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1953 AND ALL THAT. A TALE OF TWO SCIENCES*

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> "Must we geneticists become bacteriologists, physiological chemists and physicists, simultaneously with being zoologists and botanists? Let us hope so."
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> —H. J. Muller, 1922[1]

1. The Problem

Toward the end of their paper announcing the molecular structure of DNA, James Watson and Francis Crick remark, somewhat laconically, that their proposed structure might illuminate some central questions of genetics.[2] Thirty years have passed since Watson and Crick published their famous discovery. Molecular biology has indeed transformed our understanding of heredity. The recognition of the structure of DNA, the understanding of gene replication, transcription and translation, the cracking of the genetic code, the study of gene regulation, these and other breakthroughs have combined to answer many of the questions that

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[1] "Variation due to change in the individual gene," reprinted in J. A. Peters ed., *Classic Papers in Genetics* (Englewood Cliffs, N.J.: Prentice-Hall, 1959), pp. 104–116. Citation from p. 115.

[2] "Molecular Structure of Nucleic Acids," *Nature* 171 (1953), pp. 737–738; reprinted in Peters, op. cit., pp. 241–243. Watson and Crick amplified their suggestion in "Genetic Implications of the Structure of Deoxyribonucleic Acid" *Nature* 171 (1953), pp. 934–937.

baffled classical geneticists. Muller's hope—expressed in the early days of classical genetics—has been amply fulfilled.

Yet the success of molecular biology and the transformation of classical genetics into molecular genetics bequeath a philosophical problem. There are two recent theories which have addressed the phenomena of heredity. One, *classical genetics,* stemming from the studies of T. H. Morgan, his colleagues and students, is the successful outgrowth of the Mendelian theory of heredity rediscovered at the beginning of this century. The other, *molecular genetics,* descends from the work of Watson and Crick. What is the relationship between these two theories? How does the molecular theory illuminate the classical theory? How exactly has Muller's hope been fulfilled?

There used to be a popular philosophical answer to the problem posed in these three connected questions: classical genetics has been reduced to molecular genetics. Philosophers of biology inherited the notion of reduction from general discussions in philosophy of science, discussions which usually center on examples from physics. Unfortunately attempts to apply this notion in the case of genetics have been vulnerable to cogent criticism. Even after considerable tinkering with the concept of reduction, one cannot claim that classical genetics has been (or is being) reduced to molecular genetics.[3] However, the antireductionist point is typically nega-

[3]The most sophisticated attempts to work out a defensible version of reductionism occur in articles by Kenneth Schaffner. See, in particular, "Approaches to Reduction," *Philosophy of Science* 34 (1967), pp. 137–147; "The Watson-Crick Model and Reductionism," *British Journal for the Philosophy of Science* 20 (1969), pp. 325–348; "The Peripherality of Reductionism in the Development of Molecular Biology," *Journal of the History of Biology* 7 (1974), pp. 111–139; and "Reductionism in Biology: Prospects and Problems," R. S. Cohen *et al.* eds., *PSA 1974,* (Boston: D. Reidel, 1976), pp. 613–632. See also Michael Ruse, "Reduction, Replacement, and Molecular Biology," *Dialectica* 25 (1971), pp. 38–72; and William K. Goosens, "Reduction by Molecular Genetics," *Philosophy of Science* 45 (1978), pp. 78–95. A variety of antireductionist points are made in David Hull, "Reduction in Genetics—Biology or Philosophy?" *Philosophy of Science* 39 (1972), pp. 491–499; and Chapter 1 of *Philosophy of Biological Science* (Englewood Cliffs, N.J.: Prentice-Hall, 1974); in Steven Orla Kimbrough, "On the Reduction of Genetics to Molecular Biology," *Philosophy of Science* 46 (1979), pp. 389–406 and in Ernst Mayr, *The Growth of Biological Thought* (Cambridge, Mass.: Harvard University Press, 1982), pp. 59–63.

tive.[4] It denies the adequacy of a particular solution to the problem of characterizing the relation between classical genetics and molecular genetics. It does not offer an alternative solution.

My aim in this paper is to offer a different perspective on intertheoretic relations. The plan is to invert the usual strategy. Instead of trying to force the case of genetics into a mold, which is alleged to capture important features of examples in physics, or resting content with denying that the material can be forced, I shall try to arrive at a view of the theories involved and the relations between them that will account for the almost universal idea that molecular biology has done something important for classical genetics. In so doing, I hope to shed some light on the general questions of the structure of scientific theories and the relations which may hold between successive theories. Since my positive account presupposes that something is wrong with the reductionist treatment of the case of genetics, I shall begin with a diagnosis of the foibles of reductionism.

2. What's Wrong with Reductionism?

Ernest Nagel's classic treatment of reduction[5] can be simplified for our purposes. Scientific theories are regarded as sets of state-

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[4]Typically, though not invariably. In a suggestive essay, "Reductive Explanation: A Functional Account," (R. S. Cohen *et al.* op. cit. pp. 671–710), William Wimsatt offers a number of interesting ideas about intertheoretic relations and the case of genetics. Also provocative are Nancy Maull's "Unifying Science Without Reduction," *Studies in the History and Philosophy of Science* 8 (1977), pp. 143–171; and Lindley Darden and Nancy Maull, "Interfield Theories," *Philosophy of Science* 44 (1977), pp. 43–64. My chief complaint about the works I have cited is that unexplained technical notions—"mechanisms," "levels," "domain," "field," "theory"—are invoked (sometimes in apparently inconsistent ways), so that no precise answer to the philosophical problem posed in the text is ever given. Nevertheless, I hope that the discussion of the later sections of this paper will help to articulate more fully some of the genuine insights of these authors, especially those contained in Wimsatt's rich essay.

[5]E. Nagel, *The Structure of Science* (New York: Harcourt Brace, 1961), Chapter 11. A simplified presentation can be found in Chapter 8 of C. G. Hempel, *Philosophy of Natural Science* (Englewood Cliffs, N.J.: Prentice-Hall, 1966).

ments.[6] To reduce a theory T_2 to a theory T_1, is to deduce the statements of T_2 from the statements of T_1. If there are nonlogical expressions which appear in the statements of T_2, but do not appear in the statements of T_1, then we are allowed to supplement the statements of T_1 with some extra premises connecting the vocabulary of T_1 with the distinctive vocabulary of T_2 (so-called *bridge principles*). Intertheoretic reduction is taken to be important because the statements which are deduced from the reducing theory are supposed to be explained by this deduction.

Yet, as everyone who has struggled with the paradigm cases from physics knows all too well, the reductions of Galileo's law to Newtonian mechanics and of the ideal gas laws to the kinetic theory do not exactly fit Nagel's model. Study of these examples suggests that, to reduce a theory T_2 to a theory T_1, it suffices to deduce the laws of T_2 from a suitably modified version of T_1, possibly augmented with appropriate extra premises.[7] Plainly, this sufficient condition is dangerously vague.[8] I shall tolerate its vagueness, pro-

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[6]Quite evidently, this is a weak version of what was once the "received view" of scientific theories, articulated in the works of Nagel and Hempel cited in the previous note. A sustained presentation and critique of the view is given in the Introduction to F. Suppe ed., *The Structure of Scientific Theories* (Urbana: University of Illinois Press, 1973). The fact that the standard model of reduction presupposes the thesis that theories are reasonably regarded as sets of statements has been noted by Clark Glymour, "On Some Patterns of Reduction," *Philosophy of Science* 36 (1969), pp. 340–353, 342; and by Jerry Fodor (*The Language of Thought*, New York: Crowell, 1975, p. 11, footnote 10). Glymour endorses the thesis; Fodor is skeptical about it.

[7]Philosophers often suggest that, in reduction, one derives *corrected* laws of the reduced theory from an *unmodified* reducing theory. But this is not the way things go in the paradigm cases: one doesn't correct Galileo's law by using Newtonian mechanics; instead, one neglects "insignificant terms" in the Newtonian equation of motion for a body falling under the influence of gravity; similarly, in deriving the Boyle-Charles law from kinetic theory (or statistical mechanics), it is standard to make idealizing assumptions about molecules, and so obtain the exact version of the Boyle-Charles law; subsequently, corrected versions are generated by "subtracting" the idealizing procedures. Although he usually views reduction as deriving a corrected version of the reduced theory, Schaffner notes that reduction might sometimes proceed by modifying the reducing theory ("Approaches to Reduction," p. 138; "The Watson-Crick Model and Reductionism" p. 322). In fact, the point was already made by Nagel, op. cit.

[8]In part, because modification might produce an inconsistent theory that would permit the derivation of anything. In part, because of the

posing that we understand the issue of reduction in genetics by using the examples from physics as paradigms of what "suitable modifications" and "appropriate extra premises" are like. Reductionists claim that the relation between classical genetics and molecular biology is sufficiently similar to the intertheoretical relations exemplified in the examples from physics to count as the same type of thing: to wit, as intertheoretical reduction.

It may seem that the reductionist thesis has now become so amorphous that it will be immune to refutation. But this is incorrect. Even when we have amended the classical model of reduction so that it can accommodate the examples that originally motivated it, the reductionist claim about genetics requires us to accept three theses:

(R1) Classical genetics contains general laws about the transmission of genes which can serve as the conclusions of reductive derivations.

(R2) The distinctive vocabulary of classical genetics (predicates like '① is a gene', '① is dominant with respect to ②') can be linked to the vocabulary of molecular biology by bridge principles.

(R3) A derivation of general principles about the transmission of genes from principles of molecular biology would explain why the laws of gene transmission hold (to the extent that they do).

I shall argue that each of the theses is false, offering this as my diagnosis of the ills of reductionism.

Before offering my criticisms, it may help to explain why reductionism presupposes (R1)–(R3). If the relation between classical genetics and molecular biology is to be like that between the theory of ideal gases and the kinetic theory (say), then we are going to

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traditionally vexing problem of the proper form for bridge principles in heterogeneous reductions in physics. The former problem is discussed in Glymour, op. cit., p. 352 and in Dudley Shapere, "Notes towards a Post-Positivistic Interpretation of Science" (P. Achinstein and S. Barker, eds. *The Legacy of Logical Positivism,* Baltimore: Johns Hopkins University Press, 1971). For discussion of the latter issue, see Larry Sklar, "Types of Inter-Theoretic Reduction," *British Journal for the Philosophy of Science* 18 (1967), pp. 109–124; Robert Causey, "Attribute Identities in Microreductions," *Journal of Philosophy* 69 (1972), pp. 407–422; and Berent Enc, "Identity Statements and Micro-reductions," *Journal of Philosophy* 73 (1976), pp. 285–306. The concerns that I shall raise are orthogonal to these familiar areas of dispute.

need to find general principles, identifiable as the central laws of classical genetics, which can serve as the conclusions of reductive derivations. (We need counterparts for the Boyle-Charles law.) These will be general principles about genes, and, because classical genetics seems to be a theory about the inheritance of characteristics, the only likely candidates are laws describing the transmission of genes between generations. [So reductionism leads to (R1).] If we are to derive such laws from molecular biology, then there must be bridge principles connecting the distinctive vocabulary figuring in the laws of gene transmission (presumably expressions like '① is a gene', and perhaps '① is dominant with respect to ②') with the vocabulary of molecular biology. [Hence (R2).] Finally, if the derivations are to achieve the goal of intertheoretical reduction then they must explain the laws of gene transmission. [(R3).]

Philosophers often identify theories as small sets of general laws. However, in the case of classical genetics, the identification is difficult and those who debate the reducibility of classical genetics to molecular biology often proceed differently. David Hull uses a characterization drawn from Dobzhansky: classical genetics is "concerned with gene differences; the operation employed to discover a gene is hybridization: parents differing in some trait are crossed and the distribution of the trait in hybrid progeny is observed."[9] This is not unusual in discussions of reduction in genetics. It is much easier to identify classical genetics by referring to the subject matter and to the methods of investigation, than it is to provide a few sentences that encapsulate the content of the theory.

Why is this? Because when we read the major papers of the great classical geneticists or when we read the textbooks in which their work is summarized, we find it hard to pick out *any* laws about genes. These documents are full of informative statements. Together, they tell us an enormous amount about the chromosomal arrangement of particular genes in particular organisms, about the

[9]Hull, *Philosophy of Biological Science,* p. 23, adapted from Theodosius Dobzhansky, *Genetics of the Evolutionary Process* (New York: Columbia University Press, 1970), p. 167. Similarly molecular genetics is said to have the task of "discovering how molecularly characterized genes produce proteins which in turn combine to form gross phenotypic traits" (Hull *ibid.;* see also James D. Watson, *Molecular Biology of the Gene,* Menlo Park, Ca., W. A. Benjamin, 1976, p. 54).

effect on the phenotype of various mutations, about frequencies of recombination, and so forth.[10] In some cases, we might explain the absence of formulations of general laws about genes (and even of reference to such laws) by suggesting that these things are common knowledge. Yet that hardly accounts for the nature of the textbooks or of the papers that forged the tools of classical genetics.

If we look back to the pre-Morgan era, we do find two general statements about genes, namely Mendel's Laws (or "Rules"). Mendel's second law states that, in a diploid organism which produces haploid gametes, genes at differenct loci will be transmitted independently; so, for example, if *A, a* and *B, b* are pairs of alleles at different loci, and if an organism is heterozygous at both loci, then the probabilities that a gamete will receive any of the four possible genetic combinations, *AB, Ab, aB, ab,* are all equal.[11] Once it was recognized that genes are (mostly) chromosomal segments, (as biologists discovered soon after the rediscovery of Mendel's laws), we understand that the law will not hold in general: alleles which are on the same chromosome (or, more exactly, close together on the same chromosome) will tend to be transmitted together because (ignoring recombination)[12] one member of each homologous pair is distributed to a gamete.[13]

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[10]The phenotype/genotype distinction was introduced to differentiate the observable characteristics of an organism from the underlying genetic factors. In subsequent discussions the notion of phenotype has been extended to include properties which are not readily observable (for example, the capacity of an organism to metabolize a particular amino acid). The expansion of the concept of phenotype is discussed in my paper "Genes," *British Journal for the Philosophy of Science* 33 (1982), pp. 337–359.

[11]"A *locus* is the place on a chromosome occupied by a gene. Different genes which can occur at the same locus are said to be *alleles.* In diploid organisms, chromosomes line up in pairs just before the meiotic division that gives rise to gametes. The matched pairs are pairs of *homologous chromosomes.* If different alleles occur at corresponding loci on a pair of homologous chromosomes, the organism is said to be *heterozygous* at these loci.

[12]*Recombination* is the process (which occurs before meiotic division) in which a chromosome exchanges material with the chromosome homologous with it. Alleles which occur on one chromosome may thus be transferred to the other chromosome, so that new genetic combinations can arise.

[13]Other central Mendelian claims also turn out to be false. The Mendelian principle that if an organism is heterozygous at a locus then the probabilities of either allele being transmitted to a gamete are equal falls

Now it might seem that this is not very important. We could surely find a correct substitute for Mendel's second law by restricting the law so that it only talks about genes on nonhomologous chromosomes. Unfortunately, this will not quite do. There can be interference with normal cytological processes so that segregation of nonhomologous chromosomes need not be independent.[14] However, my complaint about Mendel's second law is not that it is incorrect: many sciences use laws that are clearly recognized as approximations. Mendel's second law, amended or unamended, simply becomes irrelevant to subsequent research in classical genetics.

We envisaged amending Mendel's second law by using elementary principles of cytology, together with the identification of genes as chromosomal segments, to correct what was faulty in the unamended law. It is the fact that the application is so easy and that it can be carried out far more generally that makes the "law" it generates irrelevant. We can understand the transmission of genes by analyzing the cases that interest us from a cytological perspective—by proceeding from "first principles," as it were. Moreover, we can adopt this approach whether the organism is haploid, diploid or polyploid, whether it reproduces sexually or asexually, whether the genes with which we are concerned are or are not on homologous

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afoul of cases of meiotic drive. (A notorious example is the *t*-allele in the house mouse, which is transmitted to 95% of the sperm of males who are heterozygous for it and the wild-type allele; see R. C. Lewontin and L. C. Dunn, "The Evolutionary Dynamics of a Polymorphism in the House Mouse," *Genetics* 45 (1960), pp. 705–722. Even the idea that genes are transmitted across the generations, unaffected by their presence in intermediate organisms, must be given up once we recognize that intra-allelic recombination can occur.

[14]To the best of my knowledge, the mechanisms of this interference are not well understood. For a brief discussion, see J. Sybenga, *General Cytogenetics* (North-Holland, 1972), pp. 313–314. In this paper, I shall use "segregation distortion" to refer to cases in which there is a propensity for nonhomologous chromosomes to assort together. "Meiotic drive" will refer to examples in which one member of a pair of homologous chromosomes has a greater probability of being transmitted to a gamete. The literature in genetics exhibits some variation in the use of these terms. Let me note explicitly that, on these construals, both segregation distortion and meiotic drive will be different from *nondisjunction,* the process in which a chromosome together with the whole (or a part) of the homologous chromosome is transmitted to a gamete.

chromosomes, whether or not there is distortion of independent chromosomal segregation at meiosis. Cytology not only teaches us that the second law is false; it also tells us how to tackle the problem at which the second law was directed (the problem of determining frequencies for pairs of genes in gametes). The amended second law is a restricted statement of results obtainable using a general technique. What figures largely in genetics after Morgan is the technique, and this is hardly surprising when we realize that one of the major research problems of classical genetics has been the problem of discovering the distribution of genes *on the same chromosome,* a problem which is beyond the scope of the amended law.

Let us now turn from (R1) to (R2), assuming, contrary to what has just been argued, that we can identify the content of classical genetics with general principles about gene transmission. (Let us even suppose, for the sake of concreteness, that the principles in question are Mendel's laws—amended in whatever way the reductionist prefers.) To derive these principles from molecular biology, we need a bridge principle. I shall consider first statements of the form

(*) (x) (x is a gene $\leftrightarrow$ Mx)

where 'Mx' is an open sentence (possibly complex) in the language of molecular biology. Molecular biologists do not offer any appropriate statement. Nor do they seem interested in providing one. I claim that no appropriate bridge principle can be found.

Most genes are segments of DNA. (There are some organisms—viruses—whose genetic material is RNA; I shall henceforth ignore them.) Thanks to Watson and Crick, we know the molecular structure of DNA. Hence the problem of providing a statement of the above form becomes that of saying, in molecular terms, which segments of DNA count as genes.

Genes come in different sizes, and, for any given size, we can find segments of DNA of that size that are not genes. Therefore genes cannot be identified as segments of DNA containing a particular number of nucleotide pairs. Nor will it do to give a molecular characterization of those codons (triplets of nucleotides) that initiate and terminate transcription, and take a gene to be a segment of DNA between successive initiating and terminating codons. In the

first place, mutation might produce a *single* allele containing within it codons for stopping and restarting transcription.[15] Secondly, and much more importantly, the criterion is not general since not every gene is transcribed on mRNA.

The latter point is worth developing. Molecular geneticists recognize regulatory genes as well as structural genes. To cite a classic example, the operator region in the *lac* operon of *E. coli* serves as a site for the attachment of protein molecules, thereby inhibiting transcription of mRNA and regulating enzyme production.[16]

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[15]This point raises some interesting issues. It is common practice in genetics to count a segment of DNA as a single gene if it was produced by mutation from a gene. Thus many mutant alleles are viewed as DNA segments in which modification of the sequence of bases has halted transcription too soon, with the result that the gene product is truncated and nonfunctional. My envisaged case simply assumes that a second mutation occurs further down the segment so that transcription starts and stops in two places, generating two useless gene products. The historical connection with the original allele serves to identify the segment as one gene.

Conversely, where there is no historical connection to any organism, one may have qualms about counting a DNA segment as a gene. Suppose that, in some region of space, a quirk of nature brings together the constituent atoms for the white eye mutant in *Drosophila melanogaster,* and that the atoms become arranged in the right way. Do we have here a *Drosophila* gene? If the right answer is "No" then it would seem that a molecular structure only counts as a gene given an appropriate history. I hasten to add that "appropriate histories" need not simply involve the usual biological ways in which organisms transmit, replicate and modify genes: one can reasonably hope to synthesize genes in the laboratory. The case seems analogous to questions that arise about personal identity. If a person's psychological features are replicated by a process that sets up the "right sort of causal connection" between person and product, then we are tempted to count the product as the surviving person. Similarly, if a molecular structure is generated in a way that sets up "the right sort of causal connection" between the structure and some prior gene then it counts as a gene. In both cases, causal connections of "the right sort" may be set up in everyday biological ways and by means of deliberate attempts to replicate a prior structure.

[16]So called *structural genes* direct the formation of proteins by coding for RNA molecules. They are "transcribed" to produce *messenger* RNA (mRNA) which serves as a more immediate "blueprint" for the construction of the protein. Transcription is started and stopped through the action of regulatory genes. In the simplest regulatory system (that of the *lac* operon) an area adjacent to the structural gene serves as a "dumping ground" for a molecule. When concentration of the protein product becomes too high, the molecule attaches to this site and transcription halts; when more protein is required, the cell produces a molecule that removes

Moreover, it is becoming increasingly obvious that genes are not always transcribed, but play a variety of roles in the economy of the cell.[17]

At this point, the reductionist may try to produce a bridge principle by brute force. Trivially, there are only a finite number of terrestrial organisms (past, present and future) and only a finite number of genes. Each gene is a segment of DNA with a particular structure and it would be possible, in principle, to provide a detailed molecular description of that structure. We can now give a molecular specification of the gene by enumerating the genes and disjoining the molecular descriptions.[18] The point made above, that the segments which we count as genes do not share any structural property can now be put more precisely: any instantiation of (*) which replaces 'M' by a structural predicate from the language of molecular biology will insert a predicate that is essentially disjunctive.

Why does this matter? Let us imagine a reductionist using the enumerative strategy to deduce a general principle about gene transmission. After great labor, it is revealed that all actual genes satisfy the principle. I claim that more than this is needed to reduce a *law* about gene transmission. We envisage laws as sustaining counterfactuals, as applying to examples that might have been but which did not actually arise. To reduce the law it is necessary to show how possible but nonactual genes would have satisfied it. Nor can we achieve the reductionist's goal by adding further disjuncts to the envisaged bridge principle. For although there are only

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the inhibiting molecule from the neighborhood of the structural gene, and transcription begins again. (For much more detail, see Watson, op. cit., Chapter 14, and M. W. Strickberger, *Genetics* (New York: Macmillan, 1976), Chapter 29.)

[17]The situation is complicated by the existence of "introns"—segments within genes whose products under transcription are later excised—and by the enormous amount of repetitive DNA that most organisms seem to contain. Moreover, the regulatory systems in eukaryotes appear to be much more complicated than the prokaryote systems (of which the *lac* operon is *one* paradigm). For a review of the situation, as of a few years ago, see Eric H. Davidson, *Gene Expression in Early Development* (New York: Academic Press, 1976).

[18]The account will be even more complicated if we honor the suggestion of footnote 15, and suppose that, for a molecular structure to count as a gene, it must be produced in the right way.

finitely many *actual* genes, there are indefinitely many genes which *might* have arisen.

At this point, the reductionist may protest that the deck has been stacked. There is no need to produce a bridge principle of the form (*). Recall that we are trying to derive a general law about the transmission of genes, whose paradigm is Mendel's second law. Now the gross logical form of Mendel's second law is:

(1) (x) (y) ((Gx & Gy) → Axy).

We might hope to obtain this from statements of the forms

(2) (x) (Gx → Mx)

(3) (x) (y) ((Mx & My) → Axy)

where 'Mx' is an open sentence in the language of molecular biology. Now there will certainly be true statements of the form (2): for example, we can take 'Mx' as 'x is composed of DNA v.x is composed of RNA'. The question is whether we can combine some such statement with other appropriate premises—for example, some instance of (3)—so as to derive, and thereby explain (1). No geneticist or molecular biologist has advanced any suitable premises, and with good reason. We discover true statements of the form (2) by hunting for weak necessary conditions on genes, conditions which have to be met by genes but which are met by hordes of other biological entities as well. We can only hope to obtain *weak* necessary conditions because of the phenomenon that occupied us previously: from the molecular standpoint, genes are not distinguished by any common structure. Trouble will now arise when we try to show that the weak necessary condition is jointly sufficient for the satisfaction of the property (independent assortment at meiosis) that we ascribe to genes. The difficulty is illustrated by the example given above. If we take 'Mx' to be 'x is composed of DNA v.x is composed of RNA' then the challenge will be to find a general law governing the distribution of all segments of DNA and RNA!

I conclude that (R2) is false. Reductionists cannot find the bridge principles they need, and the tactic of abandoning the form (*) for something weaker is of no avail. I shall now consider (R3). Let us

concede both of the points that I have denied, allowing that there are general laws about the transmission of genes and that bridge principles are forthcoming. I claim that exhibiting derivations of the transmission laws from principles of molecular biology and bridge principles would not explain the laws, and, therefore, would not fulfill the major goal of reduction.

As an illustration, I shall use the envisaged amended version of Mendel's second law. Why do genes on nonhomologous chromosomes assort independently? Cytology provides the answer. At meiosis, chromosomes line up with their homologues. It is then possible for homologous chromosomes to exchange some genetic material, producing pairs of recombinant chromosomes. In the meiotic division, one member of each recombinant pair goes to each gamete, and the assignment of one member of one pair to a gamete is probabilistically independent of the assignment of a member of another pair to that gamete. Genes which occur close on the same chromosome are likely to be transmitted together (recombination is not likely to occur between them), but genes on nonhomologous chromosomes will assort independently.

This account is a perfectly satisfactory explanation of why our envisaged law is true to the extent that it is. (We recognize how the law could fail if there were some unusual mechanism linking particular nonhomologous chromosomes.) To emphasize the adequacy of the explanation is not to deny that it could be extended in certain ways. For example, we might want to know more about the mechanics of the process by which the chromosomes are passed on to the gametes. In fact, cytology provides such information. However, appeal to molecular biology would not deepen our understanding of the transmission law. Imagine a successful derivation of the law from principles of chemistry and a bridge principle of the form (*). In charting the details of the molecular rearrangements the derivation would only blur the outline of a simple cytological story, adding a welter of irrelevant detail. Genes on nonhomologous chromosomes assort independently because nonhomologous chromosomes are transmitted independently at meiosis, and, so long as we recognize this, we do not need to know what the chromosomes are made of.

In explaining a scientific law, L, one often provides a deduction of L from other principles. Sometimes it is possible to explain some of

the principles used in the deduction by deducing them, in turn, from further laws. Recognizing the possibility of a sequence of deductions tempts us to suppose that we could produce a better explanation of L by combining them, producing a more elaborate derivation in the language of our ultimate premises. But this is incorrect. What is relevant for the purposes of giving one explanation may be quite different from what is relevant for the purposes of explaining a law used in giving that original explanation. This general point is illustrated by the case at hand. We begin by asking why genes on nonhomologous chromosomes assort independently. The simple cytological story rehearsed above answers the question. That story generates *further* questions. For example, we might inquire why nonhomologous chromosomes are distributed independently at meiosis. To answer this question we would describe the formation of the spindle and the migration of chromosomes to the poles of the spindle just before meiotic division.[19] Once again, the narrative would generate yet further questions. Why do the chromosomes "condense" at prophase? How is the spindle formed? Perhaps in answering these questions we would begin to introduce the chemical details of the process. Yet simply plugging a molecular account into the narratives offered at the previous stages would *decrease* the explanatory power of those narratives. What is relevant to answering our original question is the fact that nonhomologous chromosomes assort independently. What is relevant to the issue of why nonhomologous chromosomes assort independently is the fact that the chromosomes are not selectively oriented toward the poles of the spindle. (We need to eliminate the doubt that, for example, the paternal and maternal chromosomes become separated and aligned toward opposite poles of the spindle.) In neither case are the molecular details relevant. Indeed, adding those details would only disguise the relevant factor.

There is a natural reductionist response. The considerations of the last paragraphs presuppose far too subjective a view of scien-

[19]Early in the process preceding meiotic division the chromosomes become more compact. As meiosis proceeds, the nucleus comes to contain a system of threads that resembles a spindle. Homologous chromosomes line up together near the center of the spindle, and they are oriented so that one member of each pair is slightly closer to one pole of the spindle, while the other is slightly closer to the opposite pole.

tific explanation. After all, even if *we* become lost in the molecular details, beings who are cognitively more powerful than we could surely recognize the explanatory force of the envisaged molecular derivation. However, this response misses a crucial point. The molecular derivation forfeits something important.

Recall the original cytological explanation. It accounted for the transmission of genes by identifying meiosis as a process of a particular kind: a process in which paired entities (in this case, homologous chromosomes) are separated by a force so that one member of each pair is assigned to a descendant entity (in this case, a gamete). Let us call processes of this kind *PS-processes.* I claim first that explaining the transmission law requires identifying PS-processes as forming a natural kind to which processes of meiosis belong, and second that PS-processes cannot be identified as a kind from the molecular point of view.

If we adopt the familiar covering law account of explanation, then we shall view the cytological narrative as invoking a law to the effect that processes of meiosis are PS-processes and as applying elementary principles of probability to compute the distribution of genes to gametes from the laws that govern PS-processes. If the illumination provided by the narrative is to be preserved in a molecular derivation, then we shall have to be able to express the relevant laws as laws in the language of molecular biology, and this will require that we be able to characterize PS-processes as a natural kind from the molecular point of view. The same conclusion, to wit that the explanatory power of the cytological account can be preserved only if we can identify PS-processes as a natural kind in molecular terms, can be reached in analogous ways if we adopt quite different approaches to scientific explanation—for example, if we conceive of explanation as specifying causally relevant properties or as fitting phenomena into a unified account of nature.

However, PS-processes are heterogeneous from the molecular point of view. There are no constraints on the molecular structures of the entities which are paired or on the ways in which the fundamental forces combine to pair them and to separate them. The bonds can be forged and broken in innumerable ways: all that matters is that there be bonds that initially pair the entities in question and that are subsequently (somehow) broken. In some cases, bonds may be formed directly between constituent molecules

of the entities in question; in others, hordes of accessory molecules may be involved. In some cases, the separation 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. I claim, therefore, that PS-processes are realized in a motley of molecular ways. (I should note explicitly that this conclusion is independent of the issue of whether the reductionist can find bridge principles for the concepts of classical genetics.)

We thus obtain a reply to the reductionist charge that we reject the explanatory power of the molecular derivation simply because we anticipate that our brains will prove too feeble to cope with its complexities.[20] The molecular account objectively fails to explain because it cannot bring out that feature of the situation which is highlighted in the cytological story. It cannot show us that genes are transmitted in the ways that we find them to be because meiosis

[20]The point I have been making is related to an observation of Hilary Putnam's. Discussing a similar example, Putnam writes: "The same explanation will go in any world (whatever the microstructure) in which those *higher level structural features* are present"; he goes on to claim that "explanation is superior not just subjectively but methodologically, . . . if it brings out relevant laws." (Putnam, "Philosophy and our Mental Life," in *Mind, Language, and Reality,* (Cambridge, Cambridge University Press, 1975), pp. 291–303, p. 296). The point is articulated by Alan Garfinkel (*Forms of Explanation,* New Haven: Yale University Press, 1981), and William Wimsatt has also raised analogous considerations about explanation in genetics.

It is tempting to think that the independence of the "higher level structural features" in Putnam's example and in my own can be easily established: one need only note that there are worlds in which the same feature is present without any molecular realization. So, in the case discussed in the text, PS-processes might go on in worlds where all objects were perfect continua. But although this shows that PS-processes form a kind which could be realized without molecular reshufflings, we know that all *actual* PS-processes do involve such reshufflings. The reductionist can plausibly argue that *if* the set of PS-processes with molecular realizations is itself a natural kind, then the explanatory power of the cytological account can be preserved by identifying meiosis as a process of this narrower kind. Thus the crucial issue is not whether PS-processes form a kind with non-molecular realizations, but whether those PS-processes which have molecular realizations form a kind that can be characterized from the molecular point of view. Hence, the easy strategy of responding to the reductionist must give way to the approach adopted in the text. (I am grateful to the editors of *The Philosophical Review* for helping me to see this point.)

is a PS-process and because any PS-process would give rise to analogous distributions. Thus (R3)—like (R1) and (R2)—is false.

3. The Root of the Trouble

Where did we go wrong? Here is a natural suggestion. The most fundamental failure of reductionism is the falsity of (R1). Lacking an account of theories which could readily be applied to the cases of classical genetics and molecular genetics, the attempt to chart the relations between these theories was doomed from the start. If we are to do better, we must begin by asking a preliminary question: what is the structure of classical genetics?

I shall follow this natural suggestion, endeavoring to present a picture of the structure of classical genetics which can be used to understand the intertheoretic relations between classical and molecular genetics.[21] As we have seen, the main difficulty in trying to axiomatize classical genetics is to decide what body of statements one is attempting to axiomatize. The history of genetics makes it clear that Morgan, Muller, Sturtevant, Beadle, McClintock, and others have made important contributions to genetic theory. But the statements occurring in the writings of these workers seem to be far too specific to serve as parts of a general theory. They concern the genes of particular kinds of organisms—primarily paradigm organisms, like fruit flies, bread molds, and maize. The idea that classical genetics is simply a heterogeneous set of statements about dominance, recessiveness, position effect, nondisjunction, and so forth, in *Drosophila, Zea mays, E. coli, Neurospora,* etc. flies in the face of our intuitions. The statements advanced by the great classical geneticists seem more like *illustrations* of the theory than *components* of it. (To know classical genetics it is not necessary to know the genetics of any particular organism, not even *Drosophila melanogaster.*) But the only alternative seems to be to suppose that there are general laws in genetics, never enunciated by genet-

[21] It would be impossible in the scope of this paper to do justice to the various conceptions of scientific theory that have emerged from the demise of the "received view." Detailed comparison of the perspective I favor with more traditional approaches (both those that remain faithful to core ideas of the "received view" and those that adopt the "semantic view" of theories) must await another occasion.

icists but reconstructible by philosophers. At the very least, this supposition should induce the worry that the founders of the field, and those who write the textbooks of today, do a singularly bad job.

Our predicament provokes two main questions. First, if we focus on a particular time in the history of classical genetics, it appears that there will be a set of statements about inheritance in particular organisms, which constitutes the corpus which geneticists of that time accept: what is the relationship between this corpus and the version of classical genetic theory in force at the time? (In posing this question, I assume, contrary to fact, that the community of geneticists was always distinguished by unusual harmony of opinion; it is not hard to relax this simplifying assumption.) Second, we think of genetic theory as something that persisted through various versions: what is the relation among the versions of classical genetic theory accepted at different times (the versions of 1910, 1930, and 1950, for example) which makes us want to count them as versions of the same theory?

We can answer these questions by amending a prevalent conception of the way in which we should characterize the state of a science at a time. The corpus of statements about the inheritance of characteristics accepted at a given time is only one component of a much more complicated entity that I shall call the *practice* of classical genetics at that time. There is a common language used to talk about hereditary phenomena, a set of accepted statements in that language (the corpus of beliefs about inheritance mentioned above), a set of questions taken to be the appropriate questions to ask about hereditary phenomena, and a set of patterns of reasoning which are instantiated in answering some of the accepted questions; (also: sets of experimental procedures and methodological rules, both designed for use in evaluating proposed answers; these may be ignored for present purposes). The practice of classical genetics at a time is completely specified by identifying each of the components just listed.[22]

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[22]My notion of a practice owes much to some neglected ideas of Sylvain Bromberger and Thomas Kuhn. See, in particular, Bromberger, "A Theory about the Theory of Theory and about the Theory of Theories," (W. L. Reese ed., *Philosophy of Science, The Delaware Seminar,* New York, 1963); and "Questions," (*Journal of Philosophy* 63 (1966), pp. 597–606); and Kuhn, *The*

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.[23] This notion of pattern is intended to explicate the idea of the common structure that underlies a group of problem-solutions.

The foregoing definitions enable us to answer the two main questions I posed above. Beliefs about the particular genetic features of particular organisms illustrate or exemplify the version of genetic theory in force at the time in the sense that these beliefs figure in particular problem-solutions generated by the current practice. Certain patterns of reasoning are applied to give the answers to accepted questions, and, in making the application, one puts forward claims about inheritance in particular organisms. Classical genetics persists as a single theory with different versions at different times in the sense that different practices are linked by a chain of practices along which there are relatively small modifications in language, in accepted questions, and in the patterns for answering questions. In addition to this condition of historical connection, versions of classical genetic theory are bound by a common structure: each version uses certain expressions to characterize he-

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Structure of Scientific Revolutions (Chicago: University of Chicago Press, 1962) Chapters II–V. The relation between the notion of a practice and Kuhn's conception of a paradigm is discussed in Chapter 7 of my book *The Nature of Mathematical Knowledge* (New York: Oxford University Press, 1983).

[23]More exactly, a general argument pattern is a triple consisting of a sequence of schematic sentences (a *schematic argument*), a set of filling instructions (directions as to how dummy letters are to be replaced), and a set of sentences describing the inferential characteristics of the schematic argument (a *classification* for the schematic argument). A sequence of sentences instantiates the general argument pattern just in case it meets the following conditions: (i) the sequence has the same number of members as the schematic argument of the general argument pattern; (ii) each sentence in the sequence is obtained from the corresponding schematic sentence in accordance with the appropriate filling instructions; (iii) it is possible to construct a chain of reasoning which assigns to each sentence the status accorded to the corresponding schematic sentence by the classification. For some efforts at explanation and motivation, see my "Explanatory Unification," *Philosophy of Science* 48 (1981), pp. 507–531.

reditary phenomena, accepts as important questions of a particular form, and offers a general style of reasoning for answering those questions. Specifically, throughout the career of classical genetics, the theory is directed toward answering questions about the distribution of characteristics in successive generations of a genealogy, and it proposes to answer those questions by using the probabilities of chromosome distribution to compute the probabilities of descendant genotypes.

The approach to classical genetics embodied in these answers is supported by reflection on what beginning students learn. Neophytes are not taught (and never have been taught) a few fundamental theoretical laws from which genetic "theorems" are to be deduced. They are introduced to some technical terminology, which is used to advance a large amount of information about special organisms. Certain questions about heredity in these organisms are posed and answered. Those who understand the theory are those who know what questions are to be asked about hitherto unstudied examples, who know how to apply the technical language to the organisms involved in these examples, and who can apply the patterns of reasoning which are to be instantiated in constructing answers. More simply, successful students grasp general patterns of reasoning which they can use to resolve new cases.

I shall now add some detail to my sketch of the structure of classical genetics, and thereby prepare the way for an investigation of the relations between classical genetics and molecular genetics. The initial family of problems in classical genetics, the family from which the field began, is the family of *pedigree problems*. Such problems arise when we confront several generations of organisms, related by specified connections of descent, with a given distribution of one or more characteristics. The question that arises may be to understand the given distribution of phenotypes, or to predict the distribution of phenotypes in the next generation, or to specify the probability that a particular phenotype will result from a particular mating. In general, classical genetic theory answers such questions by making hypotheses about the relevant genes, their phenotypic effects and their distribution among the individuals in the pedigree. Each version of classical genetic theory contains one or more problem-solving patterns exemplifying this general idea, but the detailed character of the pattern is refined in later versions, so

that previously recalcitrant cases of the problem can be accommodated.

Each case of a pedigree problem can be characterized by a set of *data,* a set of *constraints,* and a question. In any example, the data are statements describing the distribution of phenotypes among the organisms in a particular pedigree, or a diagram conveying the same information. The level of detail in the data may vary widely: at one extreme we may be given a full description of the interrelationships among all individuals and the sexes of all those involved; or the data may only provide the numbers of individuals with specific phenotypes in each generation; or, with minimal detail, we may simply be told that from crosses among individuals with specified phenotypes a certain range of phenotypes is found.

The constraints on the problem consist of general cytological information and descriptions of the chromosomal constitution of members of the species. The former will include the thesis that genes are (almost always)[24] chromosomal segments and the principles that govern meiosis. The latter may contain a variety of statements. It may be pertinent to know how the species under study reproduces, how sexual dimorphism is reflected at the chromosomal level, the chromosome number typical of the species, what loci are linked, what the recombination frequencies are, and so forth. As in the case of the data, the level of detail (and thus of stringency) in the constraints can vary widely.

Lastly, each problem contains a question that refers to the organisms described in the data. The question may take several forms: "What is the expected distribution of phenotypes from a cross between *a* and *b*?" (where *a, b* are specified individuals belonging to the pedigree described by the data), "What is the probability that a cross between *a* and *b* will produce an individual having *P*?" (where *a, b* are specified individuals of the pedigree described by the data and *P* is a phenotypic property manifested in this pedigree), "Why do we find the distribution of phenotypes described in the data?" and others.

Pedigree problems are solved by advancing pieces of reasoning that instantiate a small number of related patterns. In all cases the

[24]Sometimes particles in the cytoplasm account for hereditary traits. See Strickberger, op. cit., pp. 257–265.

reasoning begins from a *genetic hypothesis*. The function of a genetic hypothesis is to specify the alleles that are relevant, their phenotypic expression, and their transmission through the pedigree. From that part of the genetic hypothesis that specifies the genotypes of the parents in any mating that occurs in the pedigree, together with the constraints on the problem, one computes the expected distribution of genotypes among the offspring. Finally, for any mating occurring in the pedigree, one shows that the expected distribution of genotypes among the offspring is consistent with the assignment of genotypes given by the genetic hypothesis.

The form of the reasoning can easily be recognized in examples—examples that are familiar to anyone who has ever looked at a textbook or a research report in genetics.[25] What interests me is the style of reasoning itself. The reasoning begins with a genetic hypothesis that offers four kinds of information: (a) Specification of the number of relevant loci and the number of alleles at each locus; (b) Specification of the relationships between genotypes and phenotypes; (c) Specification of the relations between genes and chromosomes, of facts about the transmission of chromosomes to gametes (for example, resolution of the question whether there is disruption of normal segregation) and about the details of zygote formation; (d) Assignment of genotypes to individuals in the pedigree. After showing that the genetic hypothesis is consistent with the data and constraints of the problem, the principles of cytology and the laws of probability are used to compute expected distributions of genotypes from crosses. The expected distributions are then compared with those assigned in part (d) of the genetic hypothesis.[26]

Throughout the career of classical genetics, pedigree problems are addressed and solved by carrying out reasoning of the general type just indicated. Each version of classical genetic theory contains a pattern for solving pedigree problems with a method for computing expected genotypes which is adjusted to reflect the particular

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[25]For examples, see Strickberger op. cit. Chapters 6–12, 14–17, especially Chapter 11; Peters, op. cit.; and H. L. K. Whitehouse, *Towards an Understanding of the Mechanism of Heredity* (London: Arnold, 1965).

[26]The comparison will make use of standard statistical techniques, such as the chi-square test.

form of the genetic hypotheses that it sanctions. Thus one way to focus the differences among successive versions of classical genetic theory is to compare their conceptions of the possibilities for genetic hypotheses. As genetic theory develops, there is a changing set of conditions on admissible genetic hypotheses. Prior to the discovery of polygeny and pleiotropy (for example), part (a) of any adequate genetic hypothesis was viewed as governed by the requirement that there would be a one-one correspondence between loci and phenotypic traits.[27] After the discovery of incomplete dominance and epistasis, it was recognized that part (b) of an adequate hypothesis might take a form that had not previously been allowed: one is not compelled to assign to the heterozygote a phenotype assigned to one of the homozygotes, and one is also permitted to relativize the phenotypic effect of a gene to its genetic environment.[28] Similarly, the appreciation of phenomena of linkage, recombination, nondisjunction, segregation distortion, meiotic drive, unequal crossing over, and crossover suppression, modify conditions previously imposed on part (c) of any genetic hypothesis. In general, we can take each version of classical genetic theory to be associated with a set of conditions (usually not formulated explicitly) which govern admissible genetic hypotheses. While a general form of reasoning persists through the development of classical genetics, the patterns of reasoning used to resolve cases of the pedigree problem are constantly fine-tuned as geneticists modify their views about what forms of genetic hypothesis are allowable.

So far I have concentrated exclusively on classical genetic theory as a family of related patterns of reasoning for solving the pedigree problem. It is natural to ask if versions of the theory contain patterns of reasoning for addressing other questions. I believe that they do. The heart of the theory is the theory of *gene transmission,* the family of reasoning patterns directed at the pedigree problem. Out of this theory grow other subtheories. The theory of *gene mapping* offers a pattern of reasoning which addresses questions

[27]*Polygeny* occurs when many genes affect one characteristic; *pleiotropy* occurs when one gene affects more than one characteristic.

[28]*Incomplete dominance* occurs when the phenotype of the heterozygote is intermediate between that of the homozygotes; *epistasis* occurs when the effect of a particular combination of alleles at one locus depends on what alleles are present at another locus.

about the relative positions of loci on chromosomes. It is a direct result of Sturtevant's insight that one can systematically investigate the set of pedigree problems associated with a particular species. In turn, the theory of gene mapping raises the question of how to identify mutations, issues which are to be tackled by the *theory of mutation.* Thus we can think of classical genetics as having a central theory, the theory of gene transmission, which develops in the ways I have described above, surrounded by a number of satellite theories that are directed at questions arising from the pursuit of the central theory. Some of these satellite theories (for example, the theory of gene mapping) develop in the same continuous fashion. Others, like the theory of mutation, are subject to rather dramatic shifts in approach.

4. Molecular Genetics and Classical Genetics

Armed with some understanding of the structure and evolution of classical genetics, we can finally return to the question with which we began. What is the relation between classical genetics and molecular genetics? When we look at textbook presentations and the pioneering research articles that they cite, it is not hard to discern major ways in which molecular biology has advanced our understanding of hereditary phenomena. We can readily identify particular molecular explanations which illuminate issues that were treated incompletely, if at all, from the classical perspective. What proves puzzling is the connection of these explanations to the theory of classical genetics. I hope that the account of the last section will enable us to make the connection.

I shall consider three of the most celebrated achievements of molecular genetics. Consider first the question of *replication.* Classical geneticists believed that genes can replicate themselves. Even before the experimental demonstration that all genes are transmitted to all the somatic cells of a developing embryo, geneticists agreed that normal processes of mitosis and meiosis must involve gene replication. Muller's suggestion that the central problem of genetics is to understand how mutant alleles, incapable of performing wild-type functions in producing the phenotype, are nonetheless able to replicate themselves, embodies this consensus.

Yet classical genetics had no account of gene replication. A molecular account was an almost immediate dividend of the Watson-Crick model of DNA.

Watson and Crick suggested that the two strands of the double helix unwind and each strand serves as the template for the formation of a complementary strand. Because of the specificity of the pairing of nucleotides, reconstruction of DNA can be unambiguously directed by a single strand. This suggestion has been confirmed and articulated by subsequent research in molecular biology.[29] The details are more intricate than Watson and Crick may originally have believed, but the outline of their story stands.

A second major illumination produced by molecular genetics concerns the characterization of mutation. When we understand the gene as a segment of DNA we recognize the ways in which mutant alleles can be produced. "Copying errors" during replication can cause nucleotides to be added, deleted or substituted. These changes will often lead to alleles that code for different proteins, and which are readily recognizable as mutants through their production of deviant phenotypes. However, molecular biology makes it clear that there can be *hidden* mutations, mutations that arise through nucleotide substitutions that do not change the protein produced by a structural gene (the genetic code is redundant) or through substitutions that alter the form of the protein in trivial ways. The molecular perspective provides us with a general answer to the question, "What is a mutation?" namely that a mutation is the modification of a gene through insertion, deletion or substitution of nucleotides. This general answer yields a basic method for tackling (in principle) questions of form, "Is *a* a mutant allele?" namely a demonstration that *a* arose through nucleotide changes from alleles that persist in the present population. The method is frequently used in studies of the genetics of bacteria and bacteriophage, and can sometimes be employed even in inquiries about more complicated organisms. So, for example, there is good biochemical evidence for believing that some alleles which produce resistance to pesticides in various species of insects arose through

[29]See Watson, op. cit., Chapter 9; and Arthur Kornberg *DNA Synthesis* (San Fransisco: W. H. Freeman, 1974).

nucleotide changes in the alleles naturally predominating in the population.[30]

I have indicated two general ways in which molecular biology answers questions that were not adequately resolved by classical genetics. Equally obvious are a large number of more specific achievements. Identification of the molecular structures of particular genes in particular organisms has enabled us to understand why those genes combine to produce the phenotypes they do. One of the most celebrated cases is that of the normal allele for the synthesis of human hemoglobin and the mutant allele that is responsible for sickle-cell anemia.[31] The hemoglobin molecule—whose structure is known in detail—is built up from four amino-acid chains (two "α-chains" and two "β-chains"). The mutant allele results from substitution of a single nucleotide with the result that one amino acid is different (the sixth amino acid in the β-chains). This slight modification causes a change in the interactions of hemoglobin molecules: deoxygenated mutant hemoglobin molecules combine to form long fibres. Cells containing the abnormal molecule become deformed after they have given up their oxygen, and because they become rigid, they can become stuck in narrow capillaries, if they give up their oxygen too soon. Individuals who are homozygous for the mutant gene are vulnerable to experience blockages of blood flow. However, in heterozygous individuals, there is enough normal hemoglobin in blood cells to delay the time of formation of the distorting fibres, so that the individual is physiologically normal.

This example is typical of a broad range of cases, among which are some of the most outstanding achievements of molecular genetics. In all of the cases, we replace a simple assertion about the existence of certain alleles which give rise to various phenotypes with a molecular characterization of those alleles from which we can derive descriptions of the phenotypes previously attributed.

I claim that the successes of molecular genetics which I have just briefly described—and which are among the accomplishments

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[30]See. G. P. Georghiou, "The Evolution of Resistance to Pesticides," *Annual Review of Ecology and Systematics* 3 (1972), pp. 133–168.

[31]See Watson, op. cit., pp. 189–193; and T. H. Maugh II, "A New Understanding of Sickle Cell Emerges," *Science* 211 (1981), pp. 265–267.

most emphasized in the biological literature—can be understood from the perspective on theories that I have developed above. The three examples reflect three different relations among successive theories, all of which are different from the classical notion of reduction (and the usual modifications of it). Let us consider them in turn.

The claim that genes can replicate does not have the status of a central law of classical genetic theory.[32] It is not something that figures prominently in the explanations provided by the theory (as, for example, the Boyle-Charles law is a prominent premise in some of the explanations yielded by phenomenological thermodynamics). Rather, it is a claim that classical geneticists took for granted, a claim presupposed by explanations, rather than an explicit part of them. Prior to the development of molecular genetics that claim had come to seem increasingly problematic. If genes can replicate, how do they manage to do it? Molecular genetics answered the worrying question. It provided a theoretical demonstration of the possibility of an antecedently problematic presupposition of classical genetics.

We can say that a theory presupposes a statement p if there is some problem-solving pattern of the theory, such that every instantiation of the pattern contains statements that jointly imply the truth of p. Suppose that, at a given stage in the development of a theory, scientists recognize an argument from otherwise acceptable premises which concludes that it is impossible that p. Then the presupposition p is problematic for those scientists. What they would like would be an argument showing that it is possible that p and explaining what is wrong with the line of reasoning which appears to threaten the possibility of p. If a new theory generates

[32]However, one might claim that "Genes can replicate" is a law of genetics, in that it is general, lawlike, and true. This does not vitiate my claim that the structure of classical genetics is not to be sought by looking for a set of general laws, for the law in question is so weak that there is little prospect of finding supplementary principles which can be conjoined with it to yield a representation of genetic theory. I suggest that "Genes can replicate" is analogous to the thermodynamic "law," "Gases can expand," or to the Newtonian "law," "Forces can be combined." If the only laws that we could find in thermodynamics and mechanics were weak statements of this kind we would hardly be tempted to conceive of these sciences as sets of laws. I think that the same point goes for genetics.

an argument of this sort, then we can say that the new theory gives a theoretical demonstration of the possibility of an antecedently problematic presupposition of the old theory.

A less abstract account will help us to see what is going on in the case of gene replication. Very frequently, scientists take for granted in their explanations some general property of entities that they invoke. Their assumption can come to seem problematic if the entities in question are supposed to belong to a kind, and there arises a legitimate doubt about whether members of the kind can have the property attributed. A milder version of the problem arises if, in all cases in which the question of whether things of the general kind have the property can be settled by appealing to background theory, it turns out that the answer is negative. Under these circumstances, the scientists are committed to regarding their favored entities as unlike those things of the kind which are amenable to theoretical study with respect to the property under discussion. The situation is worse if background theory provides an argument for thinking that *no* things of the kind can have the property.

Consider now the case of gene replication. For any problem-solution offered by any version of the theory of gene transmission (the central subtheory of classical genetic theory), that problem-solution will contain sentences implying that the alleles which it discusses are able to replicate. Classical genetics presupposes that a large number of identifiable genes can replicate. This presupposition was always weakly problematic because genes were taken to be complicated molecules and, in all cases in which appeal to biochemistry could be made to settle the issue of whether a molecular structure was capable of replication, the issue was decided in the negative. Muller exacerbated the problem by suggesting that mutant alleles are damaged molecules (after all, many of them were produced through x-ray bombardment, an extreme form of molecular torture). So there appeared to be a strong argument against the possibility that any mutant allele can replicate. After the work of Watson, Crick, Kornberg, and others, there was a theoretical demonstration of the allegedly problematic possibility. One can show that genes can replicate by showing that any segment of DNA (or RNA) can replicate. (DNA and RNA are the genetic materials. Establishing the power of the genetic material to replicate bypasses the problem of deciding which segments are genes. Thus the diffi-

culties posed by the falsity of [R2] are avoided.) The Watson-Crick model provides a characterization of the (principal) genetic material, and when this description is inserted into standard patterns of chemical reasoning one can generate an argument whose conclusion asserts that, under specified conditions, DNA replicates. Moreover, given the molecular characterization of DNA and of mutation, it is possible to see that although mutant alleles are "damaged" molecules, the kind of damage (insertion, deletion or substitution of nucleotides) does not affect the ability of the resultant molecule to replicate.

Because theoretical demonstrations of the possibility of antecedently problematic presuppositions involve derivation of conclusions of one theory from the premises supplied by a background theory, it is easy to assimilate them to the classical notion of reduction. However, on the account I have offered, there are two important differences. First, there is no commitment to the thesis that genetic theory can be formulated as (the deductive closure of) a conjunction of laws. Second, it is not assumed that all general statements about genes are equally in need of molecular derivation. Instead, one particular thesis, a thesis that underlies all the explanations provided by classical genetic theory, is seen as especially problematic, and the molecular derivation is viewed as addressing a specific problem that classical geneticists had already perceived. Where the reductionist identifies a general benefit in deriving all the axioms of the reduced theory, I focus on a particular derivation of a claim that has no title as an axiom of classical genetics, a derivation which responds to a particular explanatory difficulty of which classical geneticists were acutely aware. The reductionist's global relation between theories does not obtain between classical and molecular genetics, but something akin to it does hold between special fragments of these theories.[33]

The second principal achievement of molecular genetics, the account of mutation, involves a conceptual refinement of prior

[33]A similar point is made by Kenneth Schaffner in a forthcoming book on theory structure in the biomedical sciences. Schaffner's terminology is different from my own, and he continues to be interested in the prospects of global reduction, but there is considerable convergence between the conclusions that he reaches and those that I argue for in the present section.

theory. Later theories can be said to provide conceptual refinements of earlier theories when the later theory yields a specification of entities that belong to the extensions of predicates in the language of the earlier theory, with the result that the ways in which the referents of these predicates are fixed are altered in accordance with the new specifications. Conceptual refinement may occur in a number of ways. A new theory may supply a descriptive characterization of the extension of a predicate for which no descriptive characterization was previously available; or it may offer a new description which makes it reasonable to amend characterizations that had previously been accepted.[34] In the case at hand, the referent of many tokens of 'mutant allele' was initially fixed through the description "chromosomal segment producing a heritable deviant phenotype." After Bridges's discovery of unequal crossing-over at the *Bar* locus in *Drosophila,* it was evident to classical geneticists that this descriptive specification covered cases in which the internal structure of a gene was altered and cases in which neighboring genes were transposed. Thus it was necessary to retreat to the less applicable description "chromosomal segment producing a heritable deviant phenotype as the result of an internal change within an allele." Molecular genetics offers a precise account of the internal changes, with the result that the description can be made more informative: mutant alleles are segments of DNA that result from prior alleles through deletion, insertion, or substitution of nucleotides. This re-fixing of the referent of 'mutant allele' makes it possible in principle to distinguish cases of mutation from cases of recombination, and thus to resolve those controversies that frequently arose from the use of 'mutant allele' in the later days of classical genetics.[35]

Finally, let us consider the use of molecular genetics to illuminate the action of particular genes. Here we again seem to find a relationship that initially appears close to the reductionist's ideal. Statements that are invoked as premises in particular problem-solu-

[34]There are numerous examples of such modifications from the history of chemistry. I try to do justice to this type of case in "Theories, Theorists, and Theoretical Change," *The Philosophical Review* 87 (1978), pp. 519–547 and in "Genes."

[35]Molecular biology also provided significant refinement of the terms 'gene' and 'allele'. See "Genes."

tions—statements that ascribe particular phenotypes to particular genotypes—are derived from molecular characterizations of the alleles involved. On the account of classical genetics offered in Section 3, each version of classical genetic theory includes in its schema for genetic hypotheses a clause which relates genotypes to phenotypes (clause [b] in the description of a genetic hypothesis on p. 356 above). Generalizing from the hemoglobin example, we might hope to discover a pattern of reasoning within molecular genetics that would generate as its conclusion the schema for assigning phenotypes to genotypes.

It is not hard to characterize the relation just envisioned. Let us say that a theory T′ provides an *explanatory extension* of a theory T just in case there is some problem-solving pattern of T one of whose schematic premises can be generated as the conclusion of a problem-solving pattern of T′. When a new theory provides an explanatory extension of an old theory, then particular premises occurring in explanatory derivations given by the old theory can themselves be explained by using arguments furnished by the new theory. However, it does not follow that the explanations provided by the old theory can be improved by replacing the premises in question with the pertinent derivations. What is relevant for the purposes of explaining some statement S may not be relevant for the purposes of explaining a statement S′ which figures in an explanatory derivation of S.

Even though reductionism fails, it may appear that we can capture part of the spirit of reductionism by deploying the notion of explanatory extension. The thesis that molecular genetics provides an explanatory extension of classical genetics embodies the idea of a global relationship between the two theories, while avoiding two of the three troubles that were found to beset reductionism. That thesis does not simply assert that some specific presupposition of classical genetics (for example, the claim that genes are able to replicate) can be derived as the conclusion of a molecular argument, but offers a general connection between premises of explanatory derivations in classical genetics and explanatory arguments from molecular genetics. It is formulated so as to accommodate the failure of (R1) and to honor the picture of classical genetics developed in Section 3. Moreover, the failure of (R2) does not affect it. If we take the hemoglobin example as a paradigm, we can justifia-

bly contend that the explanatory extension does not require any general characterization of genes in molecular terms. All that is needed is the possibility of deriving phenotypic descriptions from molecular characterizations of the structures of *particular* genes. Thus, having surmounted two hurdles, our modified reductionist thesis is apparently within sight of success.

Nevertheless, even born-again reductionism is doomed to fall short of salvation. Although it is true that molecular genetics belongs to a cluster of theories which, taken together, provide an explanatory extension of classical genetics, molecular genetics, on its own, cannot deliver the goods. There are some cases in which the ancillary theories do not contribute to the explanation of a classical claim about gene action. In such cases, the classical claim can be derived and explained by instantiating a pattern drawn from molecular genetics. The example of human hemoglobin provides one such case. But this example is atypical.

Consider the way in which the hemoglobin example works. Specification of the molecular structures of the normal and mutant alleles, together with a description of the genetic code, enables us to derive the composition of normal and mutant hemoglobin. Application of chemistry then yields descriptions of the interactions of the proteins. With the aid of some facts about human blood cells, one can then deduce that the sickling effect will occur in abnormal cells, and, given some facts about human physiology, it is possible to derive the descriptions of the phenotypes. There is a clear analogy here with some cases from physics. The assumptions about blood cells and physiological needs seem to play the same role as the boundary conditions about shapes, relative positions and velocities of planets that occur in Newtonian derivations of Kepler's laws. In the Newtonian explanation we can see the application of a general pattern of reasoning—the derivation of explicit equations of motion from specifications of the forces acting—which yields the general result that a body under the influence of a centrally directed inverse square force will travel in a conic section; the general result is then applied to the motions of the planets by incorporating pieces of astronomical information. Similarly, the derivation of the classical claims about the action of the normal and mutant hemoglobin genes can be seen as a purely chemical derivation of the generation of certain molecular structures and of the interactions

among them. The chemical conclusions are then applied to the biological system under consideration by introducing three "boundary conditions": first, the claim that the altered molecular structures only affect development to the extent of substituting a different molecule in the erythrocytes (the blood cells that transport hemoglobin); second, a description of the chemical conditions in the capillaries; and third, a description of the effects upon the organism of capillary blockage.

The example is able to lend comfort to reductionism precisely because of an atypical feature. In effect, one concentrates on the *differences* among the phenotypes, takes for granted the fact that in all cases development will proceed normally to the extent of manufacturing erythrocytes—which are, to all intents and purposes, simply sacks for containing hemoglobin molecules—and compares the difference in chemical effect of the cases in which the erythrocytes contain different molecules. *The details of the process of development can be ignored.* However, it is rare for the effect of a mutation to be so simple. Most structural genes code for molecules whose presence or absence make subtle differences. Thus, typically, a mutation will affect the distribution of chemicals in the cells of a developing embryo. A likely result is a change in the timing of intracellular reactions, a change that may, in turn, alter the shape of the cell. Because of the change of shape, the geometry of the embryonic cells may be modified. Cells that usually come into contact may fail to touch. Because of this, some cells may not receive the molecules necessary to switch on certain batteries of genes. Hence the chemical composition of these cells will be altered. And so it goes.[36]

Quite evidently, in examples like this, (which include most of the cases in which molecular considerations can be introduced into embryology) the reasoning that leads us to a description of the phenotype associated with a genotype will be much more complicated than that found in the hemoglobin case. It will not simply consist in a chemical derivation adapted with the help of a few

[36]For examples, see N. K. Wessels *Tissue Interactions and Development* (Menlo Park, Ca.: W. A. Benjamin, 1977), especially Chapters 6, 7, 13–15; and Donald Ede, *An Introduction to Developmental Biology* (London: Blackie, 1978) especially Chapter 13.

boundary conditions furnished by biology. Instead, we shall encounter a sequence of subarguments: molecular descriptions lead to specifications of cellular properties, from these specifications we draw conclusions about cellular interactions, and from these conclusions we arrive at further molecular descriptions. There is clearly a pattern of reasoning here which involves molecular biology and which extends the explanations furnished by classical genetics by showing how phenotypes depend upon genotypes—but I think it would be folly to suggest that the extension is provided by molecular genetics alone.

In Section 2, we discovered that the traditional answer to the philosophical question of understanding the relation that holds between molecular genetics and classical genetics, the reductionist's answer, will not do. Section 3 attempted to build on the diagnosis of the ills of reductionism, offering an account of the structure and evolution of classical genetics that would improve on the picture offered by those who favor traditional approaches to the nature of scientific theories. In the present section, I have tried to use the framework of Section 3 to understand the relations between molecular genetics and classical genetics. Molecular genetics has done something important for classical genetics, and its achievements can be recognized by seeing them as instances of the intertheoretic relations that I have characterized. Thus I claim that the problem from which we began is solved.

So what? Do we have here simply a study of a particular case—a case which has, to be sure, proved puzzling for the usual accounts of scientific theories and scientific change? I hope not. Although the traditional approaches may have proved helpful in understanding some of the well-worn examples that have been the stock-in-trade of twentieth century philosophy of science, I believe that the notion of scientific practice sketched in Section 3 and the intertheoretic relations briefly characterized here will both prove helpful in analyzing the structure of science and the growth of scientific knowledge *even in those areas of science where traditional views have seemed most successful.*[37] Hence the tale of two sciences which I have

[37]I attempt to show how the same perspective can be fruitfully applied to other examples in "Explanatory Unification," Sections 3 and 4; *Abusing Science* (Cambridge: MIT Press, 1982) Chapter 2; and "Darwin's Achievement," (forthcoming in a volume of the *Pittsburgh Studies in the Philosophy of Science*).

been telling is not merely intended as a piece of local history that fills a small but troublesome gap in the orthodox chronicles. I hope that it introduces concepts of general significance in the project of understanding the growth of science.

5. Anti-Reductionism and the Organization of Nature

One loose thread remains. The history of biology is marked by continuing opposition between reductionists and anti-reductionists. Reductionism thrives on exploiting the charge that it provides the only alternative to the mushy incomprehensibility of vitalism. Anti-reductionists reply that their opponents have ignored the organismic complexity of nature. Given the picture painted above, where does this traditional dispute now stand?

I suggest that the account of genetics which I have offered will enable reductionists to provide a more exact account of what they claim, and will thereby enable anti-reductionists to be more specific about what they are denying. Reductionists and anti-reductionists agree in a certain minimal physicalism. To my knowledge, there are no major figures in contemporary biology who dispute the claim that each biological event, state or process is a complex physical event, state, or process. The most intricate part of ontogeny or phylogeny involves countless changes of physical state. What anti-reductionists emphasize is the organization of nature and the "interactions among phenomena at different levels." The appeal to organization takes two different forms. When the subject of controversy is the proper form of evolutionary theory, then anti-reductionists contend that it is impossible to regard all selection as operating at the level of the gene.[38] What concerns me here is not

[38]The extreme version of reductionism is defended by Richard Dawkins in *The Selfish Gene* (New York: Oxford University Press, 1976) and *The Extended Phenotype* (San Francisco: W. H. Freeman, 1982). For an excellent critique, see Elliott Sober and Richard C. Lewontin, "Artifact, Cause, and Genic Selection," *Philosophy of Science* 49 (1982), pp. 157–180. More ambitious forms of antireductionism with respect to evolutionary theory are advanced in S. J. Gould, "Is a new and general theory of evolution emerging?" *Paleobiology,* 6 (1980), pp. 119–130; N. Eldredge and J. Cracraft, *Phylogenetic Patterns and the Evolutionary Process* (New York: Columbia University Press, 1980); and Steven M. Stanley, *Macroevolution* (San Francisco: W. H. Freeman, 1979). A classic early source of some (but not all) later anti-reductionist themes is Ernst Mayr's *Animal Species and Evolution* (Cambridge, Harvard University Press, 1963) especially Chapter 10.

this area of conflict between reductionists and their adversaries, but the attempt to block claims for the hegemony of molecular studies in understanding the physiology, genetics, and development of organisms.[39]

A sophisticated reductionist ought to allow that, in the current practice of biology, nature is divided into levels which form the proper provinces of areas of biological study: molecular biology, cytology, histology, physiology, and so forth. Each of these sciences can be thought of as using certain language to formulate the questions it deems important and as supplying patterns of reasoning for resolving those questions. Reductionists can now set forth one of two main claims. The stronger thesis is that the explanations provided by any biological theories can be reformulated in the language of molecular biology and be recast so as to instantiate the patterns of reasoning supplied by molecular biology. The weaker thesis is that molecular biology provides explanatory extension of the other biological sciences.

Strong reductionism falls victim to the considerations that were advanced against (R3). 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. We may formulate this point in the biologists' preferred idiom by saying that the assortment of alleles is to be understood at the cytological level. What is meant by this description is that there is a pattern of reasoning which is applied to derive the description of the assortment of alleles and which involves predicates that characterize cells and their large-scale internal structures. That pattern of reasoning is to be objectively preferred to the molecular pattern which would be instantiated by the derivation that charts that complicated rearrangements of individual molecules because it can be applied across a range of cases which would look heterogeneous

[39]Gould's *Ontogeny and Phylogeny* (Harvard, 1977) provides historical illumination of both areas of debate about reductionism. Contemporary anti-reductionist arguments about embryology are expressed by Wessels (op. cit.) and Ede (op. cit.). See also G. Oster and P. Alberch, "Evolution and Bifurcation of Developmental Programs," *Evolution* 36 (1982), pp. 444–459.

from a molecular perspective. Intuitively, the cytological pattern makes connections which are lost at the molecular level, and it is thus to be preferred.

So far, anti-reductionism emerges as the thesis that there are *autonomous levels of biological explanation.* Anti-reductionism construes the current division of biology not simply as a temporary feature of our science stemming from our cognitive imperfections but as the reflection of levels of organization in nature. Explanatory patterns that deploy the concepts of cytology will endure in our science because we would foreswear significant unification (or fail to employ the relevant laws, or fail to identify the causally relevant properties) by attempting to derive the conclusions to which they are applied using the vocabulary and reasoning patterns of molecular biology. But the autonomy thesis is only the beginning of anti-reductionism. A stronger doctrine can be generated by opposing the weaker version of sophisticated reductionism.

In Section 4, I raised the possibility that molecular genetics may be viewed as providing an explanatory extension of classical genetics through deriving the schematic sentence that assigns phenotypes to genotypes from a molecular pattern of reasoning. This apparent possibility fails in an instructive way. Anti-reductionists are not only able to contend that there are autonomous levels of biological explanation. They can also resist the weaker reductionist view that explanation always flows from the molecular level up. Even if reductionists retreat to the modest claim that, while there are autonomous levels of explanation, descriptions of cells and their constituents are always explained in terms of descriptions about genes, descriptions of tissue geometry are always explained in terms of descriptions of cells, and so forth, anti-reductionists can resist the picture of a unidirectional flow of explanation. Understanding the phenotypic manifestation of a gene, they will maintain, requires constant shifting back and forth across levels. Because developmental processes are complex and because changes in the timing of embryological events may produce a cascade of effects at several different levels, one sometimes uses descriptions at higher levels to explain what goes on at a more fundamental level.

For example, to understand the phenotype associated with a mutant limb-bud allele, one may begin by tracing the tissue geome-

try to an underlying molecular structure. The molecular constitution of the mutant allele gives rise to a nonfunctional protein, causing some abnormality in the internal structures of cells. The abnormality is reflected in peculiarities of cell shape, which, in turn, affects the spatial relations among the cells of the embryo. So far we have the unidirectional flow of explanation which the reductionist envisages. However, the subsequent course of the explanation is different. Because of the abnormal tissue geometry, cells that are normally in contact fail to touch; because they do not touch, certain important molecules, which activate some batteries of genes, do not reach crucial cells; because the genes in question are not "switched on" a needed morphogen is not produced; the result is an abnormal morphology in the limb.

Reductionists may point out, quite correctly, that there is some very complex molecular description of the entire situation. The tissue geometry is, after all, a configuration of molecules. But this point is no more relevant than the comparable claim about the process of meiotic division in which alleles are distributed to gametes. Certain genes are not expressed because of the geometrical structure of the cells in the tissue: *the pertinent cells are too far apart.* However this is realized at the molecular level, our explanation must bring out the salient fact that it is the presence of a gap between cells that are normally adjacent that explains the nonexpression of the genes. As in the example of allele transmission at meiosis, we lose sight of the important connections by attempting to treat the situation from a molecular point of view. As before, the point can be sharpened by considering situations in which radically different molecular configurations realize the crucial feature of the tissue geometry: situations in which heterogeneous molecular structures realize the breakdown of communication between the cells.

Hence, embryology provides support for the stronger anti-reductionist claim. Not only is there a case for the thesis of autonomous levels of explanation, but we find examples in which claims at a more fundamental level (specifically, claims about gene expression) are to be explained in terms of claims at a less fundamental level (specifically, descriptions of the relative positions of pertinent cells). Two anti-reductionist biologists put the point succinctly:

> . . . a developmental program is not to be viewed as a linearly organized causal chain from genome to phenotype. Rather, morphology emerges as a consequence of an increasingly complex dialogue between cell populations, characterized by their geometric continuities, and the cells' genomes, characterized by their states of gene activity.[40]

A corollary is that the explanations provided by the "less fundamental" biological sciences are not extended by molecular biology alone.

It would be premature to claim that I have shown how to reformulate the anti-reductionist appeals to the organization of nature in a completely precise way. My conclusion is that, to the extent that we can make sense of the present explanatory structure within biology—that division of the field into subfields corresponding to levels of organization in nature—we can also understand the anti-reductionist doctrine. In its minimal form, it is the claim that the commitment to several explanatory levels does not simply reflect our cognitive limitations; in its stronger form, it is the thesis that some explanations oppose the direction of preferred reductionistic explanation. Reductionists should not dismiss these doctrines as incomprehensible mush unless they are prepared to reject as unintelligible the biological strategy of dividing the field (a strategy which seems to me well understood, even if unanalyzed).

The examples I have given seem to support both anti-reductionist doctrines. To clinch the case, further analysis is needed. The notion of explanatory levels obviously cries out for explication, and it would be illuminating to replace the informal argument that the unification of our beliefs is best achieved by preserving multiple explanatory levels with an argument based on a more exact criterion for unification. Nevertheless, I hope that I have said enough to make plausible the view that, despite the immense value of the molecular biology that Watson and Crick launched in 1953, molecular studies cannot cannibalize the rest of biology. Even if geneticists must become "physiological chemists" they should not give up being embryologists, physiologists, and cytologists.

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[40]Oster and Alberch, op. cit., p. 454. The diagram on p. 452 provides an equally straightforward account of their anti-reductionist position.