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THEMATIC ISSUE ARTICLE: THE MEANING OF ''THEORY'' IN BIOLOGY

Theorizing and Representational Practices in Classical Genetics

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Abstract In this paper, I wish to challenge theory-biased approaches to scientific knowledge, by arguing for a study of theorizing, as a cognitive activity, rather than of theories, as abstract structures independent from the agents' understanding of them. Such a study implies taking into account scientists' reasoning processes, and their representational practices. Here, I analyze the representational practices of geneticists in the 1910s, as a means of shedding light on the content of classical genetics. Most philosophical accounts of classical genetics fail to distinguish between the purely genetic, or Mendelian level, and the cytological one. I distinguish between them by characterizing them in terms of their respective associated representational practices. I then present how the two levels were articulated within Morgan's theory of crossing-over, and I describe the representational technique of linkage mapping, which embodies the "merging" of the Mendelian and cytological levels. I propose an analysis of the mapping scheme, as a means of enlightening the conceptual articulation of Mendelian and cytological hypotheses within classical genetics. Finally, I present the respective views of three opponents to Morgan in the 1910s, who had a different understanding of the articulation of cytology and Mendelism, and entertained different views concerning the role and proper interpretation of maps. I propose to consider these diverging perspectives as instantiating what I call different "versions" of classical genetics.

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Introduction

My aims in this article are twofold. First, I wish to argue for a study of *theorizing* rather than of *theories*. Most philosophical studies of theories construe them as abstract structures independent from the agents' understanding of them. By contrast, my approach takes into account the scientists' actual reasoning processes. This implies focusing on the concrete representational devices they use. I will argue that this is a fruitful way of clarifying the conceptual content of a science at a given time as well as its development and relations to other scientific domains.

In this article, my defense of this approach to theorizing takes the form of a historical case study in classical genetics: I analyze the representational practices of geneticists in the 1910s. My second aim is to shed light on the conceptual content of classical genetics at that time. As classical genetics was already well developed in the early 1920s, my analysis should also clarify some aspects of this theory as we know it today.

The issue of the identity of classical genetics has most of the time been tackled by philosophers of science as a preliminary to answering another question, viz., whether classical genetics has been reduced by molecular biology (Schaffner 1969; Hull 1972, 1979; Wimsatt 1976; Darden and Maull 1977; Kitcher 1984; Burian 1985; Rosenberg 1985; Waters 1990; Sarkar 1998). The question is usually put as follows: are the laws and concepts of classical genetics definable in terms of, and deducible from, the laws and concepts of molecular biology? In order to answer it,

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philosophers generally begin by proposing a reconstruction of the content of the two theories at stake in terms of their fundamental laws and concepts. Such a reconstruction most often relies on a conception of theories as abstract structures, considered as independent from the way they are used by scientists. These approaches miss important aspects of the content of classical genetics, on which I aim to shed light.

Some philosophers and historians who advocate a study of scientific *practice* have already challenged theory-centered approaches to scientific knowledge, in particular to classical genetics (e.g., Kitcher 1984; Kohler 1994; Waters 2004).[1] My approach diverges from these proposals in important respects. First, unlike Kohler's social-constructionist study of the experimental practices of geneticists, I aim to clarify the *conceptual* content of *theoretical* knowledge in genetics. Kohler's account of the construction of *Drosophila* as a laboratory tool intends to show that the geneticists' efforts were aimed towards investigating a broad range of biological phenomena rather than providing an explanation of heredity. Kohler's account thus relies on a deliberate neglect of the theoretical concerns of the geneticists. However, I am interested in practice insofar as it is an essential part of theorizing. In this regard my approach is closer to Waters', who criticizes Kohler for considering the geneticists' "theoretical interests" as irrelevant (Waters 2004, p. 785).[2] Although I am convinced by Waters' arguments, my study of classical genetics differs from his insofar as I lay stress on *representational* rather than *experimental* practices.[3] I aim at characterizing classical genetics by identifying a certain form of reasoning as embodied in the use of a certain type of representations.

Characterizing classical genetics by identifying a certain "pattern of reasoning" is precisely what Kitcher aims to do in his famous "1953 and All That: A Tale of Two Sciences" (1984). He proposes an account of the "practice" of classical genetics and its successive "versions" in order to show that molecular biology does not provide us with a reductive explanation of this theory. Despite the similarities of our approaches, I disagree with Kitcher's account in many regards.[4] I will recurrently draw comparisons between his view and mine. Briefly, his patterns of reasoning are, in my view, still too abstract units of analysis, and this leads Kitcher to mischaracterize the content of classical genetics. In particular, like almost every philosophical account of classical genetics that has been proposed in the framework of the antireductionism debate, Kitcher fails to distinguish between the purely genetic ("Mendelian") and cytological levels of analysis.

Indeed, classical genetics as it is taught and used today comprises hypotheses both at the genetic (probabilistic laws concerning the transmission of genes) and cytological (cellular processes underlying this transmission) levels. The laying and consolidation of the foundations of classical genetics in the 1910s consisted to a large extent in the articulation of these two levels. A precise understanding of the content of classical genetics thus requires the clarification of this articulation. Hence, instead of assuming that classical genetics can be studied as a whole, my proposal relies on a preliminary distinction between what I call "pure" Mendelism and cytology.

First, I distinguish between pure Mendelism and cytology by characterizing them in terms of their respective associated representational practices. I then present how the two levels were articulated within Morgan's theory of crossing-over, and I describe the representational technique of linkage mapping designed and developed by Morgan's group in the 1910s. Linkage mapping embodies the "merging" of the Mendelian and cytological levels. I thus propose an analysis of the mapping scheme as a means to enlighten the conceptual articulation of Mendelian and cytological hypotheses in classical genetics. Finally, I present the views of three of Morgan's opponents in the 1910s—Richard Goldschmidt, William Bateson, and William Castle. These geneticists had a different understanding of the articulation of cytology and Mendelism, and entertained different views concerning the role and proper interpretation of maps. I propose to consider these diverging perspectives as instantiating different "versions" of classical genetics.

Representational Practices in Mendelism and Cytology

In the early 1900s, the Mendelian study of heredity and cytology were two distinct disciplines whose relations were far from clearly understood, let alone established. After saying a word about the methodological choice of studying representational practices, I will characterize Mendelism and cytology by describing the representational practices

[1] Hull (1976), Wimsatt (1976), and Sarkar (1998) also object that Schaffner's approach focuses too much on formal considerations and that one should pay more attention to practice.

[2] Waters claims that "one cannot understand the experimental strategies that geneticists employed to advance their agendas without understanding their theoretical reasoning about transmission" (Waters 2004, p. 785, fn 3).

[3] Waters would probably consider my account as "theory-biased" as Kitcher's, since I am interested in the theoretical aspects of classical genetics and (partially) neglect experimental practices. I do not aim at giving a complete picture of classical genetics, though, but rather at clarifying some conceptual issues that have generally been neglected in the literature.

[4] Despite my criticism of Kitcher's views in this article, I acknowledge that it took me long to clarify my disagreement. I would not have been able to develop my views without having read his paper.

that are typically associated with each of them. Finally, I will distinguish my approach from Kitcher's reconstruction of classical genetics' "patterns of reasoning."

Why Focus on Representational Practices?

Scientists do not reason in the abstract. Their day-to-day work (partially) consists in producing representations—equations, diagrams, schematic drawings, etc.—of the phenomena they study. By manipulating these representations they draw inferences in order to predict and explain these phenomena. But the reasoning processes they perform in practice, when using a particular representation, depend on its very form. Indeed, using a second-order equation in order to study the motion of a pendulum does not require the same kind of reasoning process as using a graph, or even a first-order equation (Vorms 2011).

A study of theorizing as a cognitive activity, then, requires one pay attention to the concrete representational devices that are constructed and manipulated in practice. Philosophical analyses of theories traditionally aim at formally reconstructing the logical content of theories, abstracting away from the cognitive aspects of theorizing. By contrast, I claim that examining the representational practices in a given scientific domain is a way to characterize the form of reasoning at play in this domain. In some cases, such attention to representational and reasoning practices may result in a reorganization of the boundaries between scientific domains (Humphreys 2004, pp. 68–69). In the case I am interested in, this approach will enable me to clarify the conceptual articulation of Mendelism and cytology within classical genetics, which the analyses of inter-theoretical relations in terms of laws and concepts generally miss.[5]

Like my own approach, Kitcher's characterization of classical genetics in terms of patterns of reasoning also stems from a rejection of an exclusive focus on laws.[6] However, in my view his account still belongs to a positivist-like approach to theories.

> A pattern of reasoning is a sequence of *schematic sentences*, that is sentences in which certain items of nonlogical vocabulary have been replaced by dummy letters, together with a set of *filling instructions* which specify how substitutions are to be made in the schemata to produce reasoning which instantiates the pattern. This notion of pattern is intended to explicate the idea of the common structure that underlies a group of problem-solutions. (Kitcher 1984, p. 353)

In other words, patterns of reasoning are empty syntactic arguments to be filled up. They are abstract structures underlying the scientists' reasoning processes.[7] Kitcher's approach to the "practice" of a science still abstracts away from how scientists do reason in practice. By contrast, I shall focus on the *concrete* representations with which geneticists actually reason. I take these representations as *already interpreted*, by which I mean that the reasoning or computational process a given representational device enables one to perform is not detachable from the representational content of this device.[8] Theorizing has both a representational and a computational component, and a study of theorizing should account for their articulation rather than artificially divorcing them from each other.[9] Let me now present Mendelism and cytology through the lens of their associated representational practices.

Mendelism

By "Mendelism" I refer to the Mendelian study of heredity before the introduction of the cytological components of classical genetics. As I construe it, Mendelism does not correspond to Gregor Mendel's theory as it could be inferred from his paper (Mendel 1866),[10] but rather to the work of the geneticists in the early 1900s after the rediscovery of Mendel's laws. My aim is not so much to recount the history of genetics in its early years as to characterize the representational practices, which I take to be typical of the Mendelian level of analysis, throughout genetics' history.

The experimental practice of Mendelism consists of breeding (hybridization) experiments on different strains of the same species, which rely on the choice of differential characters (e.g., green versus yellow color in Mendel's

[5] The debate on the problem of the reducibility of classical genetics to molecular biology was launched by Schaffner's (1969) seminal paper. Schaffner describes the case of genetics by means of an amended version of Nagel's (1961) model of intertheoretical reduction cast in terms of nomological deduction.

[6] The classical models of intertheoretical reduction formally depend on accounts of the structure of theories in terms of hypothetico-deductive sets of statements. Kitcher's (1984, p. 339) strategy consists in denying that "classical genetics contains general laws about the transmission of genes which can serve as conclusions of reductive derivations." To him, the logical deduction of a set of statements from another set of statements is not what we expect from a scientific explanation. Rather, explaining (and understanding) a type of phenomena implies the effective implementation of forms of reasoning; hence his characterization of classical genetics in terms of patterns of reasoning rather than sets of statements.

[7] Moreover, Kitcher's account of classical genetics' patterns of reasoning is cast in linguistic terms. As will appear, the use of nonlinguistic representations in science is of central importance for my conception of theorizing. But my main point against Kitcher's account is that he conceives of patterns of reasoning independently from their interpretation and implementation in scientists' minds.

[8] See Humphreys' (2004, p. 80) arguments against the "detachable interpretation view."

[9] See also Love's (2012) considerations about "formal and material theories in philosophy of science."

[10] On Mendel's "Mendelianism" see Olby (1985, 1997).

sweet peas). Geneticists trace back the transmission of hereditary factors—genes[11]—from statistical data concerning the distribution of observable characters among individuals in successive generations.

This practice is associated with a representation of genes as discrete, stable units by means of letters or icons on which combinatorial mathematics is applied. For example, the equation

$$9AB + 3Ab + 3aB + ab$$

expresses the expected distribution of genes among the germ cells for a cross involving two genes—two pairs of differential characters. It can be considered as a symbolic expression of Mendel's second law.[12]

Geneticists also developed other formats of representation in the early 1900s, such as the double entry arrays called *Punnett squares* (Fig. 1). Punnett squares are (two-dimensional) spatial extensions of the Mendelian symbolism that facilitate computing the distribution of genotypes among individuals in successive generations. They may also facilitate one's understanding of Mendelian theory itself by expressing Mendel's laws or other probabilistic rules about the transmission patterns of some particular genes in a given species.

Note that in Punnett squares the spatial display of information is only a means to help computation. Punnett squares contain exactly the same information as their corresponding equations.[13] The spatial relations within the array do not represent any spatial structure in the physical world. Clearly, the spatial display of the symbols standing for the genes does not tell us anything about the relative location of the genes as concrete physical entities.

As such, Punnett squares belong to the type of representations I propose to call "diagrammatic." *Diagrams*, as opposed to schematic representations, are a broad class of representations including graphs, arrays, flow charts, etc. that represent *non-spatial* (e.g., causal or temporal) *relations* by means of spatial relations. On the other hand, in *schematic representations*, spatial relations do stand for

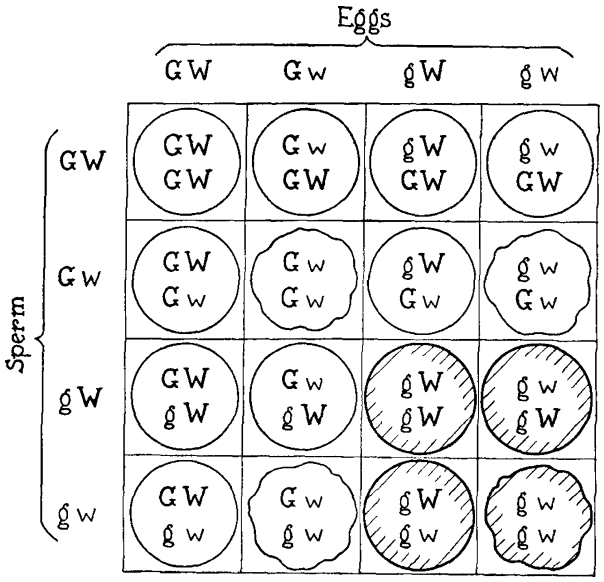

Fig. 1 Punnett square showing the expected distribution of genes among the germ cells for a cross involving two genes. It can also be considered as a diagrammatic expression of Mendel's second law. From Morgan (1928, p. 9)

spatial relations. Consider, e.g., the schematic drawing of a cell or chromosome: it might well distort the distances and abstract away from many aspects of its target considered irrelevant for the sake of the hypotheses it serves to express (Lynch 1988). But it has to conserve the topological, if not the metric, relationships of its target.[14]

Whether linguistic (equations) or diagrammatic (Punnett squares), Mendelian symbolism consists in representing genes as discrete entities to which combinatorial mathematics can be applied. Using this symbolism does not imply any assumption about the physicochemical nature of genes, nor about their mode of action.[15] As such, Mendelism conceives of genes as mere operational, abstract units whose transmission is supposed to follow probabilistic laws. Its method consists in a quantitative analysis of data obtained through breeding experiments.

[11] The term "gene" was introduced by Willem Johannsen in 1909. However, until 1917, Morgan's group would speak of "Mendelian factors."

[12] Mendel introduced this symbolism to present the statistical data obtained through his experiments on sweet peas as well as the probabilistic laws he inferred from these results. It is not clear what exactly he intended to represent by the letters—germ cells or "genes"? Note that for what we call "homozygous" individuals he used only one letter (*A*, rather than *AA*, as did the geneticists in the early 1900s). However, he undeniably introduced the practice of representing the genetic material as discrete entities and using combinatorial mathematics.

[13] To be sure, this is not exactly true, since the square gives us additional information about which phenotypes correspond with genotypes. However, these are iconic additions that have little to do with the spatial format of the square itself.

[14] In work in progress I elaborate this distinction between diagrams and schematic drawings as well as the kind of theorizing and of abstraction attached to each of these types of representations.

[15] Note that such agnosticism about the physicochemical nature and behavior of genes is also characteristic of classical genetics (as including the cytological level). Only molecular genetics will address such issues, which are epigenetic in nature, not genetic. Morgan (1917) explicitly presents the distinction between the problems of heredity and of development, as well as the temporary neglect of the latter, as a methodological necessity, which should eventually lead to a better understanding of gene action.

Cytology

Cytology, the study of cells and cellular processes, relies on imaging techniques, in particular on the use of microscopes. Contrary to Mendelian representations, the representational devices that are produced, used, and studied by cytologists represent concrete, physical (spatiotemporal) entities, such as cells and their components (chromosomes).

Following the typology proposed above, cytological representations are typically schematic representations. Theorizing in cytology (partially) consists in interpreting, and abstracting away from, raw images obtained by microscopes[16] by neglecting irrelevant information and highlighting some (often invisible) aspects of the object being represented, such as the boundaries between its components (which are themselves theoretical constructs).[17] The schematic drawings so obtained express hypotheses about the morphological properties of cells and chromosomes, as well as about their spatiotemporal behavior (e.g., the behavior of chromosomes during mitosis and meiosis).

Even when highly abstract, and very different from the raw images obtained by microscope, cytological representations remain schematic in the sense that spatial relationships do stand for spatial relationships. They could be mapped onto a microscope image of the same object.

What is Wrong with Kitcher's Characterization of Classical Genetics?

Kitcher's notion of a "pattern of reasoning" leads him to what I consider a (partially) wrong characterization of classical genetics. According to Kitcher, the typical reasoning associated with classical genetics consists in answering "questions about the distribution of characteristics in successive generations of a genealogy" by "using the probabilities of chromosome distribution to compute the probabilities of descendant genotypes" (Kitcher 1984, p. 354). These questions concerning the distribution of characteristics, to which the typically genetic patterns of reasoning are applied, are "pedigree problems."

> Each case of a pedigree problem can be characterized by a set of *data* [statements describing the distribution of phenotypes among the organisms in a particular pedigree, or a diagram conveying the same information], a set of *constraints* [general cytological information and descriptions of the chromosomal constitution of the members of the species], and a question [that refers to the organisms described in the data]. (Kitcher 1984, p. 355)

Solving such a problem, Kitcher claims, consists first in advancing a "genetic hypothesis" that specifies "the relevant genes, their phenotypic effects and their distribution among the individuals in the pedigree" (p. 354). Second, on the basis of this hypothesis, and given the constraints on the problem, "one computes the expected distribution of genotypes among the offspring" (p. 356).

I have two worries about Kitcher's description of classical genetics' patterns of reasoning. First, I think it misses a core aspect of theorizing in genetics embodied in the Mendelian representational practice as I have characterized it. To clarify this point, let me consider again the pedigree problems as described by Kitcher. I guess I do not misrepresent Kitcher's views when I claim that the pedigree problems and the patterns of reasoning that serve to solve them are embodied in the pedigree diagrams one can find in any genetics textbook. Consider the diagram in Fig. 2 in light of Kitcher's description of pedigree problems quoted above. The *data* about the distribution of phenotypes and genotypes are given by the form and color of the symbols standing for the individuals (circles for females, squares for males). The *constraints* are given by the icons representing chromosomes, whose colors indicate their origin and the genes they carry (see caption). The question is: how can a

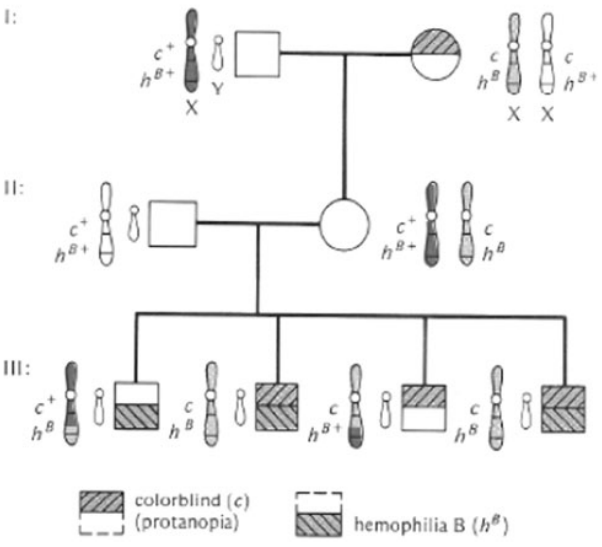

Fig. 2 Pedigree of a family segregating for the effects of two sex-linked genes, *colorblindness* (*c*) and *hemophilia* (h^B). The X chromosome linkages responsible for the phenotypes of generation III males are colored according to their origin: black (grandfather I-1) and grey (grandmother I-2). From Strickberger (1985, p. 301)

[16] The notion of "raw image" calls for clarification. Images obtained by microscope or other imaging techniques are themselves the result of much data processing and interpretation relying on theoretical constructs. But this topic is beyond the scope of this article.

[17] For developments of this idea, see Lynch (1988) and Maienschein (1991).

healthy female, whose father is healthy and mother is color-blind, mating with a healthy male who does not carry any gene responsible for either color-blindness or hemophilia, give birth to four male children with genetic diseases (more precisely: one hemophilic, one color-blind, and two suffering from both diseases)?

Pedigree diagrams obviously offer a *framework* within which one can express Mendel's laws, as well as more particular transmission patterns. In the case of the diagram in Fig. 2, knowing the transmission patterns of the genes responsible for hemophilia and color-blindness, and more generally of sex-linked genes, enables one to manipulate the diagram so as to explain the outcome in generation III. However, the very form of the pedigree diagram does not contain as such any Mendelian hypothesis. It could serve—and has actually served—to express various theories of heredity. One could posit non-Mendelian rules of manipulation and impose non-Mendelian constraints on a pedigree diagram. In fact, family trees are much more ancient than Mendelism.[18] Hence pedigree diagrams, which quite clearly embody pedigree problems as described by Kitcher, do not exhibit the specificity of Mendelian reasoning as characterized above.

Moreover—and this leads me to my second worry about Kitcher's account—the cytological information conveyed by the icons representing chromosomes imposes *external* constraints on how to manipulate the diagrams. But these constraints do not embody any specifically cytological explanation, as the very same constraints could be imposed by non-cytological information. The manipulation rules of the diagram have nothing to do with the cytological explanation of the genetic phenomena. Indeed, the proper explanation provided by cytology is mechanistic in kind. As I have suggested, the typical representations associated with the cytological level are schematic. I do not deny that the spatial disposition of the colors in the icons representing chromosomes in Fig. 2 is explanatory; nor am I suggesting that Kitcher is unaware of the fact that cytology provides a mechanistic explanation of the genetic phenomena.[19] But the kind of explanation that the information conveyed by the disposition of the colors in Fig. 2 provides does not play any role *within* the diagram and within the very pattern of reasoning as Kitcher describes it.

Hence I claim that Kitcher's account obscures the explanatory import of cytology by conflating classical genetics' pattern of reasoning with the cytological level of explanation. To clarify what exactly is the explanatory import of cytology to genetics we must first distinguish the representational practices of Mendelism and cytology. In the following I will show how the two have "merged" within a new representational technique, which embodies the birth of classical genetics.

Crossing-Over and Linkage Mapping

In 1911 Morgan adopted the chromosome theory of heredity, which states that the chromosomes are the physical basis of the genetic material. At the same time, he formulated the theory of crossing-over as a mechanistic explanation of the genetic phenomenon called "partial linkage." Morgan's "conversion,"[20] together with his mechanistic explanation, contributed to the launch of the research program of the Drosophila group composed of Morgan and his students Alfred Sturtevant, Hermann Muller, and Calvin Bridges, integrating the genetic (Mendelian) and cytological levels into one theory. Before turning to the analysis of the technique of linkage mapping I will briefly recall what Morgan's theory consists of.

Morgan's Theory of Crossing-Over

Two empirical discoveries—one genetic, the other cytological—prompted Morgan's adoption of the chromosome theory of heredity and his formulation of the theory of crossing-over.

Genetic Level: Partial Linkage

From 1905 on, geneticists observed a phenomenon that seems to contradict Mendel's second law. New data[21] showed that some genes tend to be inherited together without being always so. For example, genes of *Lathyrus odoratus* responsible for the color of the petals and the shape of the seeds appear to be "partially linked" or "coupled." Contrary to what Mendel's second law predicts, they are not randomly redistributed: partially linked genes are inherited together in more than 50 % of the cases, but their association is not systematic—they are

[18] One could even argue that the form of the pedigree diagrams is a remainder of previous conceptions of heredity. According to Gayon (2000): "For Mendelian genetics, a pedigree was a tool, and no longer a fundamental concept. In previous theories built upon the concept of ancestral heredity, heredity was the sum total of influences received from the ancestors. […] In this context, 'heredity' was nearly synonymous with 'descent' or 'lineage,' or else 'pedigree.' For Mendelism, the *origin* of characters was an irrelevant issue. The pedigrees had nothing to tell us about the *nature* of heredity; they were only tools for inferring the genetic structure of individuals."

[19] He analyzes this in detail in terms of what he calls "*PS*-processes" ("*PS*" standing for "pair-separation").

[20] On Morgan's intellectual evolution see Carlson (1967) and Allen (1978).

[21] The first case of partial linkage (or rather "coupling of traits") was reported by Bateson et al. (1905).

inherited together in less than 100 % of the cases. A few years later, Morgan (1910) observed that sex-linked genes (i.e., genes that appear to be linked to what was assumed to be the genes responsible for sex determination)[22] were themselves partially linked to each other.

Cytological Level: Chromosomes' Intertwining

One cytological discovery played a determining role in Morgan's adoption of the chromosome theory of heredity by suggesting a mechanistic explanation for the phenomenon of partial linkage. In 1909, the cytologist Janssens observed that homologous chromosomes intertwine during meiosis. He conjectured that homologous chromosomes might exchange segments while intertwining. Note, however, that no such exchange was observed before the 1930s. Janssens called this putative physical exchange of segments of chromosomes "chiasmatypie." This cytological hypothesis prompted Morgan's proposition of a mechanistic explanation of partial linkage.

The Mechanistic Model of Crossing-Over

Morgan hypothesized that groups of genes might be linked together on "linkage groups," which he identified with chromosomes. Linkage would explain the tendency of the genes concerned to be transmitted together during the formation of the germ cells. Hence, when *chiasmatypie* occurs, the genes lying on the portions being exchanged are also exchanged. This would explain that in some cases genes that are usually linked are inherited separately. Morgan and Cattell (1912) labeled such gene exchange "crossing-over." Its result, the separate redistribution of partially linked genes, is called "recombination." As shown in Fig. 3, the hypothesis of crossing-over provides a mechanistic explanation of the genetic phenomenon of partial linkage.

Additivity and Linearity

A crucial hypothesis underlies Morgan's model: genes are supposed to be linearly ordered along the chromosome like beads on a string. An important aspect of partial linkage suggested this hypothesis. Data concerning the recombination frequencies of different pairs of genes (i.e., the frequency with which partially linked genes are redistributed separately) showed that such frequencies were additive, meaning that for three genes A, B, and C belonging to the same linkage group the recombination frequency $R(AC)$ of A and C is the sum of the recombination frequencies $R(AB)$ and $R(BC)$. Hence the hypothesis of linearity.

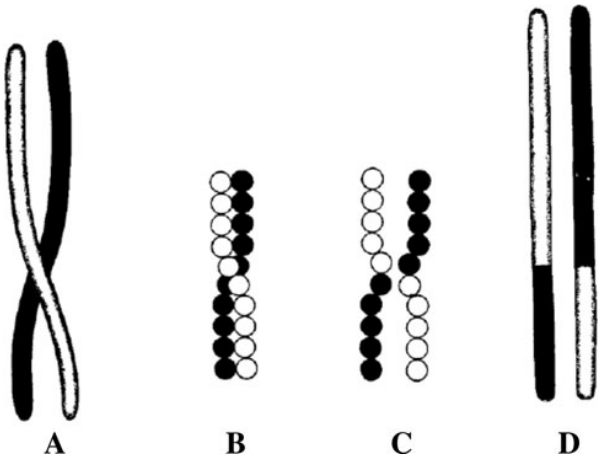

Fig. 3 The mechanistic model of crossing-over. From Morgan et al. (1915, p. 60)

Proportionality of Recombination Frequency and Distance

Morgan drew an important conclusion from his mechanistic model: he suggested that the recombination frequency of two genes (observed to be constant for any given pair of genes and different for different pairs), being a consequence of the frequency of breaks occurring on the linkage groups, is a function of the *distance* between the genes concerned. Indeed, as appears in the model, the more distant two genes are from each other, the more a break between them is likely to occur, and hence the more likely they are to be redistributed separately.

Before turning to the presentation and analysis of the technique of linkage mapping I want to just insist that the explanation provided by the theory of crossing-over is a mechanistic one, well expressed by the schematic drawing in Fig. 3. Hence, crossing-over does not belong to "pure Mendelism." The reasoning processes in which it appears do not consist in applying combinatorial mathematics to discrete, operational units. However, one has to be careful and note that the schematic drawing in Fig. 3 must not represent the actual structure of chromosomes (of which no fine observation was available at the time) in order for the model to be explanatory. In fact, it does not have to represent a chromosome at all. It is enough to consider it as a representation of a linkage group, whatever the physical basis of linkage groups may be. The whole theory of crossing-over and partial linkage could indeed be stated at the genetic level, without reference to chromosomes.[23] But

[22] Already in 1891, cytologists had identified a non-paired chromosome (a chromosome lacking its homologue), which Wilson called "X." But the hypothesis of the chromosome determination of sex was controversial until the 1910s.

[23] In the first chapter of his *Theory of the Gene*, Morgan (1928) in fact states his theory without any reference to the chromosomal level.

the genetic level so construed is not "purely Mendelian": the explanation is mechanistic, and the reasoning with which it is associated is not a purely combinatorial one, since it involves spatial reasoning.

Linkage Mapping

In 1913, Sturtevant transformed Morgan's theory of crossing-over into a mapping scheme for the linkage groups. Linkage maps were primarily intended to represent the relative location of genes along the chromosomes. Assuming, with Morgan, that the recombination frequency between two genes of the same group is proportional to the distance between them, Sturtevant proposed that this frequency could be used as an index of the distance separating these two genes on the chromosome. Hence, one could map the relative location of genes on a one-dimensional graph.

On the basis of the frequencies calculated from the results of breeding experiments, Sturtevant (1913) constructs the map for the X chromosome of *Drosophila*. Table 1 displays the recombination frequencies of the genes of the group of sex-linked genes (corresponding to the X chromosome). These frequencies are themselves inferred from the statistical data of the distribution of phenotypes among various individuals in successive generations. The map in Fig. 4 displays the recombination frequencies by transforming them into visualizable distances. Genetic or mapping distance is thus initially defined as a linear function of recombination frequencies.

Sturtevant's scheme, however, is not as simple as stated above. The first complication (the only one I will consider here) is that for long distances (standing for high recombination frequencies) some experiments (Morgan 1911; Morgan and Cattell 1912) show exceptions to additivity. For two genes A and C with high recombination frequency $R(AC)$ one finds $R(AC) < R(AB) + R(BC)$.

Instead of rejecting the hypothesis of linearity (which, as I recall, was initially justified by the observation of additivity), Sturtevant hypothesized that there could be more than one crossing-over occurring on the same linkage group at the same time. As is shown in Fig. 5, double crossing-overs would cancel the recombination of the corresponding genes—the genes located at the extremities of the linkage groups, and separated by two breaks, are, in the end, inherited together (they remain on the same chromosome).

Drawing on this hypothesis, Sturtevant chooses to construct his map by relying on the short distances (low recombination frequencies).[24] Long distances on the map therefore correspond to the *sum of short distances*, rather than to the observed[25] recombination frequencies between the most distanced genes. This is obvious when one considers the table of the recombination frequencies (Table 1) and its corresponding map (Fig. 4). The table displays the proportions of crossing-over for each pair of genes (their recombination frequency), and the corresponding percentage, which is supposed to give the distance between them. Consider *BM*: the table says that, out of 693 cases, *B* and *M* were inherited separately 260 times, i.e., 37.6 % of the cases. However, on the map, the distance between *B* and

Table 1 Table of recombination frequencies (Sturtevant 1913, p. 48)

Factors concerned	Proportion of cross-overs	Percent of cross-overs
BCO	$\frac{193}{16287}$	1.2
BO	$\frac{2}{373}$	0.5
BP	$\frac{1464}{4551}$	32.2
BR	$\frac{115}{324}$	35.5
BM	$\frac{260}{693}$	37.6
COP	$\frac{224}{748}$	30.0
COR	$\frac{1643}{4749}$	34.6
COM	$\frac{76}{161}$	47.2
OP	$\frac{247}{836}$	29.4
OR	$\frac{183}{538}$	34.0
OM	$\frac{218}{404}$	54.0
CR	$\frac{236}{829}$	28.5
CM	$\frac{112}{333}$	33.6
B(C, O)	$\frac{214}{21736}$	1.0
(C, 0)P	$\frac{471}{1584}$	29.7
(C, 0)R	$\frac{2062}{6116}$	33.7
(C, 0)M	$\frac{406}{898}$	45.2
PR	$\frac{17}{573}$	3.0
PM	$\frac{109}{405}$	26.9

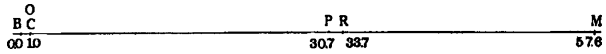

Fig. 4 Linkage map corresponding to Table 1. (Sturtevant 1913, p. 49)

Footnote 23 continued
The rest of the book is intended to show that (and how) the chromosome theory provides a good explanation of the genetic theory itself.

[24] Here I focus on the representational scheme designed by Sturtevant rather than on the experimental methods underlying it. This is not to deny that the particular genetic markers were selected for their experimental manipulation value, as much as for mapping resolution.

[25] Note that "observed" here means inferred from the statistical data about the distribution of phenotypes on the basis of already quite sophisticated genetic hypotheses.

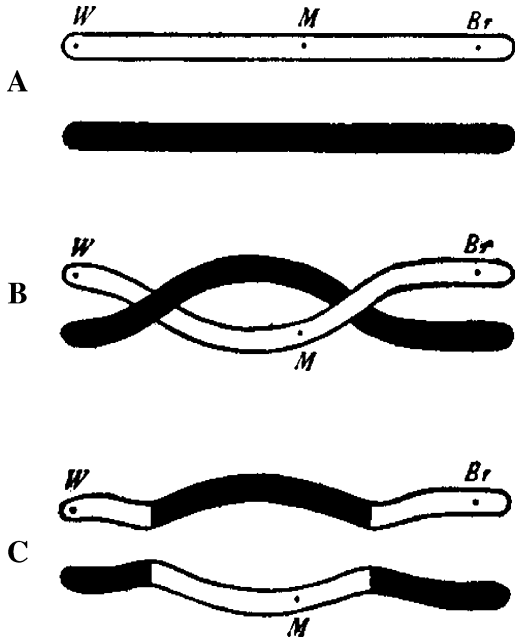

Fig. 5 Schematic representation of double crossing-over (Morgan et al. 1915, p. 62)

M is 57.6, and not 37.6, because this distance was calculated by adding up short distances rather than by relying on the recombination frequencies that could be inferred from the phenotypic data.

What and How Do Linkage Maps Represent?

What kind of representations do linkage maps belong to? Are they Mendelian representations or cytological ones? Are they diagrammatic (graphical) representations of statistical data (recombination frequencies) or schematic representations of chromosomes?

Maps as Schematic Representations of Chromosomes

At first sight one might want to argue that maps are schematic representations of the same type as the mechanistic model of crossing over (Fig. 3). True, they are not constructed the same way, but their function is nevertheless to spatially represent spatial relations, viz., the relative location of the genes on the chromosomes. In fact, highlighting (aspects of) the structure of chromosomes is what linkage (or "chromosome") maps were originally intended to do. The initial motivation and justification of the mapping scheme is Morgan's theory of crossing-over. Moreover, the very enterprise of mapping yielded confirmation of the chromosome theory and good knowledge of the structure and role of the four chromosomes of *Drosophila*. In fact, in the 1930s it became possible to map linkage maps onto cytological maps obtained through microscopy.

Hence, even if genetic distance (as already acknowledged by Sturtevant 1913)[26] might not correspond exactly to physical distance (i.e., the metric is not conserved), at least the relative ordering (topological relations) of the genes is conserved. In this sense maps are cytological-like (schematic) representations. They are to be read as chromosome-representations, although they are constructed on the basis of genetic data.

Maps as Mendelian Graphs

On the other hand, it is worth acknowledging that the technique of mapping would still have been meaningful and useful had the chromosome theory turned out to be false. In fact, maps are obtained through Mendelian means (breeding experiments and statistical analysis), and they graphically display the data contained in the corresponding tables. Even if they did not represent any real physical structure, maps could still serve as inference tools to visualize statistical data. They would contain no more information than the corresponding tables, but they would be much more efficient than them as enhancers of computation.

From such a perspective, maps are mere graphical extensions of the Mendelian symbolism, like Punnett squares. They are only metaphorically spatial (like a temperature graph, prompting one to say that temperature is "high" or "low"), distances standing for mere probabilities. They are pure graphical presentations of statistical data without spatial meaning. I will now show that this interpretation is untenable.

Maps Have a Spatial Meaning

Although nothing proves that mapping distances correspond to real distances within the chromosomes, they do not merely correspond to simple statistical data. Indeed, as I have explained, distances on the map do not always correspond to the observed recombination frequencies, but rather to the real physical exchanges one might infer from adding up smaller recombination frequencies. Long distances stand for the putative probability of real, physical crossing-over to occur. True, the physical basis of linkage groups could be something else than chromosomes. But the mechanistic model underlies the very mapping scheme. And, as we have seen, the mechanistic model is not

[26] Sturtevant (1913, p. 49) indeed hypothesized that chromosomes might not be equally likely to break on every point, which would imply that distance is a measure of strength combined with length.

reducible to pure Mendelism, since it involves spatial reasoning.

Whatever the physical basis of linkage groups may be, the maps need to be interpreted in a spatial way (spatial relations within the map must be interpreted as standing for spatial relations) to be properly read—in order for the information on recombination frequencies (genetic data) to be retrieved. For someone who does not assume that distances in the map stand for physical distances (or at least spatial relations for spatial relations), it would prove practically impossible to properly use the map.[27]

Hence, mapping distance is not a purely Mendelian concept, and linkage maps are not purely Mendelian representations: they involve a mechanistic hypothesis. They involve the very idea of the spatiality of the genes and their location in a non-metaphorical way. Sturtevant's mapping scheme exceeds pure Mendelism. Maps remain Mendelian representations insofar as they rely on statistical data, but the Mendelian symbolism is "integrated" into another form of representation, which implies mechanistic, spatial thinking.

Analyzing maps enables us to identify an essential aspect of classical genetics, viz., the integration of a conception of heredity in terms of probabilities and its mechanistic explanation. I hope to have shown that this analysis better captures the explanatory import of cytology than does Kitcher's approach.[28]

Maps are not mere graphical presentations of statistical data. Their format (rules of construction and interpretation) involves a theoretical hypothesis. As such they are genuine theoretical representations. Linkage maps embody the articulation of the genetic and cytological levels in a way that no abstract reconstruction of theories (or patterns of reasoning) captures. They are at the same time Mendelian and cytological-like representations. Representations such as linkage maps are not the expression of an underlying theory (or pattern of reasoning), but the very locus of theorizing.

From the Agents' Point of View: Versions of Genetics

From the moment when the mapping technique was designed, the construction and analysis of the genetic maps of *Drosophila* became the object of a genuine research program on which geneticists concentrated their efforts for at least two decades. In addition to yielding detailed knowledge of many genetic phenomena, the mapping enterprise provided geneticists with growing evidence in favor of the chromosome theory by enabling them to relate genetic phenomena (i.e., drawn from the analysis of data obtained by breeding experiments) with cytological phenomena (e.g., chromosomal aberrations).

Today, the cytological and Mendelian levels are both considered as integral parts of classical genetics. However, until the early 1920s the articulation of these two levels was far from clearly understood, and many geneticists still rejected Morgan's theory. In this last section I sketch the respective positions of three opponents to Morgan, viz., Richard Goldschmidt, William Bateson, and William Castle. Each of them holds a different view about the kind of explanation cytology can provide to genetics. As a consequence, they have different conceptions of the status and meaning of linkage maps. I will propose to consider these diverging perspectives as instances of what I call different "versions" of genetics. Let me first say a word about what I mean by "version."

Theories and Their Versions

Theories are not monolithic blocks. They do not have clear-cut boundaries distinguishing them from other theories either synchronically or diachronically. Moreover, within what is usually considered as one and the same theory, there can be variations that traditional accounts (both in the logical-empiricist and in the Kuhnian traditions) ignore. Kitcher's study of the "versions" of classical genetics is an attempt to give a more fine-grained account of the historical development of classical genetics. For Kitcher, versions are successive implementations of the same pattern of reasoning—different ways of "filling up" the syntactic scheme, so to speak.

My notion of "version" also stems from the conviction that approaching theories as monolithic blocks is unsatisfactory. However, my approach is more agent-centered than Kitcher's. Not only is it worth studying intra-theoretical variations, but one should not neglect either that theories do not exist independent of the minds of the agents who develop, understand, and use them. To borrow Griesemer and Wimsatt's (1989, p. 87) words, "theories require theorizers, and abstract entities, entifiers." I rather construe versions as different *ways of understanding and practicing a theory*, different perspectives on it, which can be synchronic as well as diachronic. A version of a theory is, so to speak, the theory *as understood by an agent*. One's version of a theory is one's own way of using and reasoning with this theory. It is the theory as implemented in one's reasoning processes, which could be defined as the set of mental representations and inferential paths one makes use

[27] In fact, as I have shown in Vorms (2012), if one refuses to consider them as theoretical representations bearing a mechanistic explanation of the genetic phenomena, maps are far from being handy predictive tools.

[28] Interestingly, Kitcher (1984, pp. 357–358) classifies linkage mapping as a "subtheory."

of when learning, developing, and applying a given theory to the phenomena.

Different formulations of the same theory, as for instance the Newtonian, Lagrangian, and Hamiltonian formulations of classical mechanics, can be considered as expressions of different versions of this theory. Although logically equivalent, their conceptual architecture is different; they do not relate to the empirical world by means of the same concepts, and the deductive order of their different principles is not the same.[29] They do not facilitate the same inferences, nor do they prompt one to follow the same inferential paths. They are both *representationally* and *computationally* different. Depending on which formulation is used, one does not get the same understanding of the phenomena and of what classical mechanics says about them.[30]

But even theories that admit of only one standard formulation can be understood and used in different, though consistent,[31] ways by different agents. One's version of a theory depends on the way one has learned it, one's background knowledge, reasoning habits, theoretical commitments, skills, etc. Strictly speaking there are as many versions of a theory as there are agents using, and reasoning with, it; moreover, individual agents may themselves change their views throughout their career, or depending on the context in which they are using the theory. However, according to what one is interested in, it is quite reasonable to abstract away from individuals and identify types of versions (Vorms 2010). For instance, in the present case, when studying the debates between Morgan's group and his opponents, I take the Morgan group's version as one and only.

Contrary to what is the case in classical mechanics, the versions of classical genetics I will now present are not logically equivalent. The notion of version is a way to account for intra-theoretical variations, both in well-developed and established sciences (like classical mechanics) and theories at early stages of their development.[32] Classical genetics as taught and used today can be described as a stabilized and enriched version of Morgan's version. In the 1910s, though, Morgan's version was only one way among others to understand the articulation of the Mendelian level (on which all agreed) and the cytological level.

Three Non-Morganian Versions of Genetics

The chromosome theory of heredity at the core of Morgan's version of genetics (and classical genetics as we know it today) was still challenged in the 1910s, even by orthodox Mendelians such as Bateson. However, my analysis will show that the object of disagreement between Morgan and his opponents is not as simple as the alternative between accepting and rejecting the chromosome theory. The three protagonists I will introduce here were all Mendelians in the sense that they accepted all the purely Mendelian hypotheses presented above.[33] Yet, beyond this agreement, they held very different views of the physical basis of these phenomena. The various hypotheses constituting Morgan's theory that may seem non-dissociable in retrospect were in fact susceptible to being held independently from each other.[34]

Goldschmidt (1917): "Crossing-over Without Chiasmatypie"

Richard Goldschmidt was one of the fiercest opponents to Morgan's theory. One reason for this was that he would reject the distinction between the study of heredity and the study of development, which is at the core of Morgan's methodology from 1910 on.[35] For Goldschmidt, a theory of the gene had to say something about the mechanism of gene action.[36] The model he proposed was intended to make room, if not to give central stage, to gene action.[37]

[29] The core principle of the Newtonian formulation is Newton's second law, whose central concept is the concept of "force." The core principle of the Hamiltonian formulation is the principle of least action, which is expressed in terms of energy. Every principle and law can be retrieved from the two formulations, but their place in the deductive architecture changes: what is a fundamental principle here becomes a derived consequence there, and vice versa.

[30] This case, and its analysis in terms of versions, is worth comparing with Kuhn's views on the incommensurability of the Newtonian and Einsteinian "paradigms". I am developing this in a work in progress. On the versions of classical mechanics, see Barberousse (2008).

[31] It is worth emphasizing that I do not consider misuses and misunderstandings of a theory as proper versions of it. The inferences that one is entitled to draw are extremely constrained by the logical relations between the concepts of a theory as well as by the empirical phenomena. But my point is that, given these constraints, there still exist various possible inferential paths (Vorms 2010). In some cases, such as classical mechanics, they are logically equivalent. In less-developed sciences such as the classical genetics of the 1910s they may be much less compatible.

[32] The analysis in terms of version tends to both attenuate inter-theoretical differences—*contra* Kuhn's (1962/1970) dramatic notion of incommensurability—and to emphasize intra-theoretical variations (which are obscured by Kuhn's holism).

[33] From that point of view, Goldschmidt is the more problematic. But, as we will see, he accepted many Morganian hypotheses, though understanding and articulating them differently.

[34] For a more detailed study of the various epistemic attitudes geneticists (in particular Morgan's group and Castle) could entertain towards the different components of Morganian genetics see Vorms (2012).

[35] See note 15.

[36] For a study of Goldschmidt's physiological and developmental genetics see Allen (1974), and Dietrich (2000).

[37] For a detailed analysis of Goldschmidt's (1917) model see Wimsatt (1987).

Goldschmidt accepted two of the Morgan group's most fundamental hypotheses that were controversial elsewhere (even for "purer" Mendelians than Goldschmidt), viz., the chromosome theory and the linearity hypothesis. However, he rejected *chiasmatypie* as the hypothesis of a physical exchange of portions of chromosomes. Note that he did accept the hypothesis of a *genetic* crossing-over, that is, of an orderly exchange of genes between homologous chromosomes. But he denied that this phenomenon be related to a *physical* exchange at the chromosomal level. According to him, chromosomes would dissolve during cellular divisions. During such dissolution, genes would move into the cytoplasm, where they would cause their phenotypical effects. Chromosomes would then re-form during the next cellular division, with some genes having changed position. From this perspective, genes are not *portions* of chromosomes. They are rather attached to the chromosomes by some biochemical forces.

Goldschmidt's explanation of the relative motion of genes is indeed in terms of "forces." To each allele is associated a force of a given intensity. The more different two alleles are regarding their force, the closer to each other they lay on the map. Hence, Goldschmidt would not deny the usefulness and relevance of linkage maps, which he interprets as a handy representation of the relative forces of the genes. But, to him, genetic distances do not have any spatial meaning. Despite his acceptance of the chromosome theory of heredity, he does not interpret maps as representing the relative location of the genes on their chromosomal support. Mapping distances rather stand for differences of intensity between the biochemical forces associated to the genes.

Goldschmidt's theory was flawed in many ways. In particular, as Sturtevant (1917) shows, it cannot account for the phenomenon of multiple crossing-overs (which Goldschmidt could not reject, since he accepted linearity). In Goldschmidt's model, indeed, genes' exchanges on a locus should be independent from genes' exchanges on another locus. My point here is not to assess the virtues and flaws of Goldschmidt's position, though. What this brief overview shows is that one could accept many fundamental hypotheses of Morgan's theory (crossing-over, linearity, and the chromosome theory) while holding thoroughly different views on heredity and on the way hereditary phenomena articulate with their cytological basis. Because of the poor knowledge of the structure of chromosomes available at the time, the statement according to which chromosomes are the physical basis of the genes underdetermines the way one construes this "location" of genes "on" chromosomes.

Bateson's (1916) Criticism

Bateson was a Mendelian from the outset. Contrary to Goldschmidt (who remained an opponent to Morgan's theory until the 1950s), Bateson's criticism is thus internal to "orthodox" Mendelism. However, in 1916, he still rejected most of the hypotheses underlying the Morganian model of crossing-over.

Bateson (1916) rejects the chromosome theory. He nevertheless admits the relevance of the mapping technique, and he does accept the linearity hypothesis. His rejection of the chromosome theory comes with a rejection of the hypothesis of a genetic crossing-over (whatever its physical support be). He considers crossing-over as part of a battery of ad hoc hypotheses aimed at saving the chromosome theory, rather than a *confirmation* of it. Because no observational proof of a chromosomes' break during meiosis was available at that time, Bateson considered the whole edifice as built on sand.

Bateson's position, when compared with Goldschmidt's, shows that one could accept the fundamental Mendelian principles (those which are expressed in the Mendelian symbolism, as defined above), while rejecting the chromosome theory, and even the representation of genes as material particles.[38] Despite the fact that the invention of linkage maps was prompted by Morgan's theory of crossing-over, which was itself historically (if not conceptually) correlated with his acceptation of the chromosome theory, Bateson's position shows that maps could be of great relevance, independently from their interpretation in terms of chromosomes.

Castle (1919) and Linearity

Like Bateson, Castle is a "genuine" Mendelian. However, his criticism is, so to speak, diametrically opposed to Bateson's. He accepts the chromosome theory, the hypotheses of *chiasmatypie* and (simple) crossing-over, and the mapping scheme insofar as it consists in representing recombination frequencies as distances. But he rejects one of the fundamental hypotheses of Morgan's theory (which Bateson accepts), viz., linearity. In fact, he rejects the whole mechanistic model of which linearity is a crucial element, and proposes a three-dimensional model of map underlain by a chemical understanding of crossing-over.[39]

[38] Indeed, Bateson's representation of genes was in terms of charges rather than material particles; such representation had the advantage of offering an explanation of embryologic development.

[39] I have studied the debate between Morgan's group and Castle in some detail in Vorms (2012).

Conclusions and Further Thoughts on the Versions of Genetics

My purpose here is not to assess the (un)warranted character of the different versions of genetics I have presented. My point is only to show that professional geneticists in the 1910s could entertain different attitudes towards the various hypotheses constituting classical genetics, thus having different versions of this theory. Even when accepting the chromosome theory (which was the case of both Goldschmidt and Castle), one could have a very different understanding of the articulation of the cytological and genetic level. According to whether one conceives of genes as material particles, forces, or charges, one would interpret differently the very idea of their "location on" chromosomes. Moreover, according to one's conception of the proper object and aim of genetics (whether or not it is to explain development and make room for gene action), one would not represent genes and their physical basis the same way. As a consequence, one would have a different understanding of the role of maps, and of the meaning of mapping distance.

To conclude, I want to suggest that a study of the representational practices of geneticists may also shed light on the articulation of the different levels of analysis of classical genetics *today*. And this might be a good preliminary to approaching the question of the relations of classical genetics to molecular biology.[40] Indeed, focusing on representational practices, and clarifying how practices originating in different disciplines may be articulated within a given scientific domain, may shed new light on the issue of intertheoretical relations. A study of representational practices and the scientists' own versions of a theory is a good way to capture the conceptual articulation of a science in a way that formal approaches miss.

Even today, biologists might entertain different versions of classical genetics, according to their background, training, and the context in which they use this theory. I suggest that the question of whether classical genetics can be reduced to molecular biology might be a pragmatic question worth approaching from the agents' point of view.[41]

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[40] See Vance (1996) for similar considerations about the levels of analysis in genetics from the perspective of experimentation methods.

[41] Patrice David (personal communication, October 2009) suggested that such a perspective might show that most biologists today, who "believe they practice a unified science," have a "dissociated mind" that appeals, according to the context, to different versions of genetics. Passing from one to the other requires "a long training."

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