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Theorising and Representational Practices in Classical Genetics,
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Penultimate version

My aims in this paper are twofold. First, I wish to argue for a study of *theorising*, rather than of *theories*. Most philosophical studies of theories construe them as abstract structures independent from the agents' understanding of them. By contrast, the approach I advocate 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 time, as well as its development, and its relations to other scientific domains.

Here, my defence of such an approach to theorising takes the form of a historical case study in classical genetics. I analyse the representational practices of the geneticists in the 1910's. My second aim is thus to shed light on the conceptual content of classical genetics at that time. But, since classical genetics was already well developed in the early 1920's, 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, namely whether classical genetics has been reduced by molecular biology (see Schaffner 1969, Hull 1972, 1979, Wimsatt 1976, Darden & Maull 1977, Kitcher 1984, Rosenberg 1985, Burian 1985, Waters 1990, Sarkar 1998). The question is usually put as follows: Are the laws and concepts of classical genetics deducible from (and definable in terms of) the laws and concepts of molecular biology? In order to answer it, 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, and reasoned with, by scientists. These approaches miss important aspects of the content of classical genetics, on which my study aims to shed light.

Some philosophers and historians, who advocate a study of scientific *practice*, have already challenged theory-centred approaches to scientific knowledge, and in particular to classical genetics (e.g., Kitcher 1984, Kohler 1994, Waters 2004).¹ However, my approach diverges from these various proposals in important respects. Firstly, unlike Kohler's socio-constructivist study of the experimental practices of geneticists, my proposal aims 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

¹ Hull 1976, Wimsatt 1976, and Sarkar 1988 also object that Schaffner's approach focuses too much on formal considerations, and that one should pay more attention to practice.

geneticists. However, I am interested in practice insofar as it is an essential part of theorising. To this regard, my approach is closer to Waters', who reproaches Kohler for considering as irrelevant the geneticists' "theoretical interests" (Waters, 2004, 785).² Although I am convinced by his arguments, my study of classical genetics differs from Waters', insofar as I lay stress on *representational*, rather than on *experimental* practices.³ I aim at characterising classical genetics by identifying a certain form of reasoning, which is embodied in the use of a certain type of representations.

Characterising 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 of 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 in the course of the paper. Briefly, his patterns of reasoning are, in my view, still too abstract units of analysis. And such an abstract conception, I suggest, leads Kitcher to mischaracterise the content of classical

² Waters' claims that "one cannot understand the experimental strategies that geneticists employed to advance their agendas without understanding their theoretical reasoning about transmission" (*ibid.*, fn.).

³ Waters would probably consider my account as "theory-biased", like Kitcher's, since I am interested in the theoretical aspects of classical genetics, and I (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 criticising Kitcher's views in this paper, I do acknowledge that it took me long to clarify my disagreement. I would not have been able to develop my views without having read him.

genetics. In particular, like almost every philosophical account of classical genetics that has been proposed in the framework of the reductionism/antireductionism debate, Kitcher fails to distinguish between the purely genetic (or "Mendelian") and the cytological levels of analysis.

Indeed, classical genetics, as it is taught and used today, comprises hypotheses both at the genetic level (probabilistic laws concerning the transmission of genes), and at the cytological level (cellular processes underlying this transmission). The laying and consolidation of the foundations of classical genetics, in the 1910's, 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 characterising them in terms of their respective associated representational practices. I then present how the two levels articulated within Morgan's theory of crossing-over, and I describe the representational technique of linkage mapping, which was designed and developed by Morgan's group in the 1910's. Linkage mapping embodies the "merging" of the Mendelian and cytological levels. I thus propose an analysis of the mapping scheme, as a mean to enlighten the conceptual articulation of Mendelian and cytological hypotheses within classical genetics. Finally, I present the respective views of three opponents to Morgan in the 1910's (Richard Goldschmidt, William Bateson, and William Castle). These

three geneticists had a different understanding of the articulation of cytology and Mendelism, and they 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.

REPRESENTATIONAL PRACTICES IN MENDELISM AND CYTOLOGY

In the early 1900's, 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 characterise Mendelism and cytology by describing the representational practices, which 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 abstracto*. 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 the very form of that representation. 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 (see Vorms 2011).

A study of theorising, as a cognitive activity, thus requires that 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, thus abstracting away from the cognitive aspects of theorising. By contrast, I claim that examining the representational practices in a given scientific domain is a way to characterise the form of reasoning at play in this domain. Such attention to representational and reasoning practices may result, in some cases, in a reorganisation of the boundaries between scientific domains (see Humphreys, 2004, 68–69). In the case I am interested in, this 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

Kitcher's characterisation 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, Kitcher's account still belongs to a positivist-like approach to theories.

⁵ The debate on the problem of the reducibility of classical genetics to molecular biology has been 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 inter-theoretical reduction, which is cast in terms of nomological deduction.

⁶ The classical models of inter-theoretical reduction (inherited from Nagel, 1961) are formally dependent on accounts of the structure of theories in terms of hypothetico-deductive sets of statements. Kitcher's strategy consists in denying that "classical genetics contains general laws about the transmission of genes which can serve as conclusions of reductive derivations" (1984, 339). 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

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, 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 this, I mean that the reasoning (or computational) process, which a given representational device enables one to perform, is *not detachable* from the representational content of this device.⁸ Theorising has both a representational and a computational component, and a study of theorising should account for their articulation, rather than artificially divorcing them from each other.⁹ Let me now present

this characterisation of classical genetics in terms of patterns of reasoning, rather than in terms of 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 theorising. But my main point against Kitcher's account is rather that he conceives of patterns of reasoning independently from their interpretation, and from their implementation, so to speak, in the scientists' minds.

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

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

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 1865 paper¹⁰, but rather to the work of the geneticists in the early 1900’s, 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 characterise 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 (hybridisation) experiments on different strains of the same species, which rely on the choice of differential characters (e.g., green *versus* yellow colour in Mendel’s sweet peas). Geneticists trace back the transmission of hereditary factors – genes¹¹ – 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

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

¹⁰ On Mendel’s “Mendelianism”, see Olby (1985, 1997).

¹¹ The term “gene” was introduced by Willem Joannsen in 1909. But, until 1917, Morgan’s group would rather speak of “Mendelian factors”.

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 1900's, such as the double entry arrays called "Punnett squares" (see fig. 1). Punnett squares are spatial (two-dimensional) extensions of the Mendelian symbolism, which facilitate one's computing the distribution of genotypes among individuals in successive generations. They may also facilitate one's understanding of the 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 mean to help computation. Punnett squares contain the exact 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.

¹² Mendel did introduce 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. However, it is not clear what exactly he intended to represent by the letters (germs cells or "genes")?) Note that, for what we call "homozygous" individuals, he used only one letter, rather than two (A, rather than AA, as did the geneticists in the early 1900's). 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 here, since the square gives us additional information about what phenotypes correspond with genotypes. However, these are iconic additions that have little to do with the spatial format of the square itself.

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., which represent *non-spatial relations* (e.g., causal, or temporal relations) by means of spatial relations. On the other hand, in *schematic representations*, spatial relations do stand for spatial relations. Consider for instance the schematic drawing of a cell or of a chromosome. It might well distort the distances, as well as abstract away from many aspects of its target, which are considered irrelevant for the sake of the hypotheses it serves to express (see 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, on 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.

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

¹⁵ Note that such agnosticism about the physicochemical nature and behaviour 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, and not genetic (which means that they are about development, and not about heredity). 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 *in fine* lead to a better understanding of gene action.

Cytology

Cytology is the study of cells and cellular processes. It relies on imaging techniques (in particular 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. They might be very different from the raw images obtained by the microscope.¹⁶ Theorising in cytology (partially) consists in interpreting, and abstracting away from, such raw images, 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 behaviour (e.g., the behaviour of chromosomes during mitosis and meiosis).

Even when highly abstract, 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 objet. This will prove important in the following.

¹⁶ The notion of "raw image" calls for clarification. Images obtained by microscope (or other imaging techniques) are themselves the result of an important work of data processing and interpretation, which relies on theoretical constructs. But this is beyond the scope of my paper.

¹⁷ See Lynch (1988), Maienschein (1991).

What is wrong with Kitcher's characterisation of classical genetics?

Kitcher's notion of a pattern of reasoning leads him to what I consider a (partially) wrong characterisation of classical genetics. The typical reasoning associated with classical genetics consists, according to Kitcher, 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". These questions concerning the distribution of characteristics, to which the typically genetic patterns of reasoning are applied, are what Kitcher calls "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, 355)

Solving such a problem, Kitcher claims, consists first in advancing a "genetic hypothesis", which 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 with Kitcher's description of classical genetics' patterns of reasoning. Firstly, I think it misses a core aspect of theorising in genetics, which is embodied in the Mendelian

representational practice, as I have characterised it. To clarify this point, let me consider again the pedigree problems, as described by Kitcher. I guess it would not misrepresent Kitcher's views to claim that the pedigree problems, and the patterns of reasoning that serve to solve them, are embodied in the pedigree diagrams that one can find in any genetics textbook. Consider the diagram in figure 2, in light of Kitcher's description of pedigree problems as quoted above. The *data* about the distribution of phenotypes (and genotypes) are given by the form and colour of the symbols standing for the individuals (circles for females and squares for males). The *constraints* are given by the icons representing chromosomes, whose colours indicate their origin and the genes they carry (see caption). Here, the question is: how can a healthy female, whose father is healthy and mother is colour-blind, mating with a healthy male who does not carry any gene responsible for either colour-blindness or haemophilia, give birth to four male children with genetic diseases (more precisely: one haemophilic, one colour-blind, and two suffering from the two 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 figure 2, knowing the transmission patterns of the genes responsible for haemophilia and colour-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 the Mendelian reasoning, as I have characterised it above.

Moreover, and this leads me to my *second* worry with Kitcher's account, the cytological information conveyed by the icons representing chromosomes imposes *external* constraints on how to manipulate the diagrams. But it is not explanatory *qua* cytological. By this, I mean that 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 mechanical in character. As I have suggested, the typical representations associated with the cytological level are schematic. To be clear: I am not denying that the spatial disposition of the colours within the icons representing chromosomes in figure 2 is not explanatory. Nor am I suggesting that Kitcher is unaware of the fact that cytology provides a mechanical explanation of the genetic

¹⁸ One could even argue that the form of the pedigree diagrams is a remainder of previous conceptions of heredity. See 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. It meant, rigorously speaking, that a child did not inherit from his parents alone, but from the whole series of ancestors included in his lineage. 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. This is why they could be employed in both directions: from parental to filial generations, and conversely, from filial to parental generations. This difference can be illustrated by a comparison of the standard representations of 'generations' in biometry and in Mendelian genetics."

phenomena.¹⁹ But the kind of explanation they provide 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 somehow conflating the classical genetics' pattern of reasoning with the cytological level of explanation. In order to clarify what exactly is the explanatory import of cytology to genetics, one has to first distinguish the representational practices of Mendelism on the one hand, and of cytology on the other. 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 mechanical explanation of the genetic phenomenon called "partial linkage". This "conversion"²⁰, together with this mechanical 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 (or Mendelian) and the cytological levels into one unique theory. I will briefly recall what Morgan's theory consists of, before turning to the analysis of the technique of linkage mapping.

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

²⁰ About Morgan's intellectual evolution, see Carlson (1967), Allen (1978).

Morgan's theory of crossing-over

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

Genetic level: the phenomenon of partial linkage. From 1905 on, geneticists observed a phenomenon, which seems to contradict Mendel's second law. New data²¹ showed that some genes tend to be inherited together, without being always so. For instance, genes of *Lathyrus odoratus* responsible for the colour of the petals, on the one hand, and for the shape of the seeds, on the other, appear to be "partially linked" (or "coupled"). They are not randomly redistributed, contrary to what Mendel's second law would predict: partially linked genes are inherited together in more than 50% of the cases. But their association is not systematic either (they are inherited together in less than 100% of the cases). A few years later, Morgan (1910) observed that sex-linked genes (that is, genes which 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 mechanical explanation for the phenomenon of partial linkage. In 1909, the

²¹ The first case of partial linkage (or rather "coupling of traits") is reported by Bateson, Saunders, and Punnett (1905).

²² In 1891 already, 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 1910's.

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 1930's. Janssens called this putative physical exchange of segments of chromosomes "chiasmotypie". This cytological hypothesis prompted Morgan's proposition of a mechanical explanation of partial linkage.

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

Additivity and linearity. A crucial hypothesis underlies Morgan's model, namely that genes are *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 in which partially linked genes are redistributed separately) showed that such frequencies were *additive*. Additivity means 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.

Proportionality of recombination frequency and distance. Morgan drew an important conclusion from his mechanical model. He suggested that the recombination frequency of two genes (which was 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 from each other two genes are, the more a break between them is likely to occur, and hence the more they are likely to be redistributed separately.

Before turning to the presentation and analysis of the technique of linkage mapping, let me just insist that the explanation provided by the theory of crossing-over is a mechanical one, which is well expressed by the schematic drawing in figure 3. Hence, crossing-over does not belong to "pure Mendelism". It does not consist in applying combinatorial mathematics to discrete, operational units. However, one has to be careful and note that the schematic drawing in figure 3 *does not have* to 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, with not reference to chromosomes.²³ But such genetic level is not "purely Mendelian": the explanation is mechanical in kind, and the reasoning with which it is associated is not a purely combinatorial one, since it involves spatial reasoning.

Linkage mapping

In 1913, Alfred 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*. The table in figure 4 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 figure 5 displays the recombination frequencies by transforming them into visualisable distances. Genetic (or mapping)

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

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 (and the only one I will consider here) is that, for long distances (which stand for high recombination frequencies), some experiments (Morgan 1911b, 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, remember, was initially justified by the observation of additivity), Sturtevant hypothesised that there could be more than one crossing-over occurring on the same linkage group at the same time. As appears in figure 6, 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 from this hypothesis, Sturtevant choses 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 observed²⁵ recombination frequencies between the most distanced genes. This is obvious when one considers the table of the recombination frequencies (fig. 4) and its corresponding map (fig. 5). The table displays the

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, here, "observed" means inferred from the statistical data about the distribution of phenotypes, on the basis of already quite sophisticated genetic hypothesis.

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, that is, 37,6% of the cases. However, on the map, the distance between *B* and *M* is 57,6 (and not 37,6). This is 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 rather cytological ones? Are they diagrammatic (graphical) representations of statistical data (recombination frequencies), or rather schematic representations of chromosomes?

Maps as schematic representations of chromosomes

Prima facie, one might want to argue that maps are schematic representations, of the same type of the mechanical model of crossing over (fig. 3). True, they are not constructed the same way, but their function is nevertheless to spatially represent spatial relations, namely the relative location of the genes on the chromosomes. In fact, highlighting (aspects of) the structure of chromosomes is what linkage maps (which were also called "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 1930's, it became possible to map linkage maps onto cytological maps (obtained through microscope).

Hence, even if genetic distance (as already acknowledged by Sturtevant, 1913)²⁶ might not correspond exactly to physical distance (that is, if the metric is not conserved), at least the relative ordering of the genes is conserved (the topological relations). In that sense, they 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 display in a graphical way 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 visualise statistical data. They would contain no more information than the corresponding tables, but they would be much more efficient as computation-enhancers.

²⁶ Sturtevant (1913) indeed hypothesised 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.

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, with no spatial meaning. I will now show that this interpretation is not tenable.

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 mechanical model underlies the very mapping scheme. And, as we have seen, the mechanical model is not reducible to pure Mendelism, since it involves mechanical, and hence 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 have to be interpreted as standing for spatial relations) in order 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 use properly the map.²⁷

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

Analysing maps enables us to identify an essential aspect of classical genetics, namely the integration of a conception of heredity in terms of probabilities with its mechanical 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 (their rules of construction and interpretation) involves a theoretical hypothesis. As such, they are genuine theoretical representations. Linkage maps embody the articulation of the two levels (genetic and cytological) in a way 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

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

²⁸ Interestingly, Kitcher classifies linkage mapping as a "subtheory".

underlying theory – or pattern of reasoning – but rather the very *locus* of theorising.

FROM THE AGENTS' POINT OF VIEW: THE 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 the geneticists' efforts concentrated for at least two decades. In addition to yielding detailed knowledge of many genetic phenomena, the mapping enterprise provided geneticists with growing evidence in favour 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 1920's, 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, namely 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. Contrary to what logical empiricists, as well as Kuhn, suggest, they do not have clear-cut boundaries distinguishing them from other theories (both synchronically and 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 not satisfactory. However, my approach is more agent-centred than Kitcher's. Not only is it worth studying intra-theoretical variations, but one should not neglect either that theories do not exist independently from the minds of the agents, who develop, understand, and use them. To borrow Griesemer and Wimsatt's words, "theories require theorists, and abstract entities, entifiers" (1989, 87). 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. It could be defined as the

set of mental representations and inferential paths one makes use 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 via the same concepts, and the deductive order between their different principles are 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 being used, one does not get the same understanding of the phenomena, and of what classical mechanics says about them.³⁰

But even theories, which 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, on one's background

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 emphasising 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 (see Vorms 2010). In some cases, like in the case of classical mechanics, they are logically equivalent. In less developed sciences, like in classical genetics in the 1910's, they may be much less compatible.

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. However, according to what one is interested in, it is quite reasonable to abstract away from individuals, and to identify types of versions (see 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 with 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 in theories at early stages of their development.³² Classical genetics, as it is taught and used today, can be described as a stabilised and enriched version of the Morganian version. In the 1910's, 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, which is at the core of Morgan's version of genetics (and of classical genetics as we know it today), was still challenged in the 1910's, 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

³² The analysis in terms of version tends to both attenuate inter-theoretical differences (*contra* Kuhn's dramatic notion of incommensurability) and to emphasise intra-theoretical variations (which are completely obscured by Kuhn's holism).

the alternative between accepting and rejecting the chromosome theory. The three protagonists I will introduce here are all Mendelians, in the sense that they accept all the purely Mendelian hypotheses presented above.³³ However, beyond this agreement at the Mendelian level, they hold very different views of the physical basis of these phenomena. As will appear, the various hypotheses constituting Morgan's theory, which may seem non-dissociable in retrospect, were in fact susceptible to be held independently from each other.³⁴

Goldschmidt (1917): "Crossing-over without chiasmotypie". Richard Goldschmidt was one of the fiercest opponents to Morgan's theory. One reason for that was that he would reject one of 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 him, a theory of the gene had to say something about the mechanism of gene action.³⁶ The model he proposes is intended to make a room – and even to give a central place – to gene action.³⁷

Goldschmidt accepts two of the most fundamental hypotheses of Morgan's group, which were elsewhere controversial (even for "purer" Mendelians than Goldschmidt), namely the chromosome theory and the linearity hypothesis. However, he rejects chiasmotypie, as the hypothesis of a physical exchange of portions of chromosomes. Note that he does accept the hypothesis of a *genetic* crossing-over, that

³³ From that point of view, Goldschmidt is the more problematic. But, as we will see, he accepts 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 (forthcoming).

³⁵ See fn. 11.

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

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

is, of an orderly exchange of genes between homologous chromosomes. But he denies 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 reform 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 1950's), Bateson's criticism is thus internal to "orthodox" Mendelism. However, in 1916, he still rejects 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 in section 3.1), while rejecting the chromosome theory, and even the

representation of genes as material particles³⁸. 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 acceptance 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 hypothesis of chiasmata, the hypothesis of (simple) crossing-over, as well as 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 does accept), namely linearity. In fact, he rejects the whole mechanical model, of which linearity is a crucial element, and proposes a three-dimensional model of map, which is underlain by a chemical understanding of crossing-over.³⁹

Conclusions, and further thoughts, on the versions of genetics

My purpose here is not to assess the warranted or unwarranted character of the different versions I have presented. My point is only to show that professional geneticists could, in the 1910's, entertain different attitudes towards the various hypotheses constituting classical genetics, thus having different versions of

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

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

this theory. Even when accepting the chromosome theory (which was the case of both Goldschmidt and Castle), one could have 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 a 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 would like to suggest that such a study of the representational practices of geneticists might 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 articulate within a given scientific domain, might shed a new light on the issue of inter-theoretical relations. A study of representational practices, and of what I have called the scientists' own versions of a theory, is a good way to capture the conceptual articulation of a science, in a way formal approaches miss.

Even today, biologists might entertain different versions of classical genetics, according to their background, training, and the

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

context in which they use this theory. And I suggest that the question of whether classical genetics can be reduced to molecular biology might be a pragmatic question, which is worth approaching from the agents' point of view.⁴¹

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

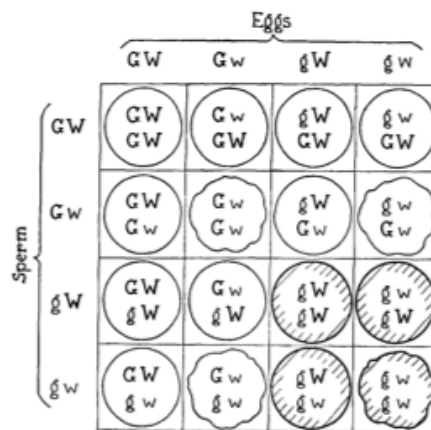


Fig. 1 Punnett square showing the expected distribution of genes among the germ cells for a cross involving two genes. It can also be considered as a diagrammatic expression of Mendel's second law. From Morgan (1928, 9).

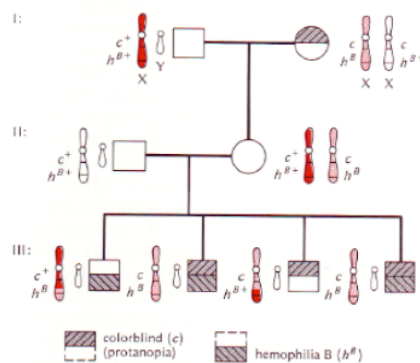


Fig. 2. Pedigree of a family segregating for the effects of two sex-linked genes, *colorblindness* (*c*) and *hemophilia* (*h^B*). The X chromosome linkages responsible for the phenotypes of generation III males are colored according to their origin: black (grandfather I-1) and grey (grandmother I-2).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

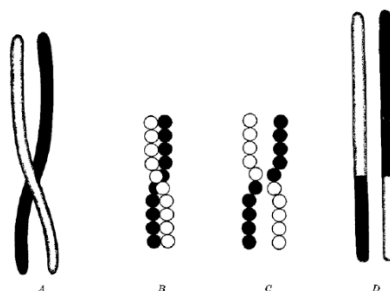


Fig. 3. The mechanical model of crossing-over. From Morgan *et al.* (1915, 60).

Factors concerned	Proportion of crossovers	Percent of crossovers
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, O) P	$\frac{471}{1584}$	29.7
(C, O) R	$\frac{2062}{6116}$	33.7
(C, O) M	$\frac{406}{898}$	45.2
PR	$\frac{17}{573}$	3.0
PM	$\frac{109}{405}$	26.9

Fig. 4. Table of the recombination frequencies (Sturtevant, 1913).

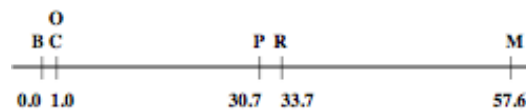


Fig. 5. Linkage map corresponding to the table in fig. 4. (Sturtevant, 1913)

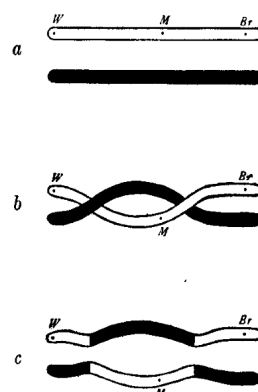


Fig. 6. Schematic representation of double crossing-over (Morgan et al., 1915).

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