \ldots$, we shall have the correlation surface for η_1, η_2, or substituting for f and g

$$
z' = \text{constant} \times e^{-\frac{1}{2}\frac{\Delta}{1-(A_{12}^2/A_{11}A_{22})}\left\{\frac{\eta_1^2}{A_{11}} + \frac{\eta_2^2}{A_{22}} - 2\eta_1\eta_2\frac{A_{12}}{A_{11}A_{22}}\right\}}.
$$

Comparing this with the formula on p. 264, we see that if ρ_{12} be the correlation coefficient of x_1, x_2 and s_1, s_2 their standard deviations

$$
\rho_{12}^2 = A_{12}^2/A_{11}A_{22} \quad s_1^2 = A_{11}/\Delta \quad s_2^2 = A_{22}/\Delta \quad \text{or} \quad \rho_{12}s_1s_2 = A_{12}/\Delta \quad . \quad . \quad (\epsilon).
$$

Thus we conclude that the standard deviation for the organ resulting from the selection is given by

$$
\Sigma^2 = \sigma^2 + \beta_1^2 s_1^2 + \beta_2 s_2^2 + \ldots + 2\beta_1\beta_2\rho_{12}s_1s_2 + \ldots
$$

Here $\sigma, \beta_1, \beta_2 \ldots$ refer to the natural or unselected correlation surface, and $s_1, s_2, \ldots \rho_{12} \ldots$ to the selection correlation surface.

(*b.*) EDGEWORTH'S *Theorem.*—We may stay for a moment over the results (ϵ) above in order to deduce Professor EDGEWORTH'S Theorem,* which we shall shortly require to use. By the theory of minors (SALMON'S 'Higher Algebra,' 1866, p. 24) we have

* Briefly stated with some rather disturbing printer's errors in the 'Phil. Mag.,' vol. 34, p. 201, 1892.

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$$\Delta^{p-1} = \begin{vmatrix} A_{11}, & A_{12}, & A_{13} \ldots \\ A_{21}, & A_{22}, & A_{23} \ldots \\ A_{31}, & A_{32}, & A_{33} \ldots \\ \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot \end{vmatrix},$$

$$= \Delta^p s_1^2 s_2^2 s_3^2 \ldots \begin{vmatrix} 1, & \rho_{12}, & \rho_{13} \ldots \\ \rho_{21}, & 1, & \rho_{23} \ldots \\ \rho_{31}, & \rho_{32}, & 1 \ldots \\ \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot \end{vmatrix}.$$

Hence $1/\Delta = s_1^2 s_2^2 s_3^2 \ldots R$, where R is the determinant formed by the correlation coefficients with a diagonal of units.

Further, if B_{11}, $B_{22} \ldots B_{12} \ldots$ be the minors of the A-determinant, and R_{11}, $R_{22}, \ldots R_{12}, \ldots$ of the R-determinant, we have (SALMON, *loc. cit.*):

$$a_{11} = B_{11}/\Delta^{p-2} = \Delta R_{11} s_1^2 s_2^2 s_3^2 \ldots / s_1^2 = R_{11}/(R s_1^2),$$
$$a_{22} = B_{22}/\Delta^{p-2} = \Delta R_{22} s_1^2 s_2^2 s_3^2 \ldots / s_2^2 = R_{22}/(R s_2^2),$$
$$a_{12} = B_{12}/\Delta^{p-2} = \Delta R_{12} s_1^2 s_2^2 s_3^2 \ldots / s_1 s_2 = R_{12}/(R s_1 s_2).$$
$$\cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot$$

Thus, the correlation surface may be written

$$z = \frac{\mu n}{(2\pi)^{\frac{1}{2}p} s_1 s_2 s_3 \ldots \sqrt{R}} e^{-\frac{1}{2R}\left(R_{11}\frac{x_1^2}{s_1^2} + R_{22}\frac{x_2^2}{s_2^2} + \ldots + 2R_{12}\frac{x_1 x_2}{s_1 s_2} + \ldots\right)}$$

where n is the total number of sets of p organs and μ is a numerical factor denoting the number of $(p+1)^{\text{th}}$ organs corresponding to each set—in inheritance what may be termed a factor of reproductivity*—which is assumed to be practically constant, if not over the whole unselected correlation surface, at least over the selected portion of it.

(*c.*) *Selection of Parentages. Correlation Coefficients for Ancestry.*—The results on p. 300 and p. 301 for the regression ζ and the standard-deviation Σ when p correlated organs are arbitrarily selected about p means will, I think, be found to express the chief features of natural selection. A few special corollaries may follow here.

Cor. 1.—If a single parentage be selected with mean h_1 above the mean of the general population and standard deviation s_1, then $\beta_1 = r_{01}\frac{\sigma_0}{\sigma_1}$, where r_{01} is the

* The variation of this factor is, however, the essential feature of reproductive selection, as I shall show on another occasion.

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correlation coefficient of parent and offspring, and σ_1, σ_0 their standard-deviations in the unselected state. Thus we have

$$\xi = r_{01}\frac{\sigma_0}{\sigma_1}h_1, \quad \Sigma^2 = \sigma_0^2(1 - r_{01}^2) + r_{01}^2\frac{\sigma_0^2}{\sigma_1^2}s_1^2.$$

If the parent and offspring are of the same sex and there be no reproductive selection, $\sigma_0 = \sigma_1$, and we have

$$\xi = r_{01}h, \quad \Sigma^2 = \sigma_0^2(1 - r_{01}^2) + r_{01}^2 s_1^2.$$

Cor. 2.—If a bi-parentage be selected with parental means h_1, h_2, standard-deviations s_1, s_2, and coefficient of assortative mating ρ_{12}, then

$$\begin{aligned}\xi &= \frac{r_{01} - r_{12}r_{02}}{1 - r_{12}^2}\frac{\sigma_0}{\sigma_1}h_1 + \frac{r_{02} - r_{12}r_{01}}{1 - r_{12}^2}\frac{\sigma_0}{\sigma_2}h_2,\\ &= \beta_1 h_1 + \beta_2 h_2,\\ \Sigma^2 &= \sigma_0^2(1 - r_{01}^2 - r_{02}^2 - r_{12}^2 + 2r_{01}r_{02}r_{12}) + \beta_1^2 s_1^2 + \beta_2^2 s_2^2 + 2\beta_1\beta_2\rho_{12}s_1 s_2.\end{aligned}$$

Let us use these results to investigate how the offspring of a selected parentage or bi-parentage degenerate. At first sight, it would appear that with our general proposition the discussion of the effect of p selections would be perfectly straightforward. So it is, but the conclusion which follows, although it might have been foreseen, is remarkable in its consequences. We have only to calculate out the β's for p selected ancestors, and we obtain the regression ζ in the descendant by putting in the values h_1, h_2, h_3, . . . of the means of the selected ancestors. For example, suppose now a parent, a grandparent, and a great-grandparent to have been selected. We can find the β's at once from the results on p. 294. If 1, 2, 3, 4 denote the successive generations, and r the correlation coefficient of parent and offspring, we find

$$r_{12} = r,\ r_{13} = r^2,\ r_{14} = r^3,\ r_{23} = r,\ r_{24} = r^2,\ r_{43} = r,$$

whence we deduce at once

$$\begin{aligned}\lambda_1 &= 1 - 2r^2 + r^4, & \nu_{12} &= r(1 - 2r^2 + r^5),\\ \nu_{13} &= \nu_{14} = 0, & 1/\chi &= (1 - r^2)^3,\end{aligned}$$

or

$$\beta_1 = r\frac{\sigma_1}{\sigma_2}, \quad \beta_2 = \beta_3 = 0. \quad \Sigma_1 = \sigma_1\sqrt{1 - r^2}.$$

Similarly, if we take offspring (1), parent (2), and maternal and paternal of the same sex, grandparents (3 and 4), we have:

$$r_{12} = r,\ r_{13} = r^2,\ r_{14} = r^2,\ r_{23} = r,\ r_{24} = r,\ r_{34} = 0,$$

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whence

$$\lambda_1 = 1 - 2r^2, \quad \nu_{12} = r(1 - 2r^2), \quad \nu_{13} = \nu_{14} = 0, \quad 1/\chi = 1 - 3r^2 + 2r^4,$$

or,

$$\beta_1 = r\frac{\sigma_1}{\sigma_2}, \quad \beta_2 = \beta_3 = 0, \quad \Sigma_1 = \sigma_1\sqrt{1 - r^2}.$$

Thus we see that in both cases the grandparents *are quite indifferent*, when the immediate parent has been selected.

These theorems can be at once generalised by means of EDGEWORTH's theorem. Suppose we select a complete parentage for p generations in the case of parthenogenetic reproduction, or a parentage of one sex, say males, in the case of sexual reproduction, then in either case our scheme of subscripts of the correlation-coefficients, $\longrightarrow$ marking a generation, is

$$1 \longrightarrow 2 \longrightarrow 3 \longrightarrow 4 \longrightarrow 5 \longrightarrow \ldots \longrightarrow p$$

and

$$R = \begin{vmatrix} 1 & r & r^2 & r^3 \ldots r^{p-1} \\ r & 1 & r & r^2 \ldots r^{p-2} \\ r^2 & r & 1 & r \ldots r^{p-3} \\ \cdot & \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot & \cdot \\ r^{p-1} & r^{p-2} & r^{p-3} \ldots & 1 \end{vmatrix}.$$

Multiply the second line by r, and subtract from the first, and we have

$$R = (1 - r^2)\,R_{11}.$$

Take R_{1q} $(q < p)$, and we have

$$R_{1q} = \begin{vmatrix} r & 1 & r & r^2 \ldots r^{q-3}, r^{q-1} \ldots r^{p-2} \\ r^2 & r & 1 & r \ldots r^{q-4}, r^{q-2} \ldots r^{p-3} \\ \cdot & \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot & \cdot \\ r^{p-1} & r^{p-2} \ldots & & r^{p-q+2}, r^{p-q} \ldots 1 \end{vmatrix}.$$

Multiply the second column by r, and subtract from the first, and we have $R_{1q} = 0$ if $q > 2$.

If $q = 2$, we have

$$R_{12} = \begin{vmatrix} r & r & r^2 \ldots r^{p-2} \\ r^2 & 1 & r \ldots r^{p-3} \\ \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot \\ r^{p-1} & r^{p-3} \ldots & 1 \end{vmatrix},$$

or, dividing the first column by r, $\quad R_{12} = rR_{11}$.

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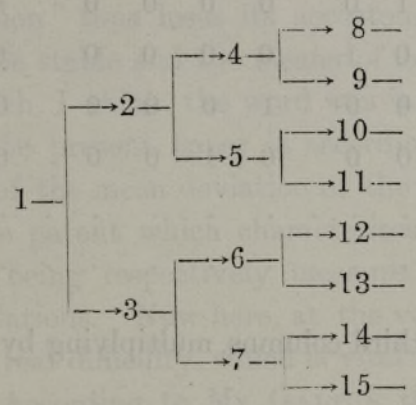
Hence $\zeta = r\frac{\sigma_1}{\sigma_2}h_2$, and $\sigma'^2 = \sigma_1^2(1 - r^2)$,
or precisely the results we should have obtained by selecting only the immediate parent.

To simplify the analysis for biparental selection, assume that the correlation coefficients of both parents are equal, and that there is no assortative mating.

We have the scheme for the correlation-coefficients subscripts, $\longrightarrow$ marking a generation:



and so on.

Thus r_{mn} is at once expressible as zero, or a power of r, the simple coefficient of correlation for parent and offspring, according as m and n do not or do lie in the direct descent.

Hence we find

$$
\mathrm{R} = \begin{vmatrix}
1 & r & r & r^2 & r^2 & r^2 & r^2 & r^3 & r^3 & r^3 & r^3 & r^3 & r^3 & r^3 & r^3 \ldots \\
r & 1 & 0 & r & r & 0 & 0 & r^2 & r^2 & r^2 & r^2 & 0 & 0 & 0 & 0 \ldots \\
r & 0 & 1 & 0 & 0 & r & r & 0 & 0 & 0 & 0 & r^2 & r^2 & r^2 & r^2 \ldots \\
r^2 & r & 0 & 1 & 0 & 0 & 0 & r & r & 0 & 0 & 0 & 0 & 0 & 0 \ldots \\
r^2 & r & 0 & 0 & 1 & 0 & 0 & 0 & 0 & r & r & 0 & 0 & 0 & 0 \ldots \\
r^2 & 0 & r & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & r & r & 0 & 0 \ldots \\
r^2 & 0 & r & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & r & r \ldots \\
r^3 & r^2 & 0 & r & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \ldots \\
r^3 & r^2 & 0 & r & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \ldots \\
r^3 & r^2 & 0 & 0 & r & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \ldots \\
r^3 & r^2 & 0 & 0 & r & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \ldots \\
r^3 & 0 & r^2 & 0 & 0 & r & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \ldots \\
r^3 & 0 & r^2 & 0 & 0 & r & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \ldots \\
r^3 & 0 & r^2 & 0 & 0 & 0 & r & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \ldots \\
r^3 & 0 & r^2 & 0 & 0 & 0 & r & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \ldots \\
\cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\
\cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot
\end{vmatrix}
$$

Add the second and third rows, multiply them by r and subtract from the first, and we find:

$$\mathrm{R} = (1 - 2r^2)\,\mathrm{R}_{11}.$$

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If $q > 3$ and $< p$ we have

$$
R_{1q} = \left|\begin{array}{cccccccccccccccc}
r & 1 & 0 & r & r & 0 & 0 & r^2 & r^2 & r^2 & r^2 & 0 & 0 & 0 & 0 & \ldots \\
r & 0 & 1 & 0 & 0 & r & r & 0 & 0 & 0 & 0 & r^2 & r^2 & r^2 & r^2 & \ldots \\
r^2 & r & 0 & 1 & 0 & 0 & 0 & r & r & 0 & 0 & 0 & 0 & 0 & 0 & \ldots \\
r^2 & r & 0 & 0 & 1 & 0 & 0 & 0 & 0 & r & r & 0 & 0 & 0 & 0 & \ldots \\
r^2 & 0 & r & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & r & r & 0 & 0 & \ldots \\
r^2 & 0 & r & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & r & r & \ldots \\
r^3 & r^2 & 0 & r & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \ldots \\
r^3 & r^2 & 0 & r & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & \ldots \\
\cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\
\cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot & \cdot
\end{array}\right|
$$

Whence, adding the second and third columns, multiplying by r and subtracting from the first, we have $R_{1q} = 0$.

Lastly, $R_{12} = rR_{11}$ and $R_{13} = rR_{11}$; for R_{12} is of the form of R_{1q} above without the second column. Divide the first column by r, and subtract the second column (the *third* of R_{1q} above) and it becomes the first of R_{11}, the remainder is identical in R_{12} and R_{11}. Hence, $R_{12} = rR_{11}$. Similarly we find R_{13}. Thus we conclude that

$$\zeta = r\,\frac{\sigma_1}{\sigma_2}\,h_2 + r\,\frac{\sigma_3}{\sigma_3}\,h_3, \qquad \sigma'^2 = \sigma_1^2\,(1 - r^2 - r^2).$$

the formulæ for biparental inheritance with equal parental correlation, and no assortative mating. The analysis for unequal parental correlation and assortative mating follows the same lines, is far more lengthy, but leads to the same result, *i.e.*, no gain by selection of the same amount, oft repeated.

(*d.*) *Secular Natural Selection and Steady Selection. Focus of Regression.*—We thus see that, on the theory with which we are concerned, a knowledge of the ancestry beyond the parents in no way alters our judgment as to the size of organ or degree of characteristic probable in the offspring, nor its variability.* *An exceptional father is as likely to have exceptional children if he comes of a mediocre stock as if he comes of an exceptional stock.* The value of ζ will be no greater nor the value of Σ_1 less if the parents have been selected for p generations than if they have been selected for one only. This result seems to me somewhat surprising, but I cannot see how it is to be escaped so long as we assume the normal distribution of frequency, which appears in so many cases to be a close approximation to fact. It is of course possible

* This seems specially noteworthy; it would seem natural to suppose that the offspring of a long selected stock would be less variable than those of one just started—that the offspring of a gradually created variety would be more stable than those so to speak of a sport. It appears not.

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that in some manner repeated selection causes a progression of the "focus of regression," by which term I would understand the mean of the general population from which selection has originally taken place. I have been very careful so far not to hazard any statement with regard to this focus of regression. I have measured only the amount by which the offspring of exceptional parents diverge, not from the mean of the parental population but from the mean of the offspring population. In this manner our formulæ allowed for the play of secular natural selection. It is quite true that the word "regression" thus loses its accustomary meaning, which it can only bear if the population be stable and the means of two generations sensibly identical; this is the case for which, I think, the word was introduced by Mr. GALTON.* The sense given to it in the present paper is accordingly a technical one; as already defined, it is the ratio of the mean deviation of the offspring of a selected parentage to the deviation in the parent which characterises the selection, the deviations in offspring and parents being respectively measured from the means of the corresponding general populations. Now here, at the very outset of our consideration of panmixia arises a very real difficulty, which is vital for the whole theory of evolution by natural selection. According to Mr. GALTON the population being stable, or no secular natural selection or reproductive selection taking place, there is a regression of the offspring of selected parents towards the mean of a certain general population, and the "grandchildren" also regress to the same mean. We shall see then that unless correlation is perfect $\left(r\,\frac{\sigma_2}{\sigma_1} = 1\right)$ no amount of continued selection would suffice to prevent a race from regressing to an original general population when that selection was suspended. Panmixia in the sense of its most ardent supporters would be demonstrated. But the difficulty is not the establishment of panmixia, but as to what is to be considered the "original general population." On the theory of evolution by natural selection that general population has itself been produced by a series of selections, and selections probably affecting its mean as well as its standard-deviation, hence how is it possible to pick out any particular stage of general population as the "focus of regression," and assert that regression of the offspring of parents now selected takes place towards that stage of evolution? Where is the focus of regression to be placed for the profile angle of man? About 80° to 90° or nearer the 40° to 70° of the anthropoid apes? The further back the better for those who believe that suspension and reversal of natural selection are identical, but no manipulating whatever of the human mortality tables would allow for a "focus of regression" very considerably below that of the current general population. Hence it would seem essential that successive selections must connote some progression of the focus of regression. This progression may be continuous with continuous natural selection, or it may take place by starts and leaps, as

* We can at once restore the true notion of regression, as Mr. GALTON points out to me, by measuring each organ or characteristic in terms of its own standard-deviation. It will then be a coefficient of correlation and a proper fraction.

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indicated in Mr. GALTON's idea of organic stability. In either case panmixia would only carry back the mean to the current focus of regression, and so be a very minute reversal of natural selection.

What our theory really shows is a regression of the offspring of selected parents towards the mean of general offspring. This latter mean, supposing no secular natural selection, can, it seems to me, only be determined by experiment. It can hardly agree with the general parental mean, if the parents themselves are the product of natural selection. On the other hand, the statistics actually obtained for stable, or sensibly stable, populations seem to mark a focus of regression close to the mean of the current population, and, therefore, a progression of the focus due to past selection.* Meanwhile, till experiment has settled how continuous selection affects the focus of regression, we may see whither extreme hypotheses lead us. Such are:

(1.) The focus of regression remains stable during selection.

(2.) The focus of regression is the mean of the population from which parents have been selected.

(*e.*) *Focus of Regression Stable during Selection.*

(i.) *Steady Selection cannot be Secular or Produce Truer Breeding.*—We have seen that on this hypothesis ancestry, as distinct from immediate parentage, is indifferent. Thus, in the case of parthenogenetic reproduction, or of sexual reproduction with one parent selected, we have seen that one selection leads to the distribution (Cor. 1, p. 301):

$$\zeta = r_{01}\frac{\sigma_0}{\sigma_1}h_1, \quad \Sigma^2 = \sigma_0^2(1 - r_{01}^2) + r_{01}^2\frac{\sigma_0^2}{\sigma_1^2}s_1^2,$$

and if out of this we again select a parentage, defined by h_1 and s_1, we shall obtain the same distribution of offspring, and this however often the process be repeated. We must increase the divergence (h_1) of the selected from the general population or its concentration ($1/s_1$) or both, if we require any progressive effect from continual selection. The same remarks apply to bi-parental selection (m_1, m_2, s_2, s_3, ρ_{12}). Persistent selection only suffices to keep the mean and variation at a definite distance from those of the general population. Or, on the hypothesis of a stationary focus of regression, we conclude that *steady selection, however long it persists, can only be periodic and not secular.*

This point seems of such importance that it may be best to illustrate it by an example drawn from our Table I. and Table III. The mean height of fathers being about 69″·2, the regression of the average sons of fathers of 6′ in height is about 1″·25, or the average height of sons of a 6′ fatherhood = 70″·45. Hence, if we select fathers forming a normal distribution of any standard round 6′, we shall have a normal distribution of sons round 70″·45. If we select a second parentage from

* The determination of the focus of regression for some organ in selected domestic ducks for several generations and comparison with the means for wild and general domestic ducks would seem a possibility.

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those taller sons averaging 6′, their sons will still only average 70″·45, and, however long we persist in this process of selection, we shall produce no secular change; the population will remain after the pth selection just where it was both as to mean and variation after the first. The only way to produce a secular change is to continually increase the standard of the selected (or to alter the focus of regression). No steady selection would appear to produce "truer breeding."

(ii.) *Panmixia and Uni-parental Regression.*—Continual selection of the same magnitude for p generations, merely giving us the same mean and variation, we may now ask what would be the effect of suspending natural selection for q generations.

Take first the case of parthenogenetic reproduction, or that of uni-parental regression. The first parentage after suspension of natural selection will have $m_2 r_3 \sigma_1/\sigma_2$ for its mean, and $\sqrt{\{\sigma_1^2 (1 - r_3^2) + s_2^2 r_3^2 \sigma_1^2/\sigma_2^2\}}$ for its standard-deviation. Successive parentages can be found by substituting these values successively in themselves for the quantities m_2 and s_2. We find at once that after q generations of suspended selection the mean of the population will differ from the focus of regression by

$$m_2 (r_3 \sigma_1/\sigma_2)^q,$$

and the standard-deviation will be given by

$$\Sigma_q^2 = \sigma_1^2 (1 - r_3^2) \frac{1 - (r_3 \sigma_1/\sigma_2)^{2q-2}}{1 - (r_3 \sigma_1/\sigma_2)^2} + (r_3 \sigma_1/\sigma_2)^{2q-2} s_1^2.$$

Now if the population simply repeat itself without any natural selection (if there be no reproductive selection at work) $\sigma_1 = \sigma_2$, and in most cases I have come across $r_3 \sigma_1/\sigma_2$ is a fraction. Hence, as q is indefinitely increased $m_2 (r_3 \sigma_1/\sigma_2)^q$ becomes indefinitely small, and $\Sigma_q^2 = \sigma_1^2$, or $= \sigma_1^2 \dfrac{1 - r_3^2}{1 - (r_3 \sigma_1/\sigma_2)^2}$, if σ_1 be not equal to σ_2.

We see, therefore, that both as to mean and variation the population with suspended natural selection tends to rapidly regress to the general population from which it was selected. This is still true if there has been a continuous secular, as distinguished from a periodic natural selection, for we have only to suppose m_2 and s_2 to be the final result of such selection. If then the focus of regression does not progress with continuous selection, all that has been asserted as to the effect of suspended natural selection holds, at least so far as concerns a return to the condition of things which prevailed when the focus of regression was the mean of the general population. But unfortunately the advocates of panmixia want more than this, namely, either an indefinite regression of the focus of regression itself, or to place it, if steady, at an indefinitely distant point. The first result would be perfectly parallel with our second hypothesis—a progression of the focus of regression,—but would demand rather a reversal than a suspension of natural selection. The second result seems quite inconsistent with any statistics of successive genera-

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tions yet taken; it demands a mortality due to natural selection, which its propounders have hardly appreciated.

(iii.) *Panmixia and Bi-parental Regression.*—The process by which corresponding results may be deduced for bi-parental selection may now be briefly indicated.

We suppose both natural selection and assortative mating to have gone on in any manner for any number of generations, the final effect, however, if the focus of regression be not changed, will be:

$$\text{Mean of males} = \beta_2 m_2 + \beta_3 m_3 = \mu_1, \text{ say,}$$
$$\text{Mean of females} = \beta'_2 m_2 + \beta'_3 m_3 = \mu'_1, \text{ say,}$$
$$(\text{S.D. of males})^2 = \sigma^2 + \beta_2^2 s_2^2 + \beta_3^2 s_3^2 + 2\beta_2\beta_3 s_2 s_3 \rho = \epsilon_1^2,$$
$$(\text{S.D. of females})^2 = \sigma'^2 + \beta'^2_2 s_2^2 + \beta'^2_3 s_3^2 + 2\beta'_2\beta'_3 s_2 s_3 \rho = \eta_1^2,$$

where m_2, m_3, s_2, s_3, ρ defines the last step of the natural and sexual selections, and β'_2, β'_3 are the regression-coefficients for females.

Now, selection of all sorts ceasing, we must use for the regression-coefficients no longer their values modified by sexual selection, but simply:

$$\beta_2 = r_3 \sigma_1/\sigma_2, \qquad \beta_3 = r_2 \sigma_1/\sigma_3,$$
$$\beta'_2 = r'_3 \sigma'_1/\sigma'_2, \qquad \beta'_3 = r'_2 \sigma'_1/\sigma'_3,$$
$$\sigma^2 = \sigma_1^2 (1 - r_2^2 - r_3^2) \qquad \sigma'^2 = \sigma'^2_1 (1 - r'^2_2 - r'^2_3),$$

obtained from the general values, p. 287, by putting $r_1 = 0$. Here r'_2 and r'_3 are respectively the maternal and paternal correlation coefficients for inheritance in the female line. Further, we have very closely $\sigma_1 = \sigma_2 = \sigma'_2$ and $\sigma'_1 = \sigma'_3 = \sigma_3$. If μ_p, μ'_p give the male and female means, ϵ_p, η_p the male and female standard-deviations, after p generations in which natural and sexual selection have both been suspended, we have:

$$\mu_p = \beta_2 \mu_{p-1} + \beta_3 \mu'_{p-1},$$
$$\mu'_p = \beta'_2 \mu_{p-1} + \beta'_3 \mu'_{p-1},$$
$$\epsilon_p^2 = \sigma^2 + \beta_2^2 \epsilon_{p-1}^2 + \beta_3^2 \eta_{p-1}^2,$$
$$\eta_p^2 = \sigma'^2 + \beta'^2_2 \epsilon_{p-1}^2 + \beta'^2_3 \eta_{p-1}^2.$$

Solving the equations for the means first, we have:

$$\mu_p = A_1 \gamma^{p-1}_1 + A_2 \gamma^{p-1}_2,$$
$$\mu'_p = A_1 \frac{(\gamma_1 - \beta_2)}{\beta_3} \gamma_1^{p-1} + A_2 \frac{\gamma_2 - \beta_2}{\beta_3} \gamma_2^{p-1},$$

where

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$$A_1 = \frac{\mu'_1\beta_3 + \mu_1(\beta_2 - \gamma_2)}{\gamma_1 - \gamma_2},$$

$$A_2 = -\frac{\mu'_1\beta_3 + \mu_1(\beta_2 - \gamma_1)}{\gamma_1 - \gamma_2},$$

and

$$\left.\begin{matrix}\gamma_1\\ \gamma_2\end{matrix}\right\} = \tfrac{1}{2}\{\beta_2 + \beta'_3 \pm \sqrt{(\beta_2 - \beta'_3)^2 + 4\beta_3\beta'_2}\}.$$

Since the β's for parental inheritance will be $< \cdot 5$, it follows that γ_1 and γ_2 are proper fractions, hence by taking p sufficiently large, we can make μ_p and μ'_p as small as we please.

This result is equally true whether the β's be those for assortative mating or not. Thus we conclude that suspended natural selection, whether accompanied by sexual selection or not, would ultimately result in a regression of means to the foci of regression of the two sexes.

(iv.) *Panmixia for Human Stature.*—It is worth while illustrating this by an example. Let us suppose that owing to natural selection, the *mean* of the male human population were pushed up to 4″ above its present level, and the *mean* of the female population were pushed up 3″ above its present level, and then let us inquire how they would regress in p generations of suspended natural selection with and without that factor of sexual selection we have termed assortative mating.

(a.) *Without Assortative Mating.*—We must take the values of the β's from Table III. :

$$\beta_2 = \cdot 4456, \quad \beta_3 = \cdot 3384, \quad \beta'_2 = \cdot 3096, \quad \beta'_3 = \cdot 2932.$$

Further

$$\mu_1 = 4'', \qquad \mu'_1 = 3''.$$

We find

$$\gamma_1 = \cdot 7069, \qquad \gamma_2 = \cdot 0419,$$
$$A_1 = 3\cdot 9549, \qquad A_2 = \cdot 0451,$$

whence

$$\mu_p = 3\cdot 9549\,(\cdot 7067)^{p-1} + \cdot 0451\,(\cdot 0419)^{p-1},$$
$$\mu'_p = 3\cdot 0538\,(\cdot 7067)^{p-1} - \cdot 0538\,(\cdot 0419)^{p-1}.$$

Thus, in four generations ($p = 5$) the males will have sunk to ·9876″ and the females to ·7626″ from the old means* before natural selection started, while in nine generations ($p = 10$), the mean of the males will have sunk to ·2036″, and the mean of the females to ·1816″ from the old means; thus the means of the general populations of both sexes have been sensibly carried back by panmixia to the focus of regression.

* The smallness of the contributions given by the second terms in the values of μ_p, μ'_p is to be noted.

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(b.) *With Assortative Mating.*—We must take the values of the β's from Table XIII.:

$$\beta_2 = \cdot 4176, \quad \beta_3 = \cdot 2997, \quad B'_2 = \cdot 2895, \quad B'_3 = \cdot 2609.$$

We find

$$\gamma_1 = \cdot 6440 \qquad \gamma_2 = \cdot 0344,$$
$$A_1 = 3\cdot 9893 \qquad A_2 = \cdot 0107,$$

whence

$$\mu_p = 3\cdot 9893\,(\cdot 6440)^{p-1} + \cdot 0107\,(\cdot 0344)^{p-1},$$
$$\mu'_p = 3\cdot 0136\,(\cdot 6440)^{p-1} - \cdot 0136\,(\cdot 0344)^{p-1}.$$

As before, we note the small importance of the second terms. After four generations ($p = 5$), we have $\mu_p = \cdot 6862$ and $\mu'_p = \cdot 5184$; while after nine generations we have $\mu_p = \cdot 1180$ and $\mu'_p = \cdot 0892$.

Now the effect of assortative mating here, even so little of it as may be detected in regard to stature in human mating, is of the exactly opposite character to what some of the current language on panmixia would have led us to believe. *The more assortative mating the more rapid is the regression.* The maximum of regression would be reached, if this factor of sexual selection exhibited perfect correlation.* Hence, assortative mating, if unaccompanied by a stringent natural selection, appears rather to emphasize than retard the action of panmixia.

(v.) *Effect of Panmixia on Variation.*—We now turn to the second part of our problem, the determination of the standard deviations after p generations of suspended natural selection and assortative mating. This involves the solution of the equations ϵ_p and η_p on p. 310.

We find

$$\epsilon_p^2 = \epsilon_1^2 + C_1 \frac{g_1^{p-1} - 1}{g_1 - 1} + C_2 \frac{g_2^{p-1} - 1}{g_2 - 1},$$

$$\eta_p^2 = \eta_1^2 + C_1 \frac{g_1 - \beta_2^2}{\beta_3^2} \frac{g_1^{p-1} - 1}{g_1 - 1} + C_2 \frac{g_2 - \beta_2^2}{\beta_3^2} \frac{g_2^{p-1} - 1}{g_2 - 1},$$

where

$$\left.\begin{matrix} g_1 \\ g_2 \end{matrix}\right\} = \tfrac{1}{2}\left\{\beta_2^2 + \beta_3'^2 \pm \sqrt{(\beta_2^2 - \beta_3'^2)^2 + 4\beta_3^2\beta_2'^2}\right.,$$

and C_1 and C_2 are to be found from

* This is not absolutely accurate, for r_2 and r_3 are not equal, so that all the β's do not take their smallest value for $r_1 = 1$. But assuming r_2 and r_3, r'_2 and r'_3 not very sensibly different, the result stated would practically follow. The whole reasoning in the text is, indeed, subject to another limitation, it is supposed that the constants of parental inheritance and of assortative mating are independent and characteristic of the race. The former, however, may really depend upon the latter. The dependence is very improbably so close as to reverse the principle stated.

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$$C_1 + C_2 = \sigma^2 + (\beta_2^2 - 1)\,\epsilon_1^2 + \beta_3^2\eta_1^2,$$

$$C_1g_1 + C_2g_2 = \beta_2^2\,(\sigma^2 + (\beta_2^2 - 1)\,\epsilon_1^2 + \beta_3^2\eta_1^2) + \beta_3^2\,(\sigma'^2 + \beta_2'^2\epsilon_1^2 + (\beta_3'^2 - 1)\,\eta_1^2).$$

ϵ_p^2 and η_p^2 can then be found at once, if the values of the constants are known.

Remembering that g_1 and g_2 will be proper fractions, we can easily find the effect of continued panmixia by putting $p = \infty$.

We have

$$\epsilon_\infty^2 = \epsilon_1^2 + \frac{C_1 + C_2 - C_1g_2 - C_2g_1}{g_1g_2 - (g_1 + g_2) + 1} = \frac{\sigma^2\,(1 - \beta_3'^2) + \sigma'^2\beta_3^2}{\beta_2^2\beta_3'^2 - \beta_3^2\beta_2'^2 - \beta_2^2 - \beta_3'^2 + 1},$$

after some rather lengthy reductions. Similarly

$$\eta_\infty^2 = \frac{\sigma^2\beta'^2_2 + \sigma'^2\,(1 - \beta_2^2)}{\beta_2^2\beta'^2_3 - \beta_3^2\beta'^2_2 - \beta_2^2 - \beta'^2_3 + 1}.$$

If we substitute in these the values of the β's, and of σ and σ' given on *p.* 310, we find:

$$\epsilon_\infty = \sigma_1, \qquad \eta_\infty = \sigma'_1.$$

Thus we see that indefinitely prolonged panmixia carries back not only the means of both sexes, but their distributions about the means to the state of affairs when the foci of regression were themselves the means of the population.*

The all-important question concerning panmixia is, as we have seen, that of the position and stability of the focus of regression, and it seems to me that this is a question which it is only possible to settle by experiments. Nor do the experiments, at least from the theoretical standpoint, seem attended by difficulties which are insuperable. It is not necessary to select a parthenogenetically reproductive race, it is not necessary even to select both parents, it would be sufficient to deal with the regression from one selected parent, if this were most convenient.† The simple test is this:—If M_1 be the mean of selected parents, m_1 the mean of their offspring, and M_2 be the mean of another group of selected parents (*e.g.*, selected out of the

* In order to ascertain whether the standard deviations would return to their old values, supposing natural selection to be suspended, but assortative mating maintained, we should have to solve a series of equations of the type:

$$\epsilon_p^2 = \sigma^2 + \beta_2^2\epsilon_{p-1}^2 + \beta_3^2\eta_{p-1}^2 + 2\beta_2\beta_3 r_1\epsilon_{p-1}\eta_{p-1},$$

$$\eta_p^2 = \sigma'^2 + \beta'^2_2\epsilon_p r + \beta'^2_3\eta_{p-1}^2 + 2\beta'_2\beta'_3 r_1\epsilon_{p-1}\eta_{p-1},$$

and then substitute the values of σ_1, σ_1' and the β's from p. 286 in ϵ_∞ and η_∞. I have not yet solved these equations. In turning the above formulae into numbers, the caution given in the footnote, p. 312, must be borne in mind, *i.e.*, the correlation coefficients for inheritance during assortative mating may differ somewhat from those holding when it is suspended.

† Perhaps a common father and series of selected mothers would give the best results.

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group m_1 by any series of selections and breedings) and m_2 their offspring-mean, is $(M_2 m_1 - M_1 m_2)/(M_2 - m_2 - M_1 + m_1)$ constant for all stages of selection? If it be, it is the stable focus of regression, and $1 - \frac{m_1 - m_2}{M_1 - M_2}$ is the coefficient of regression.

(f.) *Progression of the Focus of Regression with Natural Selection.*

(i.) *General Remarks on Regression and Fixedness of Character.*—Our first hypothesis certainly favours the general views of those who support the doctrine of panmixia, although to be quite consistent they must:

(i.) Place the focus of regression back at the zero size of an organ or the zero degree of intensity of a characteristic.

(ii.) Assume much nearer approximation to unity in their coefficients of regression than any measurement as yet suggests, or

(iii.) Demand a far higher mortality of *periodic* natural selection than has anywhere as yet been demonstrated.

Professor WEISMANN has no difficulty, apparently, about (i.): "As soon as natural selection ceases to operate upon any character, structural or functional, it begins to *disappear*." ("Essays on Heredity," 1889, p. 90.) He talks of functionless organs losing in size with the suspension of natural selection "until the last remnant finally *disappears*" (*ibid.*, p. 292), while "the disposition of the tail to become rudimentary, in cats and dogs, may be explained in the simplest way, by the process which I have formerly called panmixia," *i.e.*, suspension of natural selection (*ibid.*, p. 430). This explanation "in the simplest way" fails entirely to say whether (ii.) or (iii.) is to be accepted after assuming the truth of (i.). What is quite clear is that in the only case where either the coefficients of regression or the mortality can at present be even approximately stated neither (ii.) nor (iii.) hold. Fox-terriers and domestic ducks may be bred with a comparatively small mortality, but how great must be the coefficients of regression if their foci of regression are to be placed only as far back, say, as at general populations of jackals and wild ducks.* Apart from cases of atavism, which may be looked upon as improbable variations amply allowed for by theory, we do note, even in dogs, a regression towards a distant ancestry (DARWIN: "Animals and Plants under Domestication," vol. 1, pp. 37, *et seq.*). In these cases, however, change of environment seems in some way more important than the suspension of natural selection. We have, so far, evidence in favour of Mr. GALTON's view of positions of stability for the focus of regression. It seems, indeed, to be a general opinion among breeders that a character can be fixed, a stock made to breed truer by repeated selection.

Thus DARWIN writes on "Fixedness of Character:" "It is a general belief amongst breeders, that the longer any character has been transmitted by a breed, the more fully it will continue to be transmitted. I do not wish to dispute the truth of

* Professor WEISMANN would place the focus of regression for domestic ducks much further back, presumably in a wingless stage. ("Essays on Heredity," p. 90.)

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the proposition that inheritance gains strength simply through long continuance, but I doubt whether it can be proved. In one sense the proposition is little better than a truism; if any character has remained constant during many generations, it will be likely to continue so, if the conditions of life remain the same. So again in improving the breed, if care be taken for a length of time to exclude all inferior individuals, the breed will obviously tend to become truer, as it will not have been crossed during many generations by an inferior animal." ("Animals and Plants under Domestication," vol. 2, p. 37.)

Down to the words "if the conditions of life remain the same," all is consistent with the extreme theory of panmixia, but making a breed *truer* by selection for many generations is only consistent with belief in a progression of the focus of regression, or in a change towards unity in the coefficient of regression with continued selection. The latter alternative would, I think, be quite inconsistent with our whole theory of heredity as applied to a practically stable population. As we cannot mathematically deal with a theory of progression of the focus of regression without some hypothesis of the nature of progression with continued selection, we will assume an extreme case, and suppose the focus to progress very rapidly, *i.e.*, that offspring regress to the mean of the population from which their parents have been immediately selected. This will at least offer some explanation of animals breeding truer with persistent selection, if at the same time it leads to results inconsistent with the extreme theory of panmixia.

(ii.) *Panmixia and Bi-parental Selection.*—Let h_1, s_1 be the paternal, h_2, s_2 the maternal distribution at each selection. Then with assortative mating after p generations, the standard-deviations of the male and female populations will be of the same form as after one generation and be given by the ϵ_1, η_1 of p. 310. Now this result is not like the stable focus of regression out of accord, I think, with experience. It is noteworthy how comparatively little difference there is in the variation constants of the different races of man, although in many cases pretty severe selection may have been supposed to have been in progress for many generations. For example, the mean cephalic index varies from 70 to 83, but the probable deviation from this mean only varies from about 2 to 2·7, so that even very primitive races (where the variation is small and we may suppose the selection has been severe, or the strain is very pure), do not "breed much truer" than highly civilised races with a far less mortality. The difference between the variation of the most and least variable races is probably not more than the β-terms in the values of ϵ_1 and η_1 (p. 310) may be able to account for.

Turning now to the alteration of the male and female means in p-generations of selection, let as before β_2, β_3, β'_2, β'_3, be the regression coefficients and u_n, v_n, the distances from m_2, m_3, of the means of the male and female populations out of which the nth bi-parentage (m_2, m_3, s_2, s_3, ρ) is selected.

Hence: $u_n - (\beta_2 u_n + \beta_3 v_n)$ and $v_n - (\beta'_2 u_n + \beta'_3 v_n)$ are the distances from m_2, m_3

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of the means of the male and female populations from which the $\overline{n+1}^{\text{th}}$ bi-parentage is selected.

Thus we have the finite difference equations:

$$u_{n+1} = u_n - (\beta_2 u_n + \beta_3 v_n)$$
$$v_{n+1} = v_n - (\beta'_2 u_n + \beta'_3 v^n)$$

Assume:

$$u_n = A\chi^{n-1},\ v_n = \beta\chi^{n-1}.$$

Hence:

$$A(\chi - 1 + \beta_2) = -B\beta_3,\quad B(\chi - 1 + \beta'_3) = -A\beta'_2,$$

or,

$$(\chi - 1)^2 + (\beta_2 + \beta'_3)(\chi - 1) + \beta_2\beta'_3 - \beta_3\beta'_2 = 0,$$

or,

$$\chi_1 = 1 - \gamma_1 \text{ and } \chi_2 = 1 - \gamma_2,$$

where γ_1 and γ_2 have the same values as on p. 311. Thus:

$$u_p = A_1(1-\gamma_1)^{p-1} + A_2(1-\gamma_2)^{p-1},$$

$$v_p = A_1\frac{\gamma_1 - \beta_2}{\beta_3}(1-\gamma_1)^{p-1} + A_2\frac{\gamma_2 - \beta_2}{\beta_3}(1-\gamma_1)^{p-1};$$

where

$$A_1 = \frac{(\beta_2 - \gamma_2)m_2 + \beta_3 m_3}{\gamma_1 - \gamma_2}$$

$$A_2 = -\frac{(\beta_2 - \gamma_1)m_2 + \beta_3 m_3}{\gamma_1 - \gamma_2}.$$

Now this solution* is the same as that on p. 311, except (i.) that u_p and v_p, unlike μ_p and μ'_p, are measured from the selected means, *i.e.*, the mean heights of the male and female populations are respectively $m_2 - u_p$ and $m_3 - v_p$ after p-generations; (ii.) that in the values u_p and v_p $(1-\gamma_1)^{p-1}$ and $(1-\gamma_2)^{p-1}$ replace γ_1^{p-1} and γ_2^{p-1}. We conclude, accordingly, since γ_1, γ_2, and, therefore, $1-\gamma_1$, $1-\gamma_2$ are proper fractions, that u_p and v_p grow smaller and smaller, or, if selection be long enough continued, the means of the male and female populations will ultimately pass to the selection means.

Of course, if selection be suspended at the nth generation, regression will take place as on p. 310, but only to the nearest focus of regression, *i.e.*, $m_2 - u_n$, $m_3 - v_n$. Thus the effect of n selections has been to raise the general means permanently by these amounts.

* The uniparental or parthenogenitic results for progression of the focus follow at once by simply putting $\beta_3 = \beta'_2 = \beta'_3 = 0$ in the above formulæ.

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(iii.) *Panmixia for Human Stature.*—It is instructive to note the value of these expressions for the case of stature in man. We have at once from the numbers on p. 311, supposing p-selections of male and female populations averaging 4″ and 3″ above the present mean, the following results:

(a.) *Without Assortative Mating.*

$$u_p = 3{\cdot}9549\,({\cdot}2933)^{p-1} + {\cdot}0451\,({\cdot}9581)^{p-1},$$
$$v_p = 3{\cdot}0538\,({\cdot}2933)^{p-1} - {\cdot}0538\,({\cdot}9581)^{p-1}.$$

Thus, in five generations $(p = 5)$ $u_5 = {\cdot}0673$ and $v_5 = -{\cdot}0227$, or the male and female means have been raised 3″·9327 and 3″·0227 respectively. Thus, we see that the males have been raised by selection very near to the selection average, while the females have actually been raised beyond it.* Thus, continued selection would now keep down, and not raise, the female mean, panmixia corresponding to a rise in the mean.

(b.) *With Assortative Mating.*

$$u_p = 3{\cdot}9893\,({\cdot}3560)^{p-1} + {\cdot}0107\,({\cdot}9656)^{p-1}$$
$$v_p' = 3{\cdot}0136\,({\cdot}3560)^{p-1} - {\cdot}0136\,({\cdot}9656)^{p-1}.$$

Thus, in five generations, $u_5 = {\cdot}0744$ and $v_5 = {\cdot}0366$, or the male and female means have been raised 3″·9256 and 2″·9634 respectively. The means are accordingly raised less rapidly with this form of sexual relation, the female mean, indeed, having in the five generations not yet overshot the selection mean.

(iv.) *Concluding Remarks on Regression and Fixedness of Character.*—Accordingly on this hypothesis, with the correlation coefficients of inheritance anything like their value in man, five generations of selections of the type required in *both* parents would suffice to establish a breed. This seems more or less consonant with breeders' opinions, which, in part at any rate, may be presumed to represent their experience. If, however, anything like this hypothesis be true, then the suspension of natural selection would not be followed by a rapid regression, or even a slow persistent regression, that would require a reversal of natural selection, *i.e.*, a selection of those previously destroyed and a destruction of those previously selected. On this hypothesis, indeed, it would be probably best to keep the term panmixia for that suspension of assortative mating which we have seen assists, rather than retards, the processes of natural selection.

Several fairly sound reasons could be given why the focus of regression should be taken as the mean of the population from which the parents have been selected, but the sole safe argument appears to be *experiment.*

* This results, of course, from breeding from an average father very much taller relatively than the average mother selected.

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The two hypotheses with which we have dealt give practically the two extremes; observation and experiment are perfectly able to determine between them, or to settle whether an intermediate theory is necessary which will give a progression, but a slower progression, to the focus of regression. There are many ways in which analysis can put on the brake, if it be really needful.

At present, all this memoir proposes is to show that such subjects as inheritance, regression, assortative mating and panmixia, are capable of perfectly direct quantitative treatment, and that such treatment, and not somewhat vague discussion of individual instances or of metaphysical possibilities, is what alone can settle the chief problems of evolution. What is wanted is a wide extension of the experimental and statistical work of Mr. FRANCIS GALTON and Professor WELDON. Such numbers as appear in this memoir must be looked upon as illustrative and tentative only. I hope later to publish, for a very limited field, namely, skull measurements in man, a more complete numerical study with mathematical discussion of variation and correlation.

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