relative amount, the plane of union of the pronuclei, and so forth. For example, if the egg is oblong, the plane of cleavage will be at right angles to the long axis. If there is more deutoplasm, yolk granules, in one side of the egg than in the other, the egg will divide unequally, the larger segment including that part of the egg which is richer in deutoplasm. In the next stage internal structure of the blastomeres again may have a controlling effect. Blastomeres may again divide unequally for the same reason as before, and those which are the less cumbered with deutoplasm will divide the more rapidly. Now another factor comes in, for as soon as the organism consists of more than one cell, the interaction of one cell upon another affects the mode of cleavage and the differentiation of the cell. At the same time the cells are being affected by stimuli from without the organism, such as light, heat, gravity, and chemical composition of surrounding media. Although the cytoplasm may divide into parts differing in composition and specific gravity, the division of the idioplasm in the nucleus is always *quantitative* only. The two daughter nuclei are exactly alike. Nevertheless, the nucleus controls the differentiation of the cell, and this is affected by the different idioblasts which become active in different nuclei. Which idioblasts shall become active in any cell is determined by the different positions of the nuclei relative to the whole mass and the consequent difference in the stimuli that they receive. Hertwig quotes with approval the remark made by Driesch that "the differentiation of the cell is a function of its position."

There is no preformation of the embryo in the egg. The mosaic theory of the cleavage is a myth, and it is still more mythical to carry this mosaic idea back to the idioplasm of the egg. Preformation exists only in so far that the structure of the egg as a whole determines the character of the first cleavage. The structure of the whole embryo in the two-cell stage determines what the next step in development shall be. *The new characters that appear in ontogeny are really new formations* and not merely the becoming visible of pre-existing latent possibilities. The form of the embryo is the resultant of two factors, viz., the character of the idioplasm, and the nature of the stimuli to which it is subjected. Of these stimuli, those which arise within the organism are the most important in ontogeny, for they are the ones that are constantly changing as development proceeds. The character of the idioplasm determines primarily what the reaction shall be to any given stimulus, and this reaction in turn determines what the stimulus shall be in the next stage. Development is purely a process of epigenesis, and each stage determines only what the next stage shall be.

Wilson takes a position that is somewhat less advanced than that held by Hertwig. He points out that while it is generally true that the relative position of a blastomere determines what shall develop from it, this relation cannot be a purely geometrical or mechanical one, for in different species of eggs blastomeres may exactly correspond in origin and relative position, yet have entirely different morphological value. This is strikingly shown by a comparison of the polyclade egg with that of the annelid or gasteropod. Cells with exactly similar geometrical relations give rise to entirely different organs in the two groups.

Wilson agrees with Hertwig that the differentiation of the cytoplasm in one stage has a determining influence upon the next stage, and he explains the difference between eggs of the gasteropod and the medusa in the ability of single blastomeres to produce a whole embryo by supposing this differentiation to have begun in the former early in the history of the ovarian egg. He does not agree with Hertwig in supposing the somatic nuclei to retain their embryonic character. He points out that the facts of regeneration indicate that the nucleus has undergone a change, and he suggests that the change may be due to a process like that observed in the somatic cells of the segmenting egg of Ascaris. That is, the somatic nuclei do really differ from germ nuclei, but this is because they have given up part of their substance to the cytoplasm, and not, as Weismann would suppose, because they have undergone qualitative division.

Conclusions.—We have now completed a review of the principal fundamental conceptions to be found in theories of heredity. We have seen that the animistic theories belong to the age of mysticism, and receive no confirmation from scientific investigations. We have seen that there is reason to believe that heredity has its foundation in a physical basis, which is the protoplasm of the cell; and this may perhaps be differentiated into physiological units of some kind. Further there is much evidence from observation and experiment to show that the physical basis for heredity forms an idioplasm residing in the nucleus of the cell and more especially in those parts of the nucleus which are composed of chromatin. Whether the idioplasm of the germ cell is essentially different from that of somatic cells is doubtful. The view that such a differentiation does exist is strongly supported on morphological evidence in the single case of Ascaris. The physiological evidence is clearly against a qualitative division of the idioplasm. On the other hand, it indicates some sort of progressive differentiation in the idioplasm of somatic cells during development, a differentiation which begins earlier or later in different species. The causes of this differentiation appear to lie partly within the idioplasm itself and partly in the conditions surrounding it. Both experiment and common sense favor the view that the cell can contain no elements presenting other than purely cellular characteristics. Development is essentially a process of epigenesis. There are as many species of germ cells as there are species of organisms, and the germ cells of any single individual have probably the same degree of variability that other serially homologous organs of that individual have. The normal offspring is similar to its normal parent, because the germinal idioplasm of the two are of common origin, and both have developed under normal conditions. There is no evidence that the germ cells in an adult organism may be affected in any direct way by any changes that may take place in the cells of the body. Therefore the inheritance of acquired modifications of somatic cells is theoretically improbable.

Robert Payne Bigelow.

BIBLIOGRAPHICAL REFERENCES.

Boveri, T.: Die Entwickelung von Ascaris megalocephala mit besonderer Rücksicht auf die Kernverhältnisse, Jena, 1899.
Brooks, W. K.: The Law of Heredity, 1883.
Conklin, E. G.: Embryology of Crepidula. Jour. Morph., vol. xiii., 1897, pp. 1–226.
Crampton, H. E.: Ascidian Half-Embryo. Annals N. Y. Acad. Sci., vol. x., 1897, pp. 50–57.
Crampton and Wilson: Experimental Studies on Gasteropod Development. Archiv f. Entw. d. Org., vol. iii., 1896.
Darwin, C.: Animals and Plants under Domestication. The Descent of Man.
Davenport, C. B.: Statistical Methods, 1899.
Delage, Y.: La structure du protoplasma et les théories sur l'hérédetie, 1895. (An excellent summary, which is continued in the annual volumes of "L'Année Biologique," edited by Delage.)
Dunker, G.: Die Methode der Variations-Statistik. Arch. f. Entw.-Mech., viii., 1899, 112–183.
Ewart, J. C.: Experimental Contributions to the Theory of Heredity. A. Telegony. Proc. Royal Soc., vol. lxv., 1900, pp. 243–251. The Penycuik Experiments, London, 1899.
Galton, F.: Natural Inheritance, 1889. Hereditary Genius, 1869. (See also papers in Proc. Royal Soc., London.)
Hertwig, O.: Die Zelle und die Gewebe, 2 parts, 1893–98 (Part 1 has been translated). The Biological Problem of To-day, 1896.
Morgan, C. L.: Animal Life and Intelligence, 1891. Habit and Instinct, 1896. Animal Behavior, 1900.
Morgan, T. H.: Regeneration, 1901.
Pearson, K.: The Grammar of Science, second edition, 1900. Contributions to the Mathematical Theory of Evolution, a series of articles in the Phil. Trans. and Proc. Royal Soc., London, beginning 1894.
Romanes, J. G.: Examination of Weismannism.
Spencer, H.: Principles of Biology, new edition, 1898, vol. i., pp. 269–373.
Thompson, J. A.: History and Theory of Heredity. Proc. Roy. Soc., Edinburgh, 1889, vol. xvi., pp. 91–116. The Science of Life, an Outline of the History of Biology and its Recent Advances, 1899.
Warren, E.: Observation on Inheritance in Parthenogenesis. Proc. Roy. Soc., London, 1900, vol. lxv., pp. 154–158.
Weismann, A.: Essays upon Heredity and Kindred Biological Problems, 2 vols., 1889–92. The Germ-Plasm, a Theory of Heredity, 1893.
Wilson, E. B.: The Cell in Development and Inheritance, second edition, 1900.

HEREDITY IN RELATION TO THE DEVELOPMENT OF MORBID STATES.—It is almost of the nature of a truism to state that morbid conditions are and can only be either inherited or acquired; yet we are apt to confuse these two modes of production; hence in dealing with heredity as a factor in the development of states of disease it is essential at the outset to lay down clearly what is and what is not inherited. A little consideration shows us that only that is inherited which is the property of the individual at the beginning of the existence of that individual, for once existence has begun any disturbances set up by influences from without—by injury, infection, or intoxication—are obviously acquired. Now individual existence certainly does not begin at the moment of birth; it begins at the moment that the nuclear material of the spermatozoon fuses with the nuclear material of the ovum to form the fertilized cell. Hence that only is inherited which has been conveyed to the individual by the spermatozoon or the ovum, or which directly results from the fusion and interaction of the substances of these two parental cells.

It follows, therefore, that conditions acquired by the individual during intra-uterine existence must be carefully separated from inherited states.* Thus we have to divide morbid conditions into:

A. *Inherited.*
(1) From the father.
(2) From the mother.
(3) Resulting from the interaction of the paternal and maternal germ plasms.

B. *Acquired.*
(1) Of antenatal acquirement.
(2) Acquired during parturition.
(3) Of postnatal acquirement.

In our determination of what is truly inherited we have to recognize that infection as such cannot be inherited; syphilis and tuberculosis, for example, cannot be spoken of as inherited conditions. Not that these infections of the parent may not tell upon one or other of the conjugating germ cells and so lead to disturbed states of ovum or spermatozoon which may materially influence the resulting individual; such indirect influences may, as I shall point out later, lead to the manifestation of *parasyphilitic* and *paratuberculous* lesions; but the actual infection cannot be inherited. There is no such thing as inherited syphilis in the proper sense.

For such infection presupposes the passage of microbes from one or other parent to the offspring through the instrumentality of the germ cells. Apart from the fact that such microbes cannot strictly be regarded as properties of the parental organism, and apart from the fact that the spermatozoon, the germ cell through which this transmission is supposed often to occur, is so small and of such a structure that it cannot be conceived as a carrier of microbes save by adhesion to its exterior, were a microbe or microbes to gain entrance by any means into the ovum, that would be at the most a fortuitous inclusion, and we are not justified in imagining that it could be present without so seriously injuring the cell as to render it barren or monstrous. As a matter of fact the laborious observations of Gärtner upon the number of tubercle bacilli present in the semen of tuberculous guinea-pigs have demonstrated that the probability of a bacillus-bearing spermatozoon fertilizing an ovum is so extraordinarily minute that it may be neglected, while the statistics of Chiari upon the specific lesions of infants suffering from antenatal syphilis show that the liver is the organ most extensively involved in nearly all cases—a sure indication that the channel of infection has been from the placenta; for the blood coming from the placenta first passes through this organ, which thus bears the brunt of the infection.

The not infrequent cases in which a syphilized father begets syphilized children without the mother showing signs of general syphilis, must therefore be due to intra-uterine infection, either by passage of the specific virus from the uterine cavity through the cells of the amniotic sac (this has recently been shown possible in connection with other pathogenetic microbes), or by local infection of the fœtal placenta from the uterine cavity, or by *local* infection of the uterine mucosa and maternal placenta without extension of the disease in the mother, although with conferment of relative immunity upon the mother.

Having now cleared the ground it is possible to discuss the effects of heredity pure and simple. The inheritance of morbid conditions may be: 1. Specific or ex specie, *i.e.*, peculiar to all the members of a species. 2. Racial. 3. Familial. 4. Individual.

1. EX SPECIE.—This shows itself mainly in predispositions to certain infections including the disturbances set up by the grosser parasites. The infectious diseases of cold-blooded animals, for example, are quite different from those of warm-blooded, and, while some diseases occur inc ommon, the diseases to which man is liable are, as a series, well differentiated from those affecting cattle. Certain infections like syphilis, gonorrhœa, and typhoid would appear to affect man alone under natural conditions.

An interesting line of thought has been touched upon by Bland Sutton in his "Introduction to General Pathology," where he points out that certain structural features so characteristic as to be differential in certain species and races, are essentially inherited morbid conditions or malformations. He notes that the race of tailless trout of Islay (and we may add the race of Manx cats), the albinic or non-pigmented species of snails; the Chætodon with its remarkable osteomatoid enlargements of the bones, the race of horned men of Akim in Africa with their huge symmetrical exostoses of the malar bones, are all examples of such morbid inheritance. He is inclined to believe that horn and horny outgrowths in general have originated as pathological states, and what is more he would regard the descent of the testicle into the scrotum in man, and other animals which assume the more or less erect position, as an inherited anomaly. Another good example of inherited pathological structures is the pair of "castors" situated on the inner side of either foreleg of the horse. These familiar objects are of cuticular origin, are horny and unconnected with bone; they are constant and apparently useless. The only satisfactory explanation he can suggest for the inheritance of these anomalies is that some prepotent ancestor of the horse conveyed this to its descendants along with other more valuable properties; in other words, that there is a certain correlation in the inheritance of variations.

2. RACIAL.—Among the members of one species those of a different race show peculiar susceptibilities. Thus among the lower animals, the "Buffel" or native cattle of Austria-Hungary have been found largely insusceptible to tuberculosis, in this differing widely from ordinary domestic cattle; the race of Algerian sheep is refractory to anthrax when ordinary domestic European breeds are peculiarly susceptible. Numerous similar examples can be cited among the races of mankind; negroes are peculiarly liable to succumb to tuberculosis, they suffer from sleeping-sickness, ainhum, etc., and on the other hand appear not to be so susceptible to malaria as are white races. Malaysians show a marked tendency to contract beri-beri, they and the Hindoos (though to a somewhat less extent) and Eastern peoples in general are more liable to contract and to die from the plague than are those of European descent, although the latter show themselves more susceptible to yellow fever. In Canada, as in France, the greater severity of scarlet fever when it attacks those of Anglo-Saxon descent as compared with those of French origin is distinctly noticeable. I have already in the previous paragraph cited some examples of the racial inheritance of anomalies.

* Chantemesse and Podwyssotsky suggest for the former the term "Uterine Inheritance." I strongly object to this on the ground that in discussing inheritance, we who deal with one branch of biology must conform to the usage of those working in other branches. Intra-uterine existence is characteristic of only one division of animals and that a somewhat limited one. To speak of intra-uterine acquirement as inheritance is a sure means of continuing the present confusion which exists among medical men in connection with this subject.

3. FAMILIAL.—Very numerous examples may be cited of familial inheritance. These examples range themselves in various groups:

(I.) *Inheritance of Anomalies.* Numerous well-recorded examples of cases of polydactyly, syndactyly, abnormal shortness of the phalanges, hypospadias, phimosis, are noted as occurring through several generations.

(II.) Other conditions which may be regarded as *probably anomalies of defect*, and which are markedly inherited, are hæmophilia, albinism, Daltonism, myopia, strabismus, ichthyosis.

(III.) *Diathetic.* The last example together with the tendency to the formation of cataract may possibly come under the heading of diathesis, or liabilities to abnormal functional disturbances occurring at certain periods of life. Among these also are to be included obesity, gout, chronic rheumatism, and diabetes.

(IV.) *Infectious.* It is a matter of popular knowledge that certain families are much more susceptible than is the race in general to acquired conditions like tuberculosis or one or other zymotic disease.

(V.) *Nervous.* The familial nervous conditions are especially worthy of note, because in a remarkable proportion of cases of mental and other nervous disturbances we find a history of antecedent nervous conditions, either homœomorphic, *i.e.*, of the same order, or heteromorphic, of different type. Among the former we recognize inherited paralytic and ataxic conditions, pseudo-hypertrophic and amyotrophic paralyses, Friedreich's disease, etc.; among the latter and more frequently we find mental disorders traceable to ancestral disturbance. There is one well-known county family in England in which a state of weak-headedness varying in its manifestations is traced back to the seventeenth century and is popularly attributed to the prominent part played by a celebrated ancestor in connection with the beheading of Charles I.

"The tendency of late years has been to lay increasing emphasis upon the hereditary factor in the production of insanity, but it is doubtful whether even yet the true bearing and significance of this factor has been recognized."

Carlos F. MacDonald lays down that the so-called exciting causes of insanity play a very unimportant part in the etiology of this disease, except in so far as they may operate to bring about a lowered and disturbed state of the brain and those set in motion hitherto latent inherited conditions or tendencies. In support of this he points out that substantially every individual at some time during his life is exposed, in many cases repeatedly, to many of the so-called exciting causes of insanity, both mental and physical; and yet despite this fact we find that sanity is the rule, insanity the exception.

4. INDIVIDUAL INHERITANCE.—Thus far we have had no difficulty in recognizing and to a large extent comprehending the influence of inheritance in passing down from individual to individual of one species, race, or family, certain peculiarities of constitution. We can understand, that is, that just as there are passed down those grosser properties whereby each member of a species resembles all the rest and differs from members of other species, so there are passed down these morbid constitutional states. It is when we come to study the subject of individual inheritance, the inheritance of conditions that is which have first manifested themselves in one or other parent, that from a medical as from a general biological standpoint we find ourselves in difficulties. So that in order to understand what is and what is not capable of inheritance we find it necessary to probe deeply in order to discover if possible some principle or principles which shall make it comprehensible why certain individual characteristics can thus be passed on in later generations, why others cannot. As Virchow has pointed out, every variation from the normal is strictly a pathological state, however slight it may be, and this whether it acts favorably or unfavorably upon the individual. Hence in discussing theories of inheritance which of necessity bear upon this subject of the production and descent of variations we are still strictly within pathological limits.

But in the first place, before considering these theories, it is well to analyze individual variations, for these it will be seen may be of two orders, some being due to the interaction of the conjugating germ plasms and so truly inherited, others being of the nature of acquirements. Among the latter are conditions of excessive development of one or other function or organ, brought about by continued use of the same; the muscular development of the athlete, the specialized development of the musculature of the fingers and of the nerve centres controlling the same in a pianist, and so on. Of the same order are the general results upon the organism of disease affecting the individual, as of infection due to tuberculosis and syphilis and the zymotic diseases, of injuries, *e.g.*, to the brain, and of intoxications, *e.g.*, plumbism and alcoholism.

In addition to these we have to recognize the occasional occurrence of what is known as "spontaneous variations," resulting in the production of sports. The most clearly marked examples of these are to be found among plants; in a bed of tulips, all apparently growing under the same conditions, an occasional flower may be met with possessing four, five, seven, or eight, instead of the usual six petals. Now while one or more of these variations may be ascribed to atavism—to a breaking back to an ancestral state—all cannot be; that is, the tulip must be derived in the distant past from the precursor of the Liliaceæ having either more, or less than six petals; it cannot be derived from both. And as these conditions of excess and defect are of the same order, it is probable that atavism plays little or no part in the development of sports of this nature. Discussing such cases the only conclusion we can reach is that some influence affecting the root bud at its origin, or, it may be, the abnormal interaction of the pollen and ovum, has brought about an abnormal development. Similarly in man and animals in general, the appearance of a sport in one member of a family is rarely if ever to be ascribed to atavism, but to similar influences acting either at the time of fusion of the ovum or spermatozoon, or telling upon the embryo during the very earliest stages of development.

Further than this, admittedly, we cannot advance in the explanation of so-called spontaneous variations. I refer to this matter because it is necessary to point out that both qualitative and quantitative variations like sexdigitism, Albinism, Daltonism, and hæmophilia, may and do constantly present themselves in the individual, and it is not necessary to assume that some ancestor exhibited a like condition; they may arise *de novo*. What is more, it would seem that there is a peculiar liability for these spontaneous variations to show themselves in succeeding generations, and to become familial. The strain of creamy-white horses preserved for more than a century at Hanover originated from a single sport of this order by careful in-and-in-breeding; as again did the Mauchamp strain of merino sheep with their peculiarly silky wool.

THE INHERITANCE OF CONDITIONS ACQUIRED BY THE INDIVIDUAL.

To us as medical men it is of special interest to determine how and to what extent acquired characteristics tend to be propagated; to what extent does disease in the individual tell upon the offspring; is it possible that besides resulting, it may be, in the production of offspring of lowered vitality, the different acquired infections or constitutional states are liable to influence that offspring in a specific manner in one or other direction and so afford examples of an inheritance of acquired characters?

According to Weismann in multicellular individuals characters acquired by the individual cannot be inherited, or at least it has still to be proved that they can. This I might add is a position that is generally taken by biologists of the present day (*vide* article on *Evolution*).

Weismann's Theory.—To understand Weismann's theory it is necessary in the first place to realize the importance of the nucleus in the cellular economy. This is the controlling agent in the cell: remove it and the cell is incapable of regeneration and proliferation (Nussbaum, Verworn). What is more it is the essential agent in fecundation. As is shown by Van Beneden (1875) in animal forms and by Strassburger (1884) in plants, fecundation is essentially the fusion of portions of the nuclear material (chromatin) of the male and female germ cells. At a later date Boveri showed that equal portions of the male and female chromatin unite to give origin to the new individual. Here then in this remarkable nuclear material is the physical basis of heredity. There is no substance in the fertilized cell which is similarly and equally derived from both parental germ cells.

When now the fertilized ovum proceeds to segment and to develop into the embryo and ultimate mature form, the process is seen to be primarily a division and distribution of the chromatin, so that the nucleus dividing before any signs of fission of the cell body show themselves, there are distributed to each daughter cell apparently equal quantities of the chromatin of the parent cell. Hence, as this process of division continues and the whole cell mass of the adult individual—the whole organism—is developed, each cell in that organism contains a portion or derivative of the original chromatin of the fertilized ovum. But a differentiation is to be made out. The majority of these cells form the tissues of the body and as such are found incapable of undergoing fecundation and giving rise to new individuals. These are Weismann's *somatic cells.* Only a certain number present in the reproductive glands maintain this capacity; they are the *germ cells.* And it is these germ cells which convey to the offspring the parental germ plasm; it is through them and through their nuclear chromatin, in which, as above said, the germ plasm would seem necessarily present, that descent occurs, and as in their turn they are the direct descendants of the germ cells which gave rise to the parent individuals and so contain germ plasm directly derived from ancestor after ancestor, so their essential germ plasm is potentially eternal. It has been passed on from generation to generation; the somatic cells of one generation after another have inevitably undergone death and dissolution; they have, after the simile of Lucretius, served but as the runners passing on the torch of life from one to the other.

Thus far we must follow Weismann, and further, with him we must see that the characters of the individual do not directly depend upon the characters of the body, or soma, of the parents. For these characters are transmitted by the germ plasm of the largely undifferentiated ovum or spermatozoon—by germ cells, that is, which are *not* derived from the body cells, and which never have been in other than a latent condition within the parental body. That body has grown and become differentiated and has been acted upon by its environment, but its function throughout has been to form a protecting envelope for the germ cells.

Such being the relationship, it is inconceivable that any character acquired by the parent in the nature of a gross lesion (the loss of an arm, for example), or again in the nature of a special development of one or other faculty (as, for example, the special dexterity of the finger muscle in a pianist) can be transmitted to the offspring. And as a matter of fact we know that the children of those who have lost a limb are born with a full complement of limbs; that ritual circumcision practised for long centuries by Jews and Mohammedans has not resulted in inherited phimosis, and that the docking of tails of horses has had no effect upon the length of tail of their offspring.

These considerations, while they throw light upon the exact nature of descent and while they explain largely the likeness of successive generations in one species or family, do not explain the other important condition of *variation.* For if, as Weismann repeatedly affirms, the environment of the germ cells while in the parental organism has a minimal effect upon them, we do not so far gain any comprehension of how it is that, although derived from intermingling of the same germ plasms, the various offspring of one pair of mankind, fish, or any other animal, while resembling each other in their main features, differ obviously the one from the other, so that no two animals of the same species or even of the same family are absolutely alike. Still less do these considerations explain the occurrence, in certain of the offspring of one pair, of features absent from either parent but present in one or other ancestor (atavism).

To explain this individual variation and kindred matters Weismann has developed a most elaborate theory, so elaborate, and undergoing so many modifications as new facts have presented themselves, that it is impossible to present it in its entirety in the limits of this article. Briefly, he has conceived the germ plasm or nuclear chromatin to be composed of an enormous number of individual particles or *biophores* derived from the ancestral germ plasms on either side. Prior to fertilization a remarkable process of reduction is observable in the nuclei of both the male and female germ cells whereby, according to Weismann, at the time of fertilization each conjugating cell contains only half the number of biophores originally present. By this reduction it comes to pass that through conjugation the fertilized ovum contains the normal number of biophores, half of them derived from the male and half from the female germ cell. Thus in each successive generation the germ cells will contain biophores derived from the male and female germ cells of the previous generation. And, carrying out this conception, it will be seen at the end of say, one hundred generations, the number of representative biophores derived from the whole chain of ancestors (even if as Weismann suggests these are so small as to be quite invisible) would be so enormous, but for this process of reduction with their number being halved prior to each fertilization, that they could not be conceived as capable of being crowded into one nucleus.

Now in this process of reduction Weismann lays down that there is no regular order of discharge of the biophores or ids; they are intimately commingled; representatives or determinants derived from certain near or remote ancestral germ plasms, must drop out; and so it happens that each mature germ cell contains a different series of these. Thus each individual is the outcome of the action of a different series of combinations of these biophores, and as such differs from every other individual.

This theory also demands that if these biophores are the essential and controlling constituents of the nuclear material, then, in division or segmentation of the fertilized ovum, nuclear chromatin and with it germ plasm is transmitted to each cell forming the organism. And to explain the difference in structure and characters of the cells forming the different tissues Weismann is forced to suppose that there is a *qualitative* difference in the transmission to the different cells and that so the different organs and tissues depend for their characters upon different combinations of the biophores, which in the process of successive segmentation of the blastomeres gain entrance into and determine the composition of the nuclei of the different forms of cells. The inheritance of special, racial, and familial features peculiar to and showing themselves in structural and other features of the body is thus, according to him, essentially due to these biophores, their particular distribution and their controlling action.

These, for our present purposes, are the main outlines of Weismann's celebrated theory. Others, among whom may be mentioned Darwin, Roux, de Vries, and we ought specially to mention Francis Galton, had previously enunciated theories or considerations along the lines here indicated, but to Weismann certainly must be given the incredit of elaborating a theory which attempts to cover the whole subject. As this theory has influenced the whole study of the subject of heredity, it has been necessary to explain to this extent its main tenets.