Why the Anti-reductionist Consensus Won't Survive the Case of Classical Mendelian Genetics[1]

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Philosophers now treat the relationship between Classical Mendelian Genetics and molecular biology as a paradigm of nonreduction and this example is playing an increasingly prominent role in debates about the reducibility of theories ranging from macrosocial science to folk psychology. Patricia Churchland (1986), for example, draws an analogy between the alleged elimination of the "causal mainstay" of classical genetics and her view that today's psychological theory will be eliminated by neuroscience. Patricia Kitcher takes an autonomous rather than eliminativist view of the reported nonreduction in genetics and reasons that psychology will retain a similar autonomy from lower level sciences (1980 and 1982). Although Churchland and Kitcher offer different interpretations of the apparent failure of molecular biology to reduce classical genetics, they agree that this failure will help illuminate theoretical relations between psychology and lower level sciences. The appearance of the Mendelian example along side the usual ones from physics and chemistry marks a turning point in philosophy of science. Philosophers now look to biology in general, and the case of genetics in particular, for insights into the nature of theoretical relations. If I am correct, however, the current anti-reductionist consensus about genetics is mistaken and threatens to misguide our attempt to understand relations between other scientific theories. My aim is to defuse the arguments offered in support of the anti-reductionist consensus. Although the question of whether molecular biology is reducing Classical Mendelian Genetics will not be settled in any single paper, my critical analysis will reveal the signs of a significant theoretical reduction and uncover issues relevant to gaining a better understanding of what is now happening in genetics and of what we might expect to occur in other sciences.

The current consensus among philosophers is that, despite the appearances, Classical Mendelian Genetics (hereafter called CMG) is not being reduced to molecular biology, at least *not in the spirit of Nagel's (1961) postpositivist conception of theoretical reduction* [2] (Hull 1972 and 1974, Wimsatt 1976, Maull 1977, Darden and Maull 1977, Hooker 1981, Kitcher 1984, and Rosenberg 1985, but Schaffner 1969 and 1976, Ruse 1976, and Richardson 1979 and 1982 disagree). There are important differences within the consensus view, but according to the general anti-reductionist thrust, the relations between the levels of organization represented by the classical and molecular theories are too complex to be connected in the systematic way essential for

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a successful theoretical reduction. Anti-reductionists support this view by arguing that the gene concepts of the respective theories cannot be linked in an appropriate way. If the concepts cannot be linked, the reasoning goes, neither can the theoretical claims couched in terms of them. Hence, reduction will never be achieved. Before considering the anti-reductionists' arguments in greater detail, I will briefly describe the conception of reduction at issue and review CMG and the molecular theory of the gene.

1. Preliminaries

1a. The Spirit of Postpositivist Reduction

The consensus against reductionism in genetics has focused on Nagel's (1961) formal analysis of theoretical reduction. One of the two formal requirements set out by Nagel was that the laws of the reduced theory must be derivable from the laws and associated coordinating definitions of the reducing theory. The second formal requirement was that all terms of the reduced theory must either be contained within or be appropriately connected to the reducing theory by way of "additional assumptions." It is this condition of connectability that proponents of the consensus think cannot be satisfied in the case of genetics because of the contrasting gene concepts in the classical and molecular theories. A difficulty of relying on this formal conception is that it is couched within an account of theories discarded by most philosophers of biology.[3] In order to render the anti-reductionist consensus nontrivial, the spirit behind Nagel's conception of theoretical reduction will need to be separated from his formal analysis.

Nagel's discussion of nonformal conditions for reduction provides an opening for freeing his conception of theoretical reduction from his outmoded account of theories. In a section on these conditions, Nagel admitted, "The two formal conditions for reduction discussed in the previous section [connectability and derivability] do not suffice to distinguish trivial from noteworthy scientific achievements." (p. 358) He identified two sets of nonformal considerations to explain why the reduction of thermodynamics was a significant achievement. The first set concerned the establishment of new experimental laws that were in better agreement with a broader range of facts than were the original ones. The second set involved the discovery of surprising connections between various experimental laws.

Nagel's reliance on nonformal conditions indicates that he had an unarticulated notion of theoretical reduction which he failed to capture in his formal account. I would like to suggest, therefore, that his underlying conception of theoretical reduction can be separated from his formal treatment and in fact reformulated with respect to an updated account of theories. When I say that intertheoretical relations satisfy *the spirit of postpositivist reduction*, I simply mean that they would satisfy conditions set out in such a reformulation. Since postpositivists tended to view both explanation and reduction as special kinds of derivation, it is natural to suppose that their conception of theoretical reduction centered on the idea that reducing theories explain the success of reduced theories. Hence, the fundamental question for us is whether CMG is being explained by molecular biology According to the anti-reductionist consensus, CMG is not and will not be systematically explained by molecular biology.

1b. Classical Mendelian Genetics (CMG)

The consensus view concerns the reducibility of the theory of Classical Mendelian Genetics (CMG), not the reducibility of Mendel's theory. CMG was developed dur-

ing the first decades of this century, in large part by Thomas Hunt Morgan and his graduate students who worked on the genetics of *Drosophila*. According to the classical theory, patterns of inheritance can be explained by postulating the existence of genes. Differences in outward appearances (or phenotypes) of organisms are explained as the result of organisms inheriting different genes (or genotypes). Genes in *Drosophila* come in twos on corresponding pairs of linear chains. Each gene of a given pair has a fifty percent chance of having a copy distributed to a particular gamete (an egg or sperm). Genes located on different (nonpaired) chains assort independently from one another. Genes located on the same chain tend to be assorted together, but are sometimes distributed separately because paired chains occasionally exchange segments. The relative positions of genes can be determined by the frequency of such exchanges (on the assumption that genes located further apart from one another are assorted separately more often than genes located closer together). CMG concerns a wide range of gene behavior including, but not limited to, mutation, expression, interaction, recombination, and distribution.

The classical account of gene expression is complicated. In the simplest kind of system, two alleles with complete dominance, there are two contrasting phenotypic traits and two kinds of genes, one of which is dominant. Each trait is associated with one kind of gene and every organism has two genes. If an organism has two copies of the same gene, it exhibits the trait associated with the matching genes. If an organism has a pair of contrasting genes, it exhibits the characteristic associated with the dominant gene. This is but the simplest model of gene expression; classical geneticists have constructed models to represent systems of much greater complexity.

This abstract theory has a cytological interpretation. Gene chains are identified as chromosomes. Meiosis, the process in which chromosomes are distributed to gametes, offers an explanation of segregation and assortment. During the first division of this process, homologous chromosomes pair and then separate as two daughter cells are produced. The lack of complete linkage of genes located on the same chromosome is explained in terms of the crossing over (the exchange) of chromosomal segments.[4]

1c. The Molecular Theory

The molecular theory of the gene is based on the Watson and Crick Model of DNA. According to molecular theory, a gene is a relatively short segment of a DNA molecule, which consists of two very long chains of nucleotides held together by hydrogen bonds. The genetic information is encoded in the linear sequence of nucleotides making up individual genes. On the basis of this model and empirical studies, molecular biologists soon succeeded in explaining a number of important genetic phenomena including: gene replication; the multistep process by which the information encoded in structural genes eventually gets translated during polypeptide synthesis; and mechanisms of gene regulation. Polypeptides are the constituents of proteins and the regulation of biosynthetic pathways is for the most part directed by enzymatic proteins. Hence, the molecular explanation of how genes direct polypeptide synthesis offered an abstract picture of the biochemistry of gene expression.[5]

These successes led Kenneth Schaffner (1969) to conclude that CMG was being reduced to molecular biology. But enthusiasm for reductionism soon waned (at least among philosophers) when Michael Ruse (1971) and David Hull (1972) criticized Schaffner's specific account of the apparent reduction. Since then, these rather narrowly focused criticisms have been generalized into self-contained arguments against the general idea that CMG is being reduced. I now turn to these anti-reductionist objections.

2. Defusing the Anti-reductionist Objections

Arguments against the idea that CMG is being reduced (in the spirit of postpositivist reduction) fall into two general categories. The most prominent arguments are those aimed at showing that there are unbridgeable conceptual gaps between CMG and molecular biology. According to these arguments, subtle differences in the meaning of parallel terms from the classical and molecular theories obstruct reduction. The second category consists of arguments which conclude that molecular theory cannot deliver the explanatory power that reductionism requires. These arguments allegedly show that the explanatory relations between the classical and molecular theories are incomplete and that if a fuller explanation of Mendelian genetics is possible, it will come from a variety of biological fields, not just from molecular genetics as reductionism seems to imply. My critical analysis of these objections will not only show that the relationship between CMG and molecular biology is misunderstood, it will also reveal signs of a successful theoretical reduction in progress.

2a. The Unconnectability Objection

The unconnectability objection can be traced to David Hull's seminal works (1972 and 1974) where he proposed and defended the then heretical notion that Mendelian genetics is not being reduced by molecular biology, at least not according to Nagel's conception of theoretical reduction. The most rigorous formulation of this objection can be found in Alexander Rosenberg's provocative text (1985).

Rosenberg's opposition to reductionism in genetics rests on an alleged conceptual gap between the classical and molecular theories of genetics. He argues that relations between the gene concepts of the two theories are hopelessly complicated "many-many relations" that will forever frustrate any attempt to systematically connect the two theories. Rosenberg begins his analysis by pointing out that in CMG, genes are always identified by way of their phenotypic effects. Classical geneticists identified the gene for red eye color in *Drosophila*, for example, by following the distribution of red and white phenotypes in successive generations of a laboratory population. The reason CMG will never be reduced to molecular biology, Rosenberg argues, is that there is no manageable connection between the concept of a Mendelian phenotype and that of a molecular gene. The relation between them is complicated by the fact that scores of Mendelian phenotypes are potentially affected by an individual molecular gene and that a vast array of molecular genes are responsible for the production of any given Mendelian phenotype. Rosenberg explains the problem as follows:

> Suppose we have set out to explain the inheritance of normal red eye color in *Drosophila* over several generations. The pathway to red eye pigment production begins at many distinct molecular genes and proceeds through several alternative branched pathways. Some of the genes from which it begins are redundant, in that even if they are prevented from functioning the pigment will be produced. Others are interdependent, so that if one is blocked the other will not produce any product. Still others are "ambiguous" — belonging to several distinct pathways to different phenotypes. The pathway from the genes also contains redundant, ambiguous, and interdependent paths. If we give a biochemical characterization of the gene for red eye color either by appeal to the parts of its pathway of synthesis, or by appeal to the segments of DNA that it begins with, our molecular description of this gene will be too intricate to be of any practical explanatory upshot. (Rosenberg 1985, p. 101)

Rosenberg reasons that since Mendelian genes are identified through their phenotypes, and since the relation between molecular genes and Mendelian phenotypes is exceedingly complex, the connection between the molecular and Mendelian gene concepts must also be exceedingly complex. Hence, he concludes, CMG will forever remain beyond the reductive grasp of molecular biology. Rosenberg does not deny that molecular biologists will occasionally furnish individual accounts of various Mendelian phenomena on a piecemeal basis (as they have done with the genetics of sickle-cell anemia). He insists, however, that the unmanageably complex relations between the gene concepts of the two theories will prevent any systematic, reductive explanation of CMG in terms of molecular theory.

What Rosenberg's persuasive argument does not take into consideration is the relationship between the Mendelian gene and the Mendelian phenotype. According to the classical theory, one gene can affect different phenotypic traits and each phenotypic trait can be affected by different (nonallelic) genes. I will argue that the relationship between the Mendelian gene and the Mendelian phenotype exhibits the same complexity that Rosenberg discusses from the molecular perspective. My argument will not depend upon historical hindsight. Alfred H. Sturtevant, one of the architects of CMG, discussed the complex relation between the Mendelian gene and phenotype in his Ph.D. thesis (1916), which he wrote under T.H. Morgan. Ironically, he illustrated the point with the very same example that Rosenberg considers:

> The difference between normal red eyes and colorless (white) ones in *Drosophila* is due to a difference in a single gene. Yet red is a very complex color, requiring the interaction of at least five (and probably of very many more) different genes for its production. And these genes are quite independent, each chromosome bearing some of them. Moreover, eye-color is indirectly dependent upon a large number of other genes, such as those on which the life of the fly depends. We can then, in no sense identify a given gene with the red color of the eye, even though there is a single gene differentiating it from the colorless eye. So it is for all characters—as Wilson (1912) has put it '. . . the entire germinal complex is directly or indirectly involved in the production of every character.'[6]

The parallel between Sturtevant's and Rosenberg's accounts of the complex relationship between Mendelian phenotypes and Mendelian genes (Sturtevant's) and between Mendelian phenotypes and molecular genes (Rosenberg's) is striking. Both identify a web of relations too complex for the kind of explanation that Rosenberg seeks. My claim is that the molecular perspective offers a reductive interpretation of the complex picture offered by the classical theory. Our understanding of the biosynthetic pathways explains why there should be many-many relations between classical genes and Mendelian phenotypes.

The problem with Rosenberg's anti-reductionist line of reasoning is that it assumes that the existence of a particular gene can explain the presence of particular traits in an individual when in fact *genes can only explain* phenotypic *differences* and only *in given populations*. The presence of a gene for red eye-color on the X chromosome explains why the red-eyed *Drosophila* in a certain population have red eyes instead of white ones. The reason why classical geneticists found manageably simple relations between genes and phenotypic differences is because the genetic backgrounds against which particular genes produced differences were sufficiently uniform from one organism to another in the laboratory populations (of highly related individuals) under study. This can be explained from the molecular perspective in terms of a uniformity in relevant portions of the DNA, which in turn provided a uniform potential for bringing about certain results within the complex web of biosynthetic reactions.

Rosenberg's is but one of several lines of reasoning against the idea that the concepts of CMG and molecular biology can be systematically connected. Others focus on the problem of specifying a precise biochemical definition of a Mendelian gene. If the behavior of Mendelian genes can be explained in terms of molecular biology, some critics reason, then the central concepts of Mendelian theory must be defined in purely biochemical terms. The attempt to define a gene as a relatively short stretch of DNA won't do, the anti-reductionists point out, because not all relatively short stretches are genes. Furthermore, the attempt to define the gene in terms of a finer structure associated with a specific molecular mechanism will not work because of the diversity of molecular ways in which genes produce their effects. For example, Mendelian genes cannot be identified with reading frames (sections of DNA that are transcribed into RNA) because regulatory genes function without being transcribed. Such considerations reveal that a simple molecular definition of a Mendelian gene is not forthcoming.

The obvious response for the reductionist is simply to hold out for a disjunctive connection.[7] As we learn more about the molecular nature of Mendelian genes, we have discovered that they do not all function by way of the same mechanism. Some genes function by being transcribed into segments of RNA which code for polypeptides. Others function by regulating the transcription of neighboring genes. Furthermore, although all Mendelian genes are relatively short segments of DNA (or perhaps RNA), their finer structure varies with their role. Hence, any definition of Mendelian gene in terms of fine molecular structure will be disjunctive.

While I'm not prepared to insist that molecular biology already provides the means for completing a disjunctive definition *in terms of molecular structure*, I do think the elements for such a definition are falling in place. For the time being, I believe it suffices to point out that the behavior of specific Mendelian genes has been explained by identifying them with relatively short segments of DNA which function as units to influence the course of chemical reactions within a biochemical system. The fact that such a characterization has been sufficient for the development of molecular models of a variety of Mendelian phenomena leads me to think that the philosophers' attempt to formulate precise syntactical connections (in the form of explicit and detailed definitions) has been counterproductive. The focus on formal aspects of the postpositivist conception of reduction has led to too much haggling over syntax and not enough analysis of whether genetics exhibits the sort semantic and pragmatic features that motivated the formal account in the first place.

The Mendelian gene can be specified in molecular biology as a relatively short segment of DNA that functions as a biochemical unit. This specification provides an appropriate interpretation of the many-many relation between a Mendelian gene and phenotype. In addition, it provides a general statement of the precise connections that practicing molecular biologists have drawn between genes and phenotypes in individual cases. Most importantly, however, it has proven to be tremendously fruitful in research. For it has enabled molecular biologists to apply traditional strategies from classical genetics to uncover the biochemistry underlying many life processes. I conclude that the anti-reductionist thesis that there is some unbridgeable conceptual gap lurking between CMG and its molecular interpretation is wrong.

2b. The Explanatory Incompleteness Objection

The idea that CMG is being reduced to molecular biology has also been opposed on the grounds that molecular biology will never explain, and hence will never reduce, the classical theory of genetics. Since the postpositivist account of theoretical

reduction is centered on the idea that the reducing theory explains the reduced one, this complaint strikes at the very heart of the claim that CMG is being reduced in the spirit of postpositivism. Although this kind of objection can be found interspersed throughout the anti-reductionistic literature and seems to be an important element motivating the consensus against reductionism in genetics, it is seldom put forth as rigorously as the unconnectability objection. Nevertheless, I will reconstruct and defuse two separate arguments falling under this category.[8]

2bi.The Gory Details Argument

Anti-reductionists have argued that knowledge of the molecular makeup of genes does not enhance our understanding of their classical Mendelian behavior. For example, Philip Kitcher (1984), in his brilliant essay which marks the culmination of the anti-reductionist literature, argues that the assortment of genes is best understood at the cytological level: "The distribution of genes to gametes is to be explained, not by rehearsing the gory details of the reshuffling of the molecules, but through the observation that chromosomes are aligned in pairs just prior to the meiotic division, and that one chromosome from each matched pair is transmitted to each gamete." (Kitcher 1984, p. 370) He goes on to argue that the cytological pattern of explanation is objectively preferable because it can uniformly account for a wide range of cases that would look heterogeneous from a molecular perspective.

Kitcher does not describe a diversity of molecular processes responsible for the segregation of genes during meiosis. Instead, he offers an abstract account of the cytological explanation of gene distribution. According to his account, the distribution of genes is explained by identifying meiosis as belonging to the natural kind of "pair-separation processes." This natural kind of process, he says, is heterogeneous from the molecular perspective because different kinds of forces are responsible for bringing together and pulling apart different paired "entities". The separation of paired entities, he claims, "may occur because of the action of electromagnetic forces or even of nuclear forces; but it is easy to think of examples in which the separation is effected by the action of gravity." (Kitcher 1984, p. 350) Kitcher, I think, is *not* making the claim that some paired chromosomes are pulled apart by nuclear forces and others by the force of gravity (such a claim would be completely at odds with today's evidence). Rather, when he is discussing the multiple realizations of pair-separation processes he seems to be conceiving of a natural kind that includes processes quite unlike anything that occurs during meiosis. Hence, his reasoning only suggests that at some high level of abstraction, it is possible to draw an analogy between the process of meiosis and (yet to be specified) processes that have quite different molecular mechanisms. This is a far cry from showing that cytological theory offers a uniform explanation of a range of cases that would appear heterogeneous at the molecular level.

Although meiosis appears to be an unpromising candidate, there are other phenomena that are explained uniformly by CMG, but which are caused by a variety of molecular mechanisms. Phenomena of gene expression provide obvious examples. CMG, for instance, lumps together different kinds of gene expression under the category of dominance. This Mendelian category includes genes that code for structural proteins, genes which code for enzymes, and even regulatory genes. The molecular mechanisms by which these different kinds of genes are eventually expressed are quite different. Yet, when examining concrete cases where CMG offers a more uniform perspective, it is difficult to accept the anti-reductionist judgment that the shallow explanations of CMG are objectively preferable to the deeper accounts provided by molecular theory.

The idea that the uniformity provided by CMG gives it some sort of explanatory edge over the less uniform molecular account seems plausible only when our attention is called away from the actual biology. But even if uniformity of explanation did provide a potentially decisive advantage, there would be no reason to suppose that the uniformity represented by CMG could not also be captured within the molecular perspective through the familiar scientific practices of abstraction and idealization. The reductionists' view is not that the pictures offered by the reduced and reducing theory are the same, but that they can be connected by auxiliary assumptions such that the reducing theory stands in an explanatory relation to the reduced one. The fact that the reducing theory, when not accompanied by such auxiliary assumptions, more accurately represents the true diversity of mechanisms responsible for various processes should not be held against it.

Anti-reductionists, of course, do not deny the fact that molecular biology has greatly improved our understanding of genetics. Kitcher (1984), for example, provides an interesting discussion of various ways that molecular genetics has advanced our understanding. But they seem pessimistic when it comes to the issue of whether molecular theory will help us understand what (they think) are the essentials of CMG: the processes by which genes are distributed to gametes. The phenomena of independent assortment of nonlinked genes, it is claimed, depends only on the pairwise separation of chromosomes. The classical theory apparently tells us all we need to know: nonlinked genes are located on separate nonhomologous chromosomes and nonhomologous chromosomes segregate independently. The identification of genes as segments of a molecular double helix allegedly adds nothing to this account.

This anti-reductionist argument is problematic for two reasons: first, it becomes less plausible when we flesh it out within CMG (as opposed to Mendel's genetics) and second, it seems unduly pessimistic. To flesh the argument out within CMG, we need to consider not just the independent assortment of nonlinked genes, but also the distribution of linked ones. Recall that of central importance to the classical theory was the fact that linkage is incomplete because of the process of crossing over. At the cytological level, not much can be said about this process except that homologous chromosomes sometimes wrap around each other and swap segments during cellular division. Shortly after the double helical structure of the genetic chains was understood, however, molecular models of crossing over were proposed. The basic Holliday Model (Holliday 1964), illustrated in Figure 1, has been especially fruitful. Since then, laboratory studies have led to a more detailed, though admittedly tentative, biochemical understanding of the individual steps outlined in this model (see Potter and Dressler 1988). Our understanding of the exchange of segments between paired chains of genes is being greatly enhanced by our knowledge of the molecular structure of those chains. The biochemistry of genetic recombination is a tremendously active area of research and will bring our understanding of the classical Mendelian process of crossing over to the molecular level.

Anti-reductionists might respond by insisting that although the molecular perspective will contribute to our understanding of this bit of CMG, reductionism is a global thesis and requires that it contribute to all bits of the theory. "What about the independent assortment of non-linked genes", they might ask. "How do the molecular details improve the cytological explanation according to which nonlinked genes segregate independently because they are located on different chromosomes, which have been observed (via the microscope) to segregate independently?" This is the point at which I think the gory details objection becomes unduly pessimistic. Surely, the conjugation and separation of homologous chromosomes depends upon molecular mechanisms. While our understanding of why homologous chromosomes pair, why non-

homologous chromosomes don't pair, why separately paired chromosomes segregate independently, and so forth is not well developed, anti-reductionists haven't offered sufficient reason for thinking these questions won't eventually be answered. The answers to these questions will be given from the molecular perspective and will enhance our understanding of why non-linked genes assort independently.

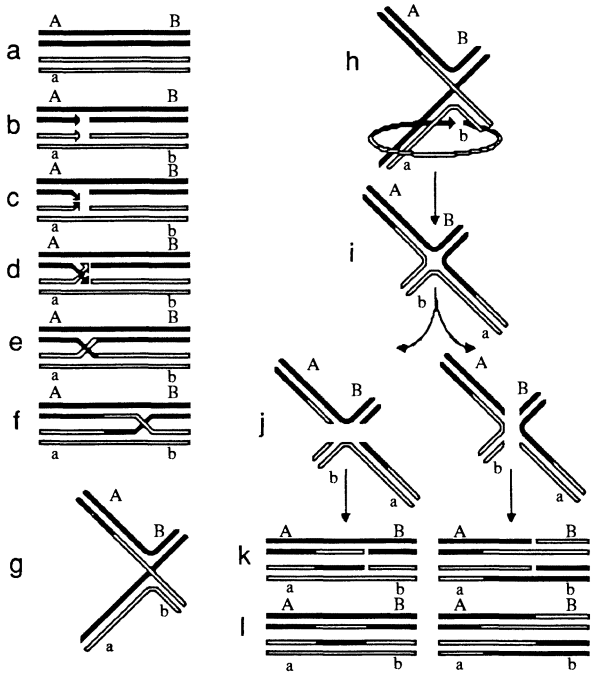

Figure 1. The Holliday Model for genetic recombination. (a) Two homologous double helices are aligned. (b) The two + or - strands are cut. (c) The free ends leave the complementary strands to which they have been hydrogen-bonded. (d) The free ends become associated with the complementary strands in the homologous double helix. (e) Ligation creates partially heteroduplex double helices. (f) Migration of the branch point occurs by continuing strand transfer by two polynuclotide chains involved in a crossover. (g) The Holliday structure shown in extended form. (h) The rotation of the structure shown in (g) can yield the form depicted in (i). Resolution of the structure shown in (i) can proceed in two ways, depending on the points of enzymatic cleavage, yielding the structures shown in (j), which can be depicted as shown in (k), and repaired to the forms shown in (1). Figure from Potter and Dressler (1979), p. 970. Explanation quoted from Suzuki et al (1986), p. 360.

Research in the general area of genetic recombination has already displayed signs identified by Nagel (see section 1. a.) as the distinguishing features of an important theoretical reduction. One sign is the discovery of surprising connections between seemingly unrelated processes. Recent biochemical research has revealed unexpected connections among the processes of recombination, replication, and repair (see Low 1988). Another sign of a significant reduction is the establishment of new experimental laws that are in better agreement with the facts. Recent lines of biochemical research hold promise for explaining why recombination is not entirely random and for helping us discover the finer patterns of genetic recombination (e.g. patterns of inter-

ference in closely spaced exchanges). Hence, even with respect to the Mendelian phenomena for which molecular explanations have tended to lag (i.e. transmission phenomena), the relation between CMG and molecular theory is beginning to exhibit characteristics corresponding to the two nonformal conditions set out by Nagel (1961) in his classic account of theoretical reduction.

The claim that the gory details of molecular biology do not enhance our understanding of key processes underlying CMG is quickly becoming outdated. There is no question that molecular theory has greatly improved our understanding of gene replication, expression, mutation, and recombination. Furthermore, it is just a matter of time before it accounts for the pair-wise coupling and separation of chromosomes during meiosis. Anti-reductionists need to justify their pessimism and explain why we should not expect molecular biology to continue on its path towards explaining CMG in accordance with the spirit of postpositivist reduction.

2bii. The Splintering Argument

The anti-reductionist literature contains hints of a way to dodge reductionism without denying the impending molecular explanation of CMG. Anti-reductionists might argue that even if a molecular explanation is imminent, the explanation will not come from molecular genetics; instead, it will come from a multitude of theories or fields of molecular biology.[9] Following Hull (1974), anti-reductionists have typically classified CMG as a theory of transmission genetics and molecular genetics as a theory of development. Presupposing this taxonomy, it might be argued that the classical and molecular theories of genetics explain different aspects of heredity. Hence, anti-reductionists might argue that even if transmission is explained at the molecular level, it will not be explained by molecular genetics.

It is tempting to dismiss such an anti-reductionist response as a case of sour grapes. "After volumes of denial", the reductionist might complain, "when the anti-reductionists are finally forced to admit that molecular biology systematically improves our understanding of classical genetics, they turn around and say that the explanation does not count because it comes from the wrong parts of molecular biology." While tempting, such a reply might miss the crux of the anti-reductionist complaint.

The issue at stake is whether molecular theory will offer a reasonably coherent explanation of CMG. The possible complaint is that molecular explanations will splinter into numerous fields. Instead of a case of one theory reducing another, one might envision a number of distinct theories explaining bits or pieces of the higher level theory. If unification is taken to be the hallmark of scientific explanation, the splintering of explanatory paths might appear to clinch the case against reductionism.

While such reasoning sounds plausible in the abstract, it depends on a number of slippery points in need of careful examination. The conceptual division between transmission and developmental genetics, for instance, though widely adopted in the philosophical literature and introductory chapters of genetic texts, has never been carefully analyzed and provides a weak footing for anti-reductionism. The chief reason offered in favor of this division, i.e. that CMG was developed on the basis of transmission studies, applies to molecular genetics as well (transmission studies have played and will continue to play an important role in the development of molecular genetics). Furthermore, the history of classical genetics supports the idea that the scope of CMG encompasses more than transmission. Debates about the Presence and Absence Hypothesis and the Position Effect, to take just two examples, clearly went beyond issues of transmission.

Perhaps the most serious obstacle to developing the splintering argument is it rests on the idea that there are significant divisions between theories of molecular biology when in fact molecular theory seems to have a diffuse structure. It is far from clear that molecular biology contains a separate theory of molecular genetics. Perhaps molecular biology consists of numerous molecular models of various phenomena, which are not organized into more discrete theories, but are loosely unified by their grounding in a set of common biochemical and biophysical principles. If this is indeed the case, the molecular explanation of CMG will not splinter into a number of different theories at the molecular level.

Developing the splintering objection would also entail substantiating premises about the structure of CMG. Anti-reductionists minimize the explanatory fit between CMG and molecular theory by de-emphasizing the parts of CMG that can be elegantly explained at the molecular level. The explanatory relations between CMG and molecular theory appear fractured, for example, when Kitcher characterizes the principle of gene replication as a "presupposition", as opposed to a "central law" of CMG (1984, p. 361). Such structural accounts of CMG depend on controversial philosophical views about the structure of scientific theories, which I believe are poorly motivated.[10] In any case, they should not be taken for granted.

The prospects for developing the splintering objection appear dim. The objection entails controversial philosophical views about the structure of theories and the nature of explanation as well as highly questionable assumptions about the taxonomy of genetics and the makeup of CMG and molecular biology.

3. Conclusion

The major objections to the view that CMG is being reduced by molecular biology have not withstood rigorous scrutiny. Perhaps the most surprising result is that the unconnectability objection was found to be so seriously flawed. In retrospect, however, the claimed unconnectability seems unlikely. After all, researchers are successfully identifying the molecular constituents and pinpointing the exact locations of genes contributing to many classically characterized traits (e.g. Duchenne Muscular Dystrophy). With sufficient experimental ingenuity, the molecular constituents and locations of the *Drosophila* genes mapped by Morgan et al could also be identified and pinpointed.[11] As a matter of fact, researchers have just determined the molecular identity of the first Mendelian gene ever discovered, the gene for wrinkled-seed character in pea plants (Bhattacharyya et al 1990). While molecular biologists have had to conquer many obstacles in their search for the molecular identity of Mendelian genes, the alleged conceptual gap between gene concepts was not one of them.

My examination of the arguments aimed at showing that molecular theory will never explain (and hence never reduce) classical genetics provides a partial explanation of why philosophers and molecular biologists disagree about the reduction of Mendelian genetics. In each case, the anti-reductionist arguments were based on admittedly brilliant philosophical analyses that appeared plausible in the abstract. But, when scrutinized with respect to the details of the actual science, the arguments were found to rest on undue pessimism, on implausible judgments of comparative explanatory value, and on highly questionable assumptions about the structure of CMG and molecular biology. Practicing geneticists believe that the classical theory can be systematically explained at the molecular level, I suggest, because they have a firm grasp of the explanatory power and structure of molecular biology.

Practicing geneticists are also well aware of the achievements that I have identified as signs of a significant theoretical reduction. These achievements include the discovery of unexpected molecular connections among several different genetic processes and promises to improve the precision of our generalizations in genetics. In addition, genetics has provided tremendously fruitful strategies for biochemical research. While such signs indicate that a theoretical reduction is in the making, I have not offered an account of that reduction. I will conclude by briefly anticipating some of the philosophical work that lies ahead.

The main philosophical task will involve reformulating the postpositivist conception of theoretical reduction. Reformulating the postpositivist conception will require an explicit account of explanation as well as an updated account of theories.[12] The anti-reductionist arguments are tacitly or explicitly linked to accounts of explanation that place a very high premium on unification. This premium is associated with the idea that theoretical reduction requires unification. While the postpositivist view assumes that reduction is accompanied by unification, it is not clear whether the view takes unification to be an essential ingredient or just an expected dividend. Unification is essential for reduction just in case it is essential for explanation. If, as I have hinted, the unificationist criterion for explanation is implausible when invoked within the nitty-gritty details of genetics, there will be strong incentive to treat unification as a valued bonus, rather than a necessary requirement in the reformulated account of theoretical reduction. The unificationist accounts of explanation and reduction, I suggest, should be assessed from the perspective of molecular biology rather than the other way around.

The reformulation of theoretical reduction will have to be carried out in terms of an explicit account of theories. Most philosophers of biology accept something akin to the semantic view, a view which holds some promise for helping us capture the spirit of postpositivist reduction. One advantage of the semantic view is that it can reportedly help us avoid the logical empiricists' preoccupation with syntactical matters, a preoccupation which plays a role in some anti-reductionist analyses. Another advantage is that its picture of piecemeal theorizing should enable us to formulate not just a conception of completed reduction, but also the conception of reduction in progress. A shortcoming of the original formulation is that it does not offer a dynamic picture of theoretical reduction. This is especially problematic with respect to genetics where the reduction is still being worked out.

Different philosophical views on the structure of theories and the nature of explanation will undoubtedly lead to different conceptions of theoretical reduction and different pictures of the theoretical relations between classical genetics and molecular biology. These, in turn, can be assessed on the basis of how well they illuminate the actual science. The question of whether CMG is being reduced deserves to be reconsidered, not just because we have good reason to suspect that the anti-reductionistic consensus is wrong, but also because it provides the opportunity to advance philosophical debates about the structure of theories and the nature of scientific explanation and theoretical reduction.

Notes

[1]I thank Bob Knox for stimulating discussions which influenced my thinking on this subject. Earlier versions of this paper were presented at the University of Pittsburgh and Indiana University where audiences provided helpful feedback. The

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[2] "Not in the spirit of the postpositivist conception of theoretical reduction" is emphasized because some critics acknowledge that there are important theoretical relations between CMG and molecular biology, but insist that these relations cannot be understood in terms of the postpositivist conception of reduction. Wimsatt (1976), for example, attacks Nagel's conception and offers his own functional account of the activities related to "explanatory reduction." The more recent literature (e.g. Kitcher 1984 and Rosenberg 1985), which heavily borrows from the earlier works, is less ambiguous and clearly denies that molecular biology will ever reduce CMG in any significant sense of "reduction". I suspect that some of the earlier papers will appear less anti-reductionist when anti-reductionism is no longer taken for granted.

[3]Rosenberg, however, clings to the old account of theories. See Waters (1990).

[4]A good primary source of CMG is Morgan (1928). Carlson (1966) offers a provocative historical account and Hull (1974) gives a succinct and clear presentation of the theory.

[5]More detailed accounts of molecular theory can be found in practically any contemporary genetics text.

[6]Quoted from Carlson 1988, p. 69.

[7]Although this is the obvious response, another is available. For, as some anti-reductionists have admitted (Hull 1974, Kitcher 1984), the derivation (or explanation) of the principles of CMG does not require the formulation of a set of necessary and sufficient molecular conditions for the terms of CMG. Necessary conditions would suffice.

[8]The basic reasoning behind the first argument and hints of the second can be found in Kitcher (1984). Elements of them can also be found in Hull (1974), Wimsatt (1976), and perhaps Maull (1977), and Darden and Maull (1977). A third argument can be constructed on the basis of Beatty's point that molecular biology will never completely explain CMG because it will never be able to reduce the evolutionary explanation of Mendelian principles (see 1983). Beatty has developed an important point about the limits of molecular reductionism in biology and it would be decisive if I was arguing that all of biology can be reduced to a science of proximate causes. But my interest concerns the question of whether the proximate theory of Mendelian genetics will be reduced by the proximate theory of molecular biology. Evolutionary questions about Mendelian phenomena will not go away upon achievement of this reduction; they will simply be reduced to evolutionary questions about molecular phenomena.

[9]Kitcher (1984), for example, suggests something along this line when he writes that "molecular genetics on its own, cannot deliver the goods" (p.366) and that "it would be folly to suggest that the [explanatory] extension is provided by molecular genetics alone." (p. 368)

[10]Kitcher's (1984, p. 361) defense of this characterization is enmeshed within his distinctive account of the structure of scientific theories. I have challenged the central motivation for his radical departure from the traditional view that theories contain law-like claims (Waters 1989 and forthcoming). If I'm correct, the principle of gene replication should be viewed as a law of CMG.

[11]*Drosophila* researchers have shifted their attention to genes that play significant roles in developmental processes. So, the search is mainly for genes with developmental significance.

[12]The depth of Kitcher's (1984) account of this case stems from the fact that he has taken into account these underlying philosophical issues. But I believe the denial of the unconnectability objection, a more explicit account of molecular biology, and different philosophical views on structure of theories and the nature of explanation will lead to a different and more illuminating picture of the situation.

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