The Chromosome Theory of Mendelian Inheritance: Explanation and Realism in Theory Construction[1]

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Cytogenetics was born of the confluence of two so-far independent fields: cytological studies and breeding studies, which merged through the identification of genes with chromosomes. In this paper I argue that genes were introduced as functional entities. Functional explanations are presented here as a subclass of inferences to the best explanation and I argue that abductive arguments do not offer conclusive proof for the existence of the entities postulated through them. However, functional explanations usually follow a scheme laid out by Fodor (1968): there is a first phase where hypothetical entities are postulated and individuated through their effects; there is a second phase where the physical structures responsible for these effects are sought. I analyze the development of both phases in the construction of the chromosome theory of Mendelian inheritance. I will argue that first-phase theories are important to set conditions of identification for the functions played by a certain structure in a given containing system. The value of the second-phase theories lies in their capacity to constrain the development of first-phase theories, thus regulating the excessive plasticity of the first-phase theories and making them more susceptible to testing by empirical evidence.

1. The Theory of the Gene

In 1886, Gregor Mendel presented the results of his experiments with the common pea, *Pisum sativum*. Mendel crossed varieties of peas that presented sharply distinct characters, *e.g.* yellow seed with green seed. The first generation hybrid plants all had yellow seeds. The second generation had yellow and green seeds in a proportion of 3:1. In crossing varieties with two different characteristics, *e.g.* yellow and round seed with green and wrinkled seed, the first generation only gave yellow and round seeds; the second generation gave all possible combinations: yellow round, green round, yellow wrinkled and green wrinkled in a proportion of 9:3:3:1. The distribution and contribution of characteristics followed the same patterns in all the crosses he carried out. Thus, what Mendel discovered was the existence of certain patterns of inheritance.

For Mendel, the regularities observed in the distribution of traits suggested the existence of underlying entities that would account for such distribution if they segregated in the formation of gametes. He pointed out the necessity of studying the cellular

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theory of fertilization in search of a possible physical basis or mechanism of inheritance. For the visible characteristics, Mendel used the word *Merkmale*. For the hypothetical entities in the germ cells, he introduced the word *Elemente*. These *Elemente* could provide an explanation of what was going on at the level of observations:

> The law of combination of different characters which governs the development of the hybrids finds therefore its foundation and explanation in the principle enunciated, that the hybrids produce egg cells and pollen cells which in equal numbers represent all constant forms which result from the combinations of the characters brought together in fertilization. (Mendel 1865, p. 32)

The genetic elements were identified by their effects. From the analysis of the hereditary pattern of a trait we infer that one or more elements are responsible for it. The Mendelian *Elemente* were thus introduced as functional entities, *i.e.*, hypothetical entities of an unknown nature whose behavior would account for the observable effects. Since the germinal cells are the only physical structure passed down from parents to offspring, Mendel pointed out the convenience of looking into this 'black box' searching for a physical structure that fulfilled the necessary requirements to constitute the mechanism of inheritance.

Functional explanations are those that attribute certain capacities or dispositions to the objects we are theorizing about. The Mendelian *Elemente* —later called genes— were introduced as those entities capable of accounting for the distribution of phenotypic traits. Genes were not introduced to explain why a certain plant had a certain trait and not another. Genes were responsible for the phenotypic traits, but what brought them into the picture was their capacity for giving rise to the specific distribution of traits found in breeding and hybridizing experiments. Mendel did not argue that the flowers are red because of the presence of red-producing entities (see Fodor 1968, p. 36). Mendel argued that there had to be elements whose segregation in the germ-cells accounted for the existence of three plants with red flowers and one plant with white flowers in the offspring of a cross between a red-flowered plant and a white-flowered plant. The fact that there were red-producing entities, and white-producing entities, was a functional definition that had been put forward by other researchers, *e.g.* De Vries, Darwin, etc. What Mendel discovered was another capacity of these trait-producing entities: whatever they were, their behaviour had to account for certain phenotypic ratios. Genes had to have the capacity of distributing themselves in a certain way. Thus, the capacity of organisms for passing down their traits in such and such a way is explained by analyzing it into the capacity of the genes carried in the germ cells to segregate in a parallel way.

The Mendelian account, then, follows the same scheme as that offered by psychological explanations, according to Fodor (1968, p. 108). There is a first phase where the hypothetical entities are individuated through reference to their alleged causal consequences. In a second phase, the physical system that exhibits the functional properties enumerated by phase-one theories is sought. Second-phase theories look into the 'black box' to find out which elements are performing the function hypothesized in the first-phase theories. I find the case of genetics especially interesting to study the development of these two types of research. The interesting feature which I want to focus on is the fact that there were two completely different and so far unrelated sciences which were looking into the phase-one and the phase-two theories. Instead of sequentially, these phases were being carried out in a parallel fashion. But, even so, the relevant structures in the cell-nucleus could be identified because of the restrictions imposed by the functional analysis provided by genetics. According to Fodor, phase-one and phase-two theories condition one another. I think that this inter-

action can be seen very well in the development of the Mendelian theory of chromosome inheritance because this theory was developed as a result of the interaction between a phase-one theory stemming from the field of genetics—the theory of the gene—and a phase-two theory put forward as a result of the cytological studies carried out in the first decade of 1900—the chromosome theory of inheritance.

In the theory of the gene, the capacity of the germinal cells to transmit the hereditary information is explained by analyzing it into the capacity of certain germinal components -the genes-, so that the behavior of the genes accounts for the manifestation of the transmissory capacity. The genes, in turn, are themselves functional entities whose capacity to be carriers of hereditary traits that show certain pattern of distribution is explained by analyzing it into their capacity to segregate and assort independently. Now, a second phase theory would require finding an structure capable of carrying out this function. However, that structure can be selected as the one having that function because of the conditions of identification provided by the functional theory. I will come back to the relations between phase-one theories and phase-two theories later.

As 'functional entities', what was the ontological status of genes? To account for breeding results genes had to be postulated, but genes were used as 'calculating units'. Researchers did not worry about their reality in the cells. Genetics, in its early stages, only required a formal concept of the gene, that is, the concept of an entity responsible for the effects observed, but lacking any intrinsic characterization. Thus, when the hypothesis of a certain number of genes was insufficient to account for a given phenotypic ratio, more genes were introduced. This procedure elicited a great deal of criticism from important researchers, specially W.E. Castle and T.H. Morgan. The use of genes as calculating units raised doubts about the value of a scheme in which the phenomena were supposed to be explained by the very same entities that had been introduced to explain them. T.H. Morgan quickly saw the flaws of this line of reasoning as to the existence of genes:

> In the modern interpretation of Mendelism, facts are being transformed into factors at a rapid rate. If one factor will not explain the facts, then two are invoked; if two prove insufficient, three will sometimes work out. The superior jugglery sometimes necessary to account for the results are often so excellently explained because the explanation was invented to explain them and then, presto! explain the facts by the very factors that we invented to account for them. I realize how valuable it has been to us to be able to marshall our results under a few simple assumptions, yet I cannot but fear that we are rapidly developing a sort of Mendelian ritual by which to explain the extraordinary facts of alternative inheritance. So long as we do not lose sight of the purely arbitrary and formal nature of our formulae, little harm will be done; and it is only fair to state that those who are doing the actual work of progress along Mendelian lines are aware of the hypothetical nature of the factor assumption. (Morgan 1909, p. 365)

Soon, even more genes came into the picture. Genes had been introduced as hypothetical entities to explain certain phenotypic data. When new data where discovered, researchers postulated more genes. This had been the strategy used to give an account of alternative inheritance, and was later the strategy to explain the heredity of quantitative characters. If the hypothesis of one gene being responsible for the appearance of a characteristic was not able to explain the patterns of inheritance of quantitative characteristics, then we can imagine several genes involved in the production of these characters. The multiple-factor hypothesis put forward by Nilsson-Ehle (1909) and E.M. East (1910) explained the inheritance of quantitative traits by the cumulative character of the effects of several genes.

Again, this procedure was soon criticized. W.E. Castle (1912) spoke out against this way of introducing *ad hoc* entities. However, East (1912) pointed out that the inference was the same as that followed in the case of alternative inheritance. If we allow the use of functional entities to explain certain data, we also have to allow their use to explain new data. If genes were introduced due to the effects seen in certain crosses, the ratios found in different crosses would allow us to postulate further genes to account for the distribution of traits observed. If the observable effects are the only criteria taken as a guide for the existence of an entity, then the same standards have to be applied when 'explaining' new observational data. Genes were calculating units, and, as such, researchers could introduce as many as necessary to account for the breeding data. (See Vicedo forthcoming a for an analysis of the Castle-East debate on the introduction of several genetic units to explain the inheritance of quantitative characters).

According to Morgan (1909, p. 365), nothing seemed too risky as long as geneticists did not lose sight of the "purely arbitrary and formal nature of our formulae", and only worked with the formal gene. But, as he pointed out, as long as researchers used these formal formulae they could not intend to be explaining the phenomena of heredity. I think Morgan was right. As I see it, functional explanations are a subset of inferences to the best explanation. These explanations can claim a right to explain only in the weak sense of explanation. If to explain something is to offer an account of how an event could be brought about, *i.e.* a 'possible' account, with no claim to being the true one, then a functional explanation is an explanation. But, if 'explaining' means giving the right account, then, a functional explanation needs to provide evidence to prove itself as the correct account of the phenomena under consideration. Further and independent evidence is necessary because functional explanations are too permissible to support belief in the existence of the entities introduced through them. A functional explanation can not claim validity only on the basis of its explanatory capacity.

However, I think that explanation can be viewed as a methodological device in the construction of theories, leaving aside the question of its epistemological virtues. In theory-construction, the explanatory capacity of a hypothesis is not a virtue per se. The fact that a hypothesis 'explains' does not raise the probability of its being the correct account of the phenomena. For example: we have certain phenomena, 'a', and we introduce 'b' to explain their existence. Then, we conclude that 'b' explains 'a'. Indeed it does so, but only because we constructed 'b' in order to explain 'a'. It is a different question whether 'b' is *the* explanation for 'a', in the sense of describing the real mechanism that brings 'a' about. As Morgan pointed out, we can not reason backwards from the facts to the causes and then, attempt to explain the facts by the very causes we invented to account for the facts. Unless, of course, we have independent support for the existence of the causes.

I presented the theory of the gene as a functional explanation. Functional explanations involve an abductive argument. An abductive argument is one in which we establish a connection between a known phenomenon and an unknown phenomenon, where the second one is hypothesized as being the cause of the first one. The behavior of the hypothetical entities or processes would account for the observable effects. I think there are two types of abductive arguments (no inferences):

Type A: We know 'a' — We know 'b'; the abductive step consists in the connection between 'a' and 'b'.

Type B: We know 'a' — We do not know 'b'; the abductive step consists in 1) hypothesizing the existence of 'b' and 2) establishing a connection between 'a' and 'b'. I will call this second type 'existential hypotheses' for the obvious reason that here it is

not only the relationship between 'a' and 'b' what it is at stake, but the very existence of 'b'. In fact, the existence of 'b' is a prerequisite to the connection between 'a' and 'b'.

Herschel (1830, p. 84) presents an example of type A: an African guide sees a flock of condors soaring in circles at a distance. Based on these signs, he guesses the presence of a lion eating the carcass of a horse in that spot. It is important to note that this way of reasoning can be classified as abductive only if the conclusion is extracted for the first time. On subsequent occasions, to reason about the presence of the lion would be the result of an inductive argument, where successful past inferences linking a=circling condors to b=lions, warrant the new connection. It would be an extrapolation from a known sample to a new case that we take to belong to the same class as the previous sample.

In the second type of abduction the argument has to warrant two things: the existence of 'b', and the connection between 'a' and 'b'. For example, the theory of the gene had to prove two things: -the existence of the genes, and -the capacity of the genes to account for the existence of the phenotypic regularities found in breeding experiments. These two claims are often confused because the only way we have to infer the existence of the genes is through their connection with the phenotypic characteristics. But I am not saying that there are two steps in the construction of a functional explanation with an existential hypothesis. We introduce genes and their capacities in a single 'explanatory move'. It is only one step and, regarding the construction of theories, abductive arguments of type B are not different from abductive arguments of type A. But, in confirmation or justification, they are different because the conclusions of arguments of type B are riskier. The lion under the circling condors is a token of a type already known in our ontological world, but genes were a new type of entity altogether when first postulated by Mendel.

Abductive arguments and the functional explanations based upon them do not provide support for our hypotheses in themselves. Some of the abductive arguments we make are good, some are bad. However, it is not the fact that a hypothesis is explanatory that makes it good. What this means is that functional explanations are not the type of explanation that deserves a monolytic epistemological attitude. That is, I can not give a certain degree of confidence to a functional explanation because it is of that sort. One has to analyze abductive arguments on a case by case basis. In such an analysis I think the most important factor to take into consideration should be the robustness of the current knowledge in the area from where the inference is drawn. For example, in the case of the lion, one would consider what our biological theories tell us about the feeding habits of the birds we see, what our geographical theories tell us about the plausibility of lions living in that territory, etc. This poses a problem for functional explanations with an existential hypothesis. When a new entity is introduced, there are no theories about it. At the beginning of the 1900's there was a theory *of* the gene, namely the theory introducing genes to explain breeding ratios, but there were not theories *about* the genes.

The theory of the gene postulated the existence of these entities in the germ cells with certain properties. However, it was not only the capacities of the genes what was being posited; it was the very existence of genes, too. Thus, were genes to exist and have the capacities researchers were attributing to them, they would explain, i.e., —correctly explain—the observed phenomena. But because of the explanatory capacity of the theory, geneticists could not claim that there really were genes and that they had certain properties. In order to truly explain the breeding data one had to resort to the idea of the biological gene, not only to the formal, functional gene. But it would be a fallacy to think that the explanatory capacity of the formal gene was evidence for the reality of the biological gene. Were genes to exist, they would explain the phenotypic data. But, simply from the

fact that they explained the observable data, one could not conclude that they existed. Genes, therefore, could only be treated as instrumental devices.

Nothing was know about the nature of genes, not even about their specific location. Function was not sufficient proof to accept genes into the biological ontology. While geneticists were checking the validity of the Mendelian scheme at the observable level, the calculating unit was sufficient. But, when the empirical adequacy of the theory was proved, biologists recognized the necessity of searching for the biological reality responsible for the observed patterns. If 'explaining' means explaining correctly, or constituting *the* explanation, then geneticists needed to show that genes existed and that they had the capacities postulated. A phase-two theory needed to come into the picture. As it happened in this particular episode of the history of science, researchers in another field were building it.

I have argued that functional explanations, as a sub-class of inferences to the best explanation, only allow for the instrumental reliability of the theories constructed. Neither the reality of the entities posited nor the truth of the theories constructed can be defended from the explanatoriness of a hypothesis. The major virtue of a functional explanation is its flexibility or elasticity since, even if the phase-two theory presents a mechanism that turns out not to exist, and, therefore, the theory proves incorrect, the phase-one theory could still be right. This elasticity, though, is its own undoing because the functional explanation is too liberal to support belief in its posited entities and processes. Does this mean that functional explanations, or more generally, inferences to the best explanation, should not play any role in science?

I think not. I think that abduction has a role to play in science, but not a role in confirmation. Actually, Charles S. Peirce, the introductor of abduction or IBE as a form of reasoning different from induction and deduction, presented abduction (or retroduction, as he used to call it) as the first stage of reasoning. Peirce did not believe that abduction on its own could provide any confidence in the hypotheses derived through its application (Peirce 1958, pp. 368-71). I do not want to divide here the scientific enterprise between the context of discovery and the context of justification because I think there are many processes in science that cut across such a division. But let's think of science as a process and focus on theory construction. In theory-construction there are many permitted moves (as in a chess game). At a certain stage of the game we can take different methodological roads. However, it is another question whether these moves will have epistemological consequences (as it is a different question whether a move in a chess-game will lead to winning the game).

Abduction is an ampliative move, but not in updating our beliefs, only in theory construction. Thus, abduction does not give us conclusions to believe, only to entertain. Abduction is an instrument to be used in constructing a theory, not in the evaluation of a totally elaborated theory. Abduction is a method or rule to build up hypotheses, not to validate them. It gives us working hypotheses, and to take or reject a working hypothesis we evaluate whether it is worthy of further development, not whether it is true. N.R. Hanson saw the importance of this evaluation: "We must attend as much to how scientific hypotheses are caught, as to how they are cooked" (Hanson 1958, p. 62). And belief is not concerned with the catching of the trout, as Hanson would put it.

2. The Chromosome Theory of Inheritance

The germ cells had to be able to transmit the hereditary elements since they were the only structure passed down from parents to offspring. The question, then, was to single out the elements inside the cell that were the specific carriers of heredity. From

theoretical considerations about mitosis, and the complex and precise behavior of chromosomes, chromosomes were the main candidates for this function, according to many researchers. However, this belief was not universally accepted. There was not any proof for a direct relationship between heredity and chromosomes.

The role of the chromosomes in heredity became evident with the experimental studies of Theodor Boveri (1862-1915). In 1902 this German professor discovered the actual nature of reduction in cellular division. He described meiosis, and in particular, synapsis. Working with the eggs of sea-urchins, he realized that the irregular distribution of chromosomes caused by multipolar division produced an abnormal development. He then concluded that each chromosome had to carry certain individual qualities which are expressed in the organism's development. Thus, Boveri showed the individuality of chromosomes and the necessity of having a complete set for the normal development of an organism (Boveri 1902).

Chromosomes were simply certain bodies in the nucleus of the germ-cells. The first question was: what do they do? When their highly precise movements were discovered, the next question was: what is their function? The physiological studies of Boveri showed experimentally their connection with heredity. Their continuity and individuality were a sign of their importance in the cell. One thing they could be doing was carrying the hereditary information. Thus, cytology was explaining the capacity of organisms to inherit the characteristics of their parents by analyzing this capacity of the germ-cells into the capacity of chromosomes to account for the existence of certain phenotypic traits. But the only direct conclusion was that chromosomes were 'related' to heredity. There was no agreement on their role, on the way they were related to heredity. I think that the functional characterization of the cell-nucleus as the carrier of the hereditary traits was too general. It did not allow to pick out clearly the elements in this systems carrying out that function because it did not provide conditions for identification, it did not constrain the physical characterization of the possible entities doing this job. The conditions for identification were specified by the field of genetics.

3. The Chromosome Theory of Mendelian Inheritance

In 1903, W.S. Sutton published "The Chromosomes in Heredity", a paper without new data, but containing an interpretation of the known cytological facts in terms of the Mendelian laws. According to Sutton the group of chromosomes was comprised of two subgroups, one of which came from the father and the other from the mother. In synapsis there was a pairing of the homologous paternal and maternal chromosomes which afterwards underwent separation in an independent fashion. A very important point is that the chromosomes retained their morphological and functional individuality throughout the cell divisions. The functional individuality of chromosomes had been proved by T. Boveri some time before. Thus, the chromosomes differ qualitatively, but from cytological facts it was known that homologous chromosomes cover the same ground in development. Sutton argued: "If this be the case chromosome A from the father and its homologue, chromosome *a* from the mother in the presynaptic cells of the offspring may be regarded as the physical bases of the antagonistic unit-character A and *a* of father and mother respectively (1903, p. 32)."

A text-book outline of this period runs as follows: the physiological and morphological studies of chromosomes complemented each other. Boveri confirmed the functional individuality of chromosomes. Sutton discovered their morphological continuity. Then, an analysis of their behavior in cell division showed their random assortment. Their behavior paralleled exactly that of the Mendelian units which had to be transmitted in an independent fashion. The connection between both phenomena was quite straightforward.

However, I argued before that function can not be seen by looking at the behavior of a system, but rather requires a link from effects to structure specific enough to single out the structure that can account for the effects. A type of chain that constrains which kind of structure can be carrying out the specific task we are focusing on. Thus, if chromosomes segregated they could be the carriers of genes. Did chromosomes segregate? Well, the best explanation of their behavior was that they were the carriers of genes and their separation in meiosis was the segregation of the genes required by Mendelian inheritance. The situation in genetics was: if there were some elements in the cells that segregated in the formation of gametes, their behavior could account for the phenotypic ratios found by Mendel. The situation in cytology was: if chromosomes were segregating, that would explain their behavior during meiosis. Both were inferences to the best explanation. And so far, none of them had much empirical evidence in its favor.

Sutton and Boveri pointed out the striking parallelism between genes and chromosomes (Sutton 1903, Boveri 1903, 1904). Both the Mendelian factors and the behavior of chromosomes seemed to obey the same laws. Both phenomena had the same essential features (if chromosomes were segregating), a fact which seemed to indicate a correlation between them. In natural sciences, correlations are seen as needing explanation. When two orders of phenomena share the same features, this coincidence requires finding out whether they are 'only' correlated; that is, whether they are phenomena co-occurring by chance or whether there is a common cause explanation or a causal relationship between the two. The behavior of chromosomes in meiosis would be a sufficient mechanism to account for the segregation and independent assortment of the genes if genes were material entities carried by the chromosomes or parts of chromosomes, i.e., if an identity (or whole-part) relation could be established between the chromosomes and the observed ratios in inheritance (For a more detailed account of the development of the chromosome theory of heredity and the chromosome theory of Mendelian inheritance, see Baxter & Farley 1979, Carlson 1966, Coleman 1965, Darden 1980, Gilbert 1978, and Whitehouse 1973).

At the beginning, the reducibility of genes to chromosomes seemed quite speculative. However, the parallelism was so close that researchers argued that nature would not build such a complicated mechanism for nothing. A mechanism perfectly able to account for the behavior of genes was existent in nature. Could it be possible that it was completely unrelated? Parsimony would seem to deny such a wasted effort in nature. Simplicity required an ontological reduction. However, simplicity was not taken as conclusive proof. As it happened, the relation between genes and chromosomes was not immediately accepted when pointed out by Boveri and Sutton. Genetics required a physical structure capable of accounting for traits-transmission. Thus, until chromosomes could be shown capable of explaining specific distribution and combination of traits, they could not be taken as the necessary mechanism for heredity.

Again, Morgan pointed out the key question. The chromosomes were a sufficient mechanism to account for the segregation of genes. Now, were they the real system bringing about the distribution of traits? The existence of correlation does not imply a relation between two phenomena. Thus, correlations are not necessarily explanatory because they might be due to chance. This is how, with hindsight, Morgan saw the problem in the first decade of the 1900's: "Even if it is admitted from such evidence that the chromosome mechanism suffices to explain Mendelian segregation and assortment, as Sutton pointed out, it still remained to be shown that we are not dealing with analogy or coincidence, but that the chromosomes are specifically related to genetic events." (Morgan 1923, p. 172)

The striking parallelism between both phenomena was not enough to establish an identity relationship between them. It was enough, though, to give more plausibility to a research program aimed at finding the precise nature of this relationship. It gave plausibility to the search for the biological gene underlying the notational gene. What genetics needed to show was that the correlation was there, and that this correlation was best accounted for in terms of an ontological reduction: that genes were pieces of chromosomes. Taking the correlation as a working hypothesis, researchers moved to find out whether the phenotypic ratios could be explained in terms of genes as pieces of chromosomes. When all the different distributions of traits observed experimentally could be accounted for—and predicted—through cytological explanations, and cytological facts could be accounted for—and predicted—by genetic explanations, the hypothesized correlation was taken as real and as explained by the reduction of genes to chromosomes. (See Vicedo forthcoming b for Morgan's later views on genes).

Thus, genetic findings at the level of a phase-one theory had enabled the elucidation of morphological structure in terms of function. Genetics provided a framework from which to understand the cytological structure. Researchers needed to discover what element in the cell-nucleus had the function of distributing the hypothetical genes in a way that could account for the phenotypic distribution of characters from parents to offspring. Genetics established the necessary conditions which the mechanism had to fulfill. The Mendelian laws had set certain constraints on the kind of mechanism which would be able to carry out the distribution of genes (see Baxter and Farley 1979). Chromosomes turned out to furnish all the requirements imposed by the results obtained at the level of observations. Finding a certain structure capable of being understood in these terms provided support to initiate a research program to convert this correlation into the appropriate relationship. Therefore, the finding of this mechanism provided evidence for the existence of genes, even if at this point the structure, nature and function of genes was still unknown. On the other hand, the finding of a function for the behavior of chromosomes was the framework that permitted the discovery of how the chromosomes were contributing to the capacity of the gametes to transmit the hereditary factors. In this fitting together of structure and function, there was a process of double-checking where each science -genetics and cytology- provided evidence for the findings of the other, and also set constraints to the possible interpretations of the new phenomena found in each field.

I have talked about the theory of the gene, a functional explanation provided by a phase-one theory to account for certain observable phenomena; and about the theory of chromosome inheritance, a phase-two theory that was only fully understood by merging with the functional explanation provided by genetics, a move that gave rise to the chromosome theory of Mendelian inheritance. I have argued that the value of the functional account was that it provided the conditions necessary to identify the structure capable of being the mechanism of inheritance. What was the value of the second-phase theory for the theory of the gene? I think that second-phase theories play an important role in theory-construction: they regulate the growth of first-phase theories. Again, I think Morgan saw this advantage in the construction of the chromosome theory of Mendelian inheritance:

> The discovery of a mechanism, that suffices to explain both the first and the second law of Mendel, has had far-reaching consequences for genetic theory, especially in relation to the discovery of additional laws; because the recognition of a mechanism that can be seen and followed demands that any extension of Mendel's theories must conform to such a recognized mechanism; and also because the apparent exceptions to Mendel's laws, that came to light before long, might, in the absence of a known mechanism, have called forth purely fictitious

> modifications of Mendel's laws or even seemed to invalidate their generality. We now know that some of these "exceptions" are due to newly discovered and demonstrable properties of the chromosome mechanism, and others to recognizable irregularities in the machine. (Morgan 1935, p. 5)

4. Concluding Remarks

I have been analyzing explanation as a methodological device, not as a criterion for the epistemological evaluation of finished and established theories. I defended that abductive arguments have to be assessed in the context of theory construction. Abductions can not be judged by their formal structure, since they are not inferences. Abductive arguments have to be judged by their validity in a given context. In this sense it will be the robustness of our previously established theoretical knowledge what will warrant the use of a given abductive argument in the process of further developing that knowledge. This is why functional explanations with an existential hypothesis are weak hypotheses to account for the phenomena under study. Functional explanations are also seen as highly underdetermined since they can be readjusted to fit the phenomena under the only constraint that they explain them. However, functional explanations are constructed through a two-step procedure and the elasticity of the first step gets promptly constrained with the introduction of a mechanism that can regulate the future development of the theory. The mechanism discovered by the second-phase theory will thus be able to distinguish between permitted moves and *ad hoc* additions to the first-phase theory. It will thus differentiate a change within a theory -no matter how radical- and a change from a theory to a new hypothesis or theory, this is, a change between theories.

The important point about interfield connections (see Darden & Maull 1977) of the type existent between phase-one and phase-two theories in the development of a functional explanation is that not only will they allow predictions between different levels—which are stronger than within the same level—but they will also forbid certain developments of the theory by invalidating many moves to make the theory fit new data. The value of functional explanations emerges clearly then when seen from the perspective of theory construction. In the particular period of the history of genetics that I have analyzed, the interaction between genetics and cytology through the correlation of genes with chromosomes proved to be important not only in the process of theory-construction, but also in the process of confirmation. However, this is a topic for another paper.

Notes

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