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Abstract

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ON THE REDUCTION OF GENETICS TO MOLECULAR BIOLOGY*

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The applicability of Nagel's concept of theory reduction, and related concepts of reduction, to the reduction of genetics to molecular biology is examined using the lactose operon in *Escherichia coli* as an example. Geneticists have produced the complete nucleotide sequence of two of the genes which compose this operon. If any example of reduction in genetics should fit Nagel's analysis, the lactose operon should. Nevertheless, Nagel's formal conditions of theory reduction are inapplicable in this case. Instead, it is argued that genetics has been partially reduced to molecular biology in the sense of token-token reduction.

I

According to one received view of science and scientific reduction—that of the logical empiricists, including Nagel—a science must have a number of experimental laws. These laws must be, among other things, universal generalizations (see Dretske 1977 and Nagel 1961, page 48). If the science is sufficiently advanced, it will also have a theory which explains the laws of the science. The question of whether one science, *S*2, is reducible to another, *S*1, is the question (under the received view) of whether the laws of *S*2 can be derived from *S*1 augmented with correspondence rules which connect terms peculiar to *S*2 with terms of *S*1. Nagel writes that

. . . when the laws of the secondary science do contain some term 'A' that is absent from the theoretical assumptions of the primary science, there are two necessary formal conditions for the reduction of the former to the latter: (1) Assumptions of some kind must be introduced which postulate suitable relations between whatever is signified by 'A' and traits represented by theoretical terms already present in the primary science. . . . it will be convenient to refer to this condition as the "condition of connectability." (2) With the help of these additional assumptions, all the laws of the secondary science, including those containing the term 'A', must be logically derivable from the theoretical premises

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and their associated coordinating definitions in the primary discipline. Let us call this the "condition of derivability." (1961, pp. 353-4)

The task of this essay is to examine the formal relations between genetics and molecular biology in order to see if they now conform to Nagelian and related forms of reduction. I shall claim that they do not and are not likely ever to do so, in spite of attempts to argue the contrary (see Goosens 1978, Ruse 1976, and Schaffner 1976). This leads to something of a paradox, for preanalytically the science of genetics has been or is in the midst of being reduced to the science of molecular biology. After all, everyone knows that genes are made of DNA. I believe that another analysis of reduction properly describes the present relation between genetics and molecular biology and I shall present this analysis in Section IV.

The argument of this essay is contained in Sections III and IV. The purpose of Section II is to present concisely material from genetics and molecular biology which will be drawn upon as subsequently needed. Section II contains information about the genetic and molecular aspects of the lactose operon, which provides a particularly good example for a study of the reduction of genetics, because it has been extensively and successfully investigated from both a genetic and a molecular point of view. Indeed, the two approaches have been very well integrated and the molecular structure of two of the genes in the lactose operon is known.

II

I propose to dispense with most of the genetic and biochemical background for the present discussion by the simple device of ignoring it. Any discussion of basic genetics and molecular biology in a short essay would be too brief to do much good. My arguments will be based upon only a few facts. Those familiar with Mendel's Laws, genetic mapping, the standard chromosomal abnormalities, dominance, what James Watson calls the Central Dogma of molecular biology (DNA is transcribed onto mRNA which is translated into protein.), the structure of DNA, and DNA replication should have little difficulty judging the correctness of my factual account. Those not independently familiar with molecular genetics will have to take it on faith that my factual account is correct.

Important for what follows is that genes are on chromosomes; chromosomes of *Escherichia coli* (*E. coli*, a common and extensively studied bacterium) are naked DNA; and DNA replicates with a mechanism and under conditions which are fairly well understood.

“Wild type” *E. coli* can live on lactose as its sole source of carbon. When *E. coli* are grown in the presence of lactose, the following three enzymes are present in significant quantity: beta-galactosidase, galactoside permease, and thiogalactoside transacetylase. When lactose is absent, these enzymes are either absent or present in minute quantities. The gene responsible for beta-galactosidase is designated the *z* gene, that for galactoside permease the *y* gene, and that for the acetylase the *a* gene. These genes map contiguously in the following order: $\frac{z}{y/a}$. The enzymes for which they are responsible are called inducible enzymes because they are present in the cell only when induced by a particular agent, in this case lactose and certain similar molecules.

A number of mutations were discovered which allowed the production of three lactose enzymes (beta-galactosidase, galactoside permease, and thiogalactoside transacetylase) even in the absence of lactose. These mutations mapped together at some distance to the left of *z* and were called constitutive mutations. This discovery led to the postulation not only of a new gene, the *i* gene, but of a new kind of gene, a regulator gene. The other genes so far mentioned, *z*, *y*, and *a*, were called structural genes. The constitutive mutations of the *i* gene are labelled i^- and the “wild” or non-mutant gene, i^+ . Interestingly, in *E. coli* merozygotes, it was found that i^+ is dominant to i^- . Other kinds of *i* mutants have been found but they need not concern us here.

Shortly after discovery of i^- mutants, mutants from another region affecting the activity of *z*, *y*, and *a* were found. These mutants were in what was called the operator region, *o*. They map adjacent to the *z* gene and cause constitutive synthesis of the lactose enzymes even in the presence of i^+ . This system—consisting of the regulator gene, the operator region, and the three lactose enzyme genes—is called the lactose operon. Although the lactose operon is an example of the simplest of gene regulating mechanisms, it is considerably more complex than described here. But what has been presented will suffice for our purposes.

Put simply, the lactose operon works as follows. The *i* gene produces a substance, called the repressor, which binds to the operator and prevents the *z*, *y*, and *a* genes from producing their characteristic messenger RNA (mRNA) and hence their characteristic enzymes. When lactose is present, it somehow causes the repressor to become ineffective and this results in the production of the lactose enzymes which metabolize lactose. After lactose is completely metabolized (and no longer present), the repressor is again effective and production of the lactose enzymes ceases.

From the above discussion we know that there is an operator region, *o*, which maps between the *i* gene and the *z* gene in the lactose operon. Because so much is known about the operator region, it presents us with some useful examples for a study of reduction. According to genetic mapping experiments, the *o* region lies between and adjacent to both the *z* and the *i* genes. By use of transducing phages, it is possible to isolate physically the genetically characterized *o* region and to study it. One such study (Dickson, et al. 1975) led to the complete sequencing of the bit of DNA which functions as the *o* region for the lactose enzymes. The study also produced a detailed model of how the control genes work.

A further complication must be introduced. The control region, *o*, is actually two genes—the promoter, *p*, and the operator, *o*. The promoter gene lies between the *i* gene and the *o* gene. The genes we have discussed are arranged as follows: *i/p/o/z/y/a/*, where *i* is a regulator gene, *p* and *o* operator genes, and *z*, *y* and *a* structural genes. Remember: all these genes were discovered, characterized, and mapped genetically. They are part and parcel of the science of genetics.

The molecular workings of the lactose operon are, as I have said, complex. There are but a few facts which will suffice for the subsequent discussion.

1. The *p* and *o* genes are bits of DNA whose base sequence has been determined.
2. The *z*, *y*, and *a* genes are transcribed onto mRNA and this mRNA is translated into the three proteins (beta-galactosidase, etc.).
3. The *o* gene is transcribed onto mRNA, but this mRNA is not translated into protein.
4. The *p* gene is neither transcribed onto mRNA nor translated into anything at all.
5. The *i* gene is transcribed and translated into protein.
6. The *p* and *o* genes overlap, i.e., they share some nucleotide pairs.

III

In the present section I shall introduce several kinds of reduction and give arguments on the question of whether any of them adequately describes the reduction of genetics to molecular biology which is actually occurring. Although my subject is the question of whether or not the entire science of genetics can be reduced to molecular biology, my arguments will often center upon the question of whether

the predicate “. . . is a gene” can be reduced to molecular biology. I do this for three reasons. First, genetics is a large science, containing too many predicates for a complete and adequate discussion in a short paper. Also, I do not believe that adding a discussion of more predicates would materially affect the conclusions to be drawn. Secondly, the concept of a gene is central to genetics; if it cannot be reduced there is reason to think that nothing very similar to the current science of genetics can be reduced. Thirdly, a great deal of work, such as that cited in Section II, has been performed which is certainly relevant to the reduction of the concept of a gene to the concepts of molecular biology.

For convenience of exposition, I shall consider the laws of a science as mapping functions between the predicates of the science. An example of a law would be:

$$Px \rightarrow Qy.$$

It reads, approximately, as ‘every x which is P brings about a y which is Q .’ The predicates of the reduced science, S_2 , will be called $Z, Y, X, \dots$ and the predicates of the reducing science, S_1 , are $A, B, C, \dots$. Thus, we may picture the two sciences considered for the purpose of reduction as follows.

$$S_2: Zx \rightarrow Wy, Yx \rightarrow Zw, \text{ etc.}$$

$$S_1: Ax \rightarrow By, Cy \rightarrow Dx, \text{ etc.}$$

The question of reduction is the question of how the statements of one science are to be connected with the statements of another science. In the case that one of the sciences contains predicates not contained in the other science, any reduction which occurs is called a heterogeneous reduction. Obviously, a reduction of genetics to molecular biology will be heterogeneous. The predicate “. . . is a gene” is part of genetics and not part of molecular biology. The same can be said for many other predicates, e.g. “. . . is epistatic to . . .,” “. . . is linked with . . .” Clearly, in a heterogeneous reduction, if the laws or concepts of one science are to be derived from the laws or concepts of another science, S_1 (the reducing science) must be augmented with statements that connect the concepts of S_1 with those of S_2 (the reducing science). That is, Nagel’s condition of connectability must be satisfied. The statements that connect concepts of S_1 and S_2 are sometimes called bridge laws and sometimes called reduction functions. I shall employ the former term.

Just what the nature of bridge laws is is a problem which has exercised philosophers considerably (see Causey 1972 and Enc 1976),

but most of that debate can be ignored here. Whatever a bridge law is, it must allow for the derivation of something in the reduced science from something in the reducing science. If, for example, we want to reduce $Z \rightarrow Y$ to $A \rightarrow B$, it is not enough that $Z \rightarrow A$ and $Y \rightarrow B$. Nor is it enough that $A \rightarrow Z$ and $B \rightarrow Y$. In neither case does $Z \rightarrow Y$ follow from just $A \rightarrow B$ and the bridge laws. We need at least $Z \rightarrow A$ and $B \rightarrow Y$. A biconditional relation between Z and A , and B and Y would also do. I shall assume that the logically weakest conditions are permissible for bridge laws. If it turns out that stronger conditions are necessary, my arguments will not be overthrown.

The first kind of reduction I shall discuss is called strong type-type reduction¹ and is perhaps the kind of reduction Nagel had in mind. In strong type-type reduction there is a simple, one-one equivalence of concepts in the two sciences, such that the laws of the reduced science, S_2 , can be derived from the laws of the reducing science, S_1 . Such a reduction would go as follows:

$$\begin{array}{l} Ax \rightarrow By \\ Zx \rightarrow Ax \\ By \rightarrow Wy \\ \hline Zx \rightarrow Wy. \end{array}$$

No one, I trust, thinks that genetics can be reduced to molecular biology in this way. It is simply not reasonable to think that such a simple, one-one correspondence obtains between the predicates of genetics and molecular biology. The example of the lactose operon (in Section II) shows at least that there is more than one kind of gene at the molecular level. From the view of molecular biology, sections of DNA which are transcribed and translated must be seen as of a type distinct from sections of DNA which are not both transcribed and translated. Yet, we have seen (in Section II) that some members from both types are counted as genes. I take it, then, that strong type-type reduction of genetics is impossible.

The above objection to strong type-type reduction may be accommodated by what I shall call weak type-type reduction. Weak type-type reduction is like strong type-type reduction except that a many-one correspondence between the types of S_1 and S_2 is permitted. This

¹I have borrowed the terms "type-type reduction" and "token-token" reduction from Fodor (1975). My analysis of what constitutes these kinds of reduction is perhaps more detailed than Fodor's and may differ from what he had in mind.

probably is the sort of reduction Nagel had in mind. A weak type-type reduction could go as follows:

$$\begin{array}{l} (Cx \vee Ax) \rightarrow (By \vee Dy) \\ Zx \rightarrow (Cx \vee Ax) \\ (Dy \vee By) \rightarrow Wy \end{array}$$

$$Zx \rightarrow Wy.$$

Weak type-type reduction may fail for two reasons. The first of these is the case in which one or both of the two sciences does not contain laws which can serve to perform the reduction. It is impossible to deduce the laws of one science from the laws of another science if one of both of the sciences does not contain the appropriate laws. In the second case, weak type-type reduction may fail because the absence of proper bridge laws prevents reductions from occurring. I hold that both conditions obtain and hence that weak type-type reduction is not sufficient for description of the current relation between genetics and molecular biology. The problems with the laws in genetics and molecular biology will be argued immediately. Support for the claim that proper bridge laws are not available is deferred to the discussion of limited type-type reduction.

Whatever the correct analysis of scientific laws is, general agreement has it that scientific laws are universal truths (see Dretske 1977). This feature requires two comments. First, from the fact laws must be universally true it follows that any statement with a *ceteris paribus* clause appended is not a law (see Moline 1975). If a law is true only under certain, but not completely specified, conditions, then it is just not universally true under the conditions specified. The second point about the requirement of universality is that even statistical laws are universal generalizations (see Nagel 1961). Very roughly, I take it that the general form of a statistical law is as follows: $Px \rightarrow Qy$, with a certain probability. Notice: non-statistical laws can be seen as special cases of statistical laws, with the probability of Qy equal to 1; and every x which is Px has the same probability of bringing about a y which is Qy . Thus it is not a problem for a statistical law if some x 's which are Px bring about Qy and some do not, so long as each x which is Px has the same probability of so doing.

Having made these remarks about scientific laws I am prepared to discuss the question of whether laws in genetics or molecular biology are universally true. I shall restrict my attention to Mendel's Second Law, an obvious candidate for a law of genetics. For reasons of economy other putative laws of genetics—e.g. Mendel's First Law,

the Hardy-Weinberg Law—will not be discussed. I believe that an examination of them would not alter the conclusions I shall draw.

Mendel's Second Law, the law of independent assortment, is usually put in rather simple form by textbooks. The following is a decent approximation of the Second Law.

If x , y , and z are genes in a living, diploid organism which produces gametes and if x and y are an allelic pair and if $x \neq y \neq z$, then in the gametes produced only two combinations of these genes are possible, x and z , or y and z , and these two combinations will be present in equal proportions.

As stated, this law is not universally true, even as a statistical generalization. Genes close together on a single chromosome will not assort randomly during meiosis and are said to be linked. Thus the above formulation of Mendel's Second Law is not universally true, for it it were every gene would have an identical probability of assorting with every other gene not its allele. This is clearly false, as the cases of gene linkage show.

Perhaps Mendel's Second Law can be modified and reformulated in light of current understanding. A tempting way to do this would be to add "neither x nor y is linked with z " to the antecedent of the Law. The difficulty with this is that the result of this operation is a tautology rather than a scientific Law, unless " x and y are linked" can be unpacked in some way other than merely saying that genes which do not obey the Second Law are linked. There is a way to surmount this difficulty, but it has problems of its own. Linkage may be characterized as follows: x and z are linked if and only if x and z are on the same chromosome. The effect of this modification would be to restrict Mendel's Second Law to genes which are on different chromosomes. Our problems are not over, however. First, it may be doubted that 'chromosome' is a concept of genetics. Chromosomes may be identified through biochemical and cytological techniques, but not through breeding experiments alone, i.e. not through genetic techniques alone. There are two ways to go. Either the move to save the Second Law can be seen as begging the question of the reduction or it can be allowed that genetics and molecular biology share some concepts. Even granting that genetics and molecular biology share some predicates, difficulties persist.

In an important sense, counting 'chromosome' as a concept of both genetics and molecular biology does beg questions about reduction. Genetics in its pure, unreduced, and Mendelian form is the science devoted to the study of the effect of various sorts of breedings on the dispersal of phenotypic characters. From a purely genetic

point of view it matters not whether vitalism is true or false, whether genes are on chromosomes, whether life is carbon based or silicon based, or whether living things share any constituents with non-living things. A difficulty with genetics, as we have seen, is that one of its most basic generalizations cannot be law-like unless concepts from another science are imported. In our example, the concept is that of a chromosome and the other science is molecular biology. This is to say that the concept, 'chromosome', has been imported into genetics and has been connected with certain other terms in genetics. And this is to admit that some kind of reduction has occurred. Thus, while there is no reduction unless Mendel's Second Law is made law-like, this cannot be achieved unless the Second Law incorporates a term from the science doing the reducing. The question of reduction is begged by counting 'chromosome' as a concept of both genetics and molecular biology, unless we account for the introduction of 'chromosome' into genetics. Bridges (1916) indicates that 'chromosome' was not imported into genetics by a reduction of the kind we are considering (weak type-type reduction), but to argue this would take us too far afield.

The reductionist is not home free even if the argument of the previous paragraph is rejected and it is agreed that genetics and molecular biology unproblematically share the concept of the chromosome, for the modified Second Law remains not universally true. There are other causes of exception to the Second Law, for example meiotic drive and segregation distortion. We might try to accommodate these in the way we accommodated linkage. Meiotic drive is any process which results in alleles being represented in unequal proportions in the gametes. Adding this, however, to the Second Law creates a tautology. Meiotic drive must be unpacked, just as linkage had to be. There are two problems. First, any unpacking of 'meiotic drive' will inevitably be done in molecular terms. These terms will be less plausibly part of genetics than 'chromosome'. Second, how the unpacking is to be done is not known. But if it is unknown, we cannot do the reduction and this violates the assumption that reduction is occurring.²

The claim that Mendel's laws, and indeed most of the generalizations in biology, are not universally true is hardly original (see Fodor 1975, Goosens 1978, and Smart 1963). There are, nevertheless, several responses which might be made by someone defending weak type-type

²There is little reason to think that qualifying the Second Law to accommodate linkage, meiotic drive, and segregation distortion would give us a universal truth. Even with these qualifications, it does not seem we would have a law.

reduction. First, this sort of reductionist might claim that there are law-like generalizations in genetics other than the Second Law and that these generalizations can be reduced to molecular biology. In reply I say that the issue is not whether there are *some* universal truths in genetics which might be reduced to molecular biology, but whether the important and central generalizations in genetics are universal truths amenable to reduction. No one, so far as I know (including Schaffner in his 1976) can point to a list of the main and the universally true generalizations in genetics. Let alone producing one, the proponents of type-type reduction (of whatever sort) have yet to provide a roster of the generalizations in genetics which both are central and are prime candidates for eventual reduction.

A second kind of response to be made by a reductionist is to argue that genetics cannot now be reduced but that with further progress we can expect real laws to be discovered and these will be reduced. In addition, the reductionist could claim that surely these real laws will closely resemble significant generalizations currently in the body of genetics. Granting that progress in genetics will continue and that the generalizations of the future will resemble those of today, the second response remains inadequate. It forces us to abandon our intuition that reduction is and has been occurring. Giving the second response is to admit that reduction has not occurred, but that prospects seem good. This, of course, does not illuminate the present relation between genetics and molecular biology and does violate the assumption that reduction has been underway. Another problem with the second response is that it is not at all clear that the generalizations of genetics can be extended and made more precise without actually merging genetics and molecular biology. We would, I suppose, want to call such a merger a reduction, but it is not the sort of reduction we have been considering. From the above discussion of Mendel's Second Law, I would argue that the presence of meiotic drive and segregation distortion prevents the Second Law from being universally true. I would argue further that it is unlikely meiotic drive and segregation distortion can be sufficiently analysed so as to make the Second Law universally true without, in the meantime, importing so much of molecular biology into genetics that the two sciences will succumb to marriage. In Section IV, I propose an account of reduction which can easily accomodate this confluence of genetics and molecular biology.

One who believed in weak type-type reduction of genetics to molecular biology has a third response to my claim that central generalizations of genetics are not universal truths and hence not laws. The reductionist could claim that I have set my standards too

high for law-likeness and that we must simply accept the fact that laws in biology, as well as in other sciences, have exceptions.³ My reply to this response is to ask about the laws of the reducing science, in our case molecular genetics. Either the laws of molecular genetics have exceptions or they do not. In the latter case, I fail to see how the criterion of deducibility can be satisfied. In the former case, if the laws of molecular biology have exceptions it would be extremely unlikely that the laws of the two sciences had exceptions in exactly the same situations (see Fodor 1975 for a similar argument), and barring this possibility the condition of deducibility cannot be satisfied.

So much for laws in genetics. What are the laws of molecular biology? Certain generalizations in molecular biology are plausibly law-like, e.g. generalizations about melting points of various biologically important compounds. But these are not the laws we need to derive the generalizations of genetics. No one, so far as I know, has produced credible analogues to Mendel's laws or the Hardy-Weinberg law. Also, there is considerable slack in the generalizations of molecular biology. In double stranded DNA purines usually oppose pyrimidines, but not always. Under cellular conditions a double stranded DNA will make a complete rotation every 34 angstroms, except where it is twisted; every human cell contains about six feet of DNA. In sum, molecular biology does not contain the laws which would allow it to reduce genetics in a weak type-type fashion.

If even weak type-type reduction is impossible given the current status of genetics and molecular biology, what is the relation between them? A still weaker sort of reduction, which I shall call limited type-type reduction, might be possible. This seems to be the kind

³I am sympathetic to this view of laws. The following comment (with emphasis added by me) is, I believe, typical of biologists when they talk about laws.

Twenty years have passed since Sandler and Novitski . . . summarized the handful of cases of meiotic drive that were then known and predicted that more examples would be found, in a wider variety of species, as the genetic study of higher organisms became more precise and extensive. It therefore seems appropriate at this symposium to inquire whether their prediction has been validated.

The answer is yes, and the answer is no; yes because the number of known examples of meiotic drive has increased from a handful to more than twenty and because the number of species exhibiting the phenomenon has increased; no *because meiotic drive is apparently an unusual phenomenon. Mendel's law of segregation is still safe.* Nevertheless, the increase in the number of examples of meiotic drive has been impressive, so much so that we can now begin to consider how—or if—the phenomenon can be put to some practical use. (Hartl 1977, p. 64)

The difficulty, for the present, with accepting a theory of laws which allows that they can have exceptions is that this move does not seem promising for supporting any kind of type-type reduction. See my argument below.

of reduction that Hull, Ruse, and Schaffner have been debating. All that is required of limited type-type reduction is that the types of the reduced science be derived from the types of the reducing science. Thus, limited type-type reduction is compatible with there being no laws in either genetics or molecular biology. Note that if limited type-type reduction fails, then so must strong and weak type-type reduction. The question of limited type-type reduction is nearly coterminous with the question of whether adequate bridge laws are possible.

David Hull has argued (1974 and 1976) against what I have called weak type-type reduction of genetics to molecular biology. His argument, however, applies to what I have called limited type-type reduction and can be reconstructed so that it deals explicitly with this kind of reduction. The argument is approximately as follows.

Let *S2* be under consideration for reduction to *S1*.

1. Deduction of a science, *S2'*, strongly analogous to *S2* from *S1* is necessary for limited type-type reduction.
2. One-many and many-many relations exist between many terms and predicates of molecular biology and genetics; hence deduction is impossible given the current terms of genetics.

3. Therefore, limited type-type reduction is impossible given current meanings of the terms and predicates of genetics and molecular biology.
4. Furthermore, any science which is deducible from molecular biology will not be strongly analogous to genetics as we know it.

5. Therefore, genetics cannot be reduced to molecular biology, in any limited type-type sense of reduction.

For further discussion of points (4) and (5) above, see Hull (1974 and 1976), Ruse (1976), and Schaffner (1976). I shall not directly discuss them further.

The reason why one-many and many-many relations between the terms and predicates of molecular biology and genetics will not do is that we want to deduce statements of genetics from molecular biology. If one-many or many-many relations exist, we shall at best be able to deduce statements such as "*P* is a gene or *P* is a *K*" where *K* is something other than a gene. This sort of statement is not part of the science of genetics.

I want to corroborate Hull's argument with one of my own. Partly because of restrictions on space, I shall not argue that every type in genetics (or even the central ones) cannot be reduced in a limited

type-type sense. My argument shall be restricted to the concept of the gene, surely a central one in genetics. For discussion of other types in genetics see Hull (1974 and 1976), Ruse (1976), and Schaffner (1976). The argument is as follows.

1. 'Gene' is a type in genetics.
 2. For limited type-type reduction of a type in genetics to molecular biology to occur there must not be one-many or many-many relations between the types in molecular biology and the types in genetics.
 3. A many-many relation exists between the types in molecular biology and 'gene', a type in genetics.
-
4. Therefore, 'gene' cannot be reduced to molecular biology in a limited type-type fashion.

The first premise is obviously true; Mendel's laws are about a certain kind of thing, genes. I have already argued for the second premise. What remains, then, is to demonstrate the truth of the third premise, since the argument is valid.

To be shown is that there are some individuals in the set of genes which belong to no type in molecular biology all of whose members are genes. This can be done conclusively only for the current states of genetics and molecular biology. Of course, genetics may be altered in future, but I take it that some sort of reduction has been occurring and our problem is to explicate what that is. I shall argue for (3) by examining some types in molecular biology and showing that they will not work for limited type-type reduction of the concept of the gene.

a.) We cannot infer from the fact that something is DNA in a living organism to the claim that it is a gene or part of a gene. Although all the *E. coli* genes known so far are composed of DNA, not all the DNA is part of some gene, even if only chromosomal DNA is counted. Especially in eucaryotes there is every reason to think that not every bit of chromosomal DNA is part of some gene. It seems that some chromosomal DNA plays a purely structural role in eucaryotic chromosomes.

Still, we know that genes are composed of DNA. Are there any types of DNA which disjunctively are co-extensional with the set of genes? I do not believe so, given the current state of molecular biology. Here the discussion of the genes of the lactose operon is helpful. Consider several candidates.

b.) That every gene is identical with some length of DNA which is transcribed onto mRNA and translated into a polypeptide is initially

plausible. True enough, the *i*, *z*, *a*, and *y* genes of the lactose operon are transcribed and translated (under standard conditions), and it may be safe to say that all sections of DNA which are transcribed and translated are genes or parts of genes. Still, reduction is not served, and for two reasons. First, not all genes are transcribed and translated. As we have seen, such is the case with the *p* and *o* genes of the lactose operon. Second, even if all genes were sections of DNA which under standard conditions were transcribed and translated, more would be needed to specify a limited type-type reduction, for the only conclusion to follow from this is that every length of DNA transcribed and translated was a gene or was part of a gene. Manifestly, 'gene' and 'part of a gene' are distinct concepts. Again, we are left with a many-many relation between molecular biology and genetics.

c.) As a third effort, it might seem promising to include as genes all segments of DNA which are merely transcribed onto mRNA. Such a move would permit us to account for the *o* gene of the lactose operon but not for the *p* gene. Also, the second objection to suggestion (b) obtains in the present case.

d.) A fourth suggestion, designed to accomodate the *p* gene of the lactose operon, is more difficult to come by. The *p* gene is not transcribed onto RNA, but this category—not transcribed onto RNA—can hardly do for the purposes of reduction. There are many segments of DNA which are not transcribed and which are neither genes nor parts of genes. Perhaps we ought to narrow the category to include only those lengths of DNA which as a whole are not transcribed but which play a role in regulating transcription of other segments of DNA. This will not do either. There is no reason to believe that such a category includes only genes and not segments of DNA which are not genes. And we still have the problem of distinguishing between a whole gene and a part of a gene.

e.) I can think of only one other plausible suggestion for finding types in molecular biology including all and only the genes of genetics. Perhaps certain kinds of nucleotide sequences will suffice for the purposes of reduction. Perhaps, for example, all and only genes are segments of DNA which end in certain ways. This is tempting because it is known that certain triplets of nucleotides (UAA, UAG, and UGA) are read as punctuation marks in protein synthesis. Even so, the suggestion will not do because not all genes have ends which are defined by one of these three stop codons. The *p* gene is not transcribed and as expected does not end with a stop codon. Interestingly, it overlaps the *o* gene, which might be taken to suggest that the *p* and *o* genes ought not be distinguished at the molecular

level. Here is a further problem for reduction, one on which I shall not dwell.

The suggestion that certain kinds of DNA nucleotide sequences correspond to genes has been undermined considerably by recent developments in eucaryotic molecular genetics (see Darnell 1978 for a review). It now appears that in eucaryotic systems DNA is often transcribed onto mRNA which is then spliced and reassembled before it is translated into protein. The lesson being drawn from this is that, in eucaryotes at least, a single gene does not need to be a continuous section of DNA. Given this, what determines where a gene begins and ends on the DNA are the structures of the enzymes which mediate the splicing and reconstruction of the mRNA, and not any inherent kind of structure in the gene itself.

There is another reason why the present suggestion will not suffice for limited type-type reduction. Even if suggestion (e) should prove correct, it has not so far and this counters the pre-analytic assumption that reduction is occurring between genetics and molecular biology.

The conclusion that limited type-type reduction of the most central concept in genetics to molecular biology has not taken place, even in part, is forced upon us.

IV

We now have something of a dilemma. On one hand it seems that reduction must be impossible for genetics and molecular biology, and on the other hand we have our pre-analytic belief that reduction is underway. After all, we know that genes are made of DNA and we even know the precise molecular structure of two genes, the *p* and *o* genes of the lactose operon. If this is not reduction, what is?

Our problem can be solved by distinguishing yet another kind of reduction. Following Fodor (see 1975) again, I shall call this kind of reduction token-token reduction. When token-token reduction occurs, individuals described in the language of one science are identified with individuals described in the language of another science. This is a weak sort of reduction; it is implied by but does not imply every kind of reduction I have discussed. The following may count as reduction functions or bridge laws:

1. (for some x, y)($Zx \cdot Ay \cdot x = y$)
2. $M = (\text{some } x \text{ such that } Ax)$
3. $N = (\text{some } x \text{ such that } Zx)$
4. $N = M$

where M is an individual constant of S_2 ; N an individual constant of S_1 ; A a predicate of S_1 ; and Z a predicate of S_2 .

Some statements with one of these forms are trivial or uninteresting. Looking at (1), no one recently has doubted that some gene is identical with some molecular entity. On the other hand, the discovery that some gene is identical with some length of DNA was momentous and has the form of (1). I suggest that interesting reduction functions be distinguished from uninteresting statements of the same form on informal grounds. What these grounds are is a difficult but not, I believe, insurmountable problem. And it is a problem for another paper.

If the range of the variables and M and N in (1)–(4) may include properties, then (1)–(4) will suffice for property reduction. For example, the property of having the trait of sickle cell anemia is (in some individuals) identical with the property of having a certain kind of defective hemoglobin.

An interesting feature of token-token reduction is that the formal condition for it does not distinguish between the reducing science and the reduced science. Identity is a symmetrical relation. There is, however, no reason why the reduced and the reducing sciences cannot be informally distinguished. A plausible way in which this might be done is to stipulate that all the individuals of the reduced science can be identified with individuals of the reducing science, but not vice versa.

What is obvious is that token-token reduction of genetics to molecular biology is partially completed. Returning to the example of the lactose operon, it has been discovered that at least some individual p genes of *E. coli* lactose operons are one and the same as a certain sequence of nucleotide pairs in double stranded DNA. A similar story can be told of the o gene. Long ago it was discovered that some genes are identical with some lengths of DNA. I have already mentioned the case of sickle cell anemia. Other examples are legion.

It might be asked what good a partial token-token reduction does to the cause of science. The answer is that it is difficult to imagine how we might obtain an understanding of the mechanisms underlying events described in S_2 without some token-token reduction of S_2 to S_1 (See Enc 1976, pp. 305–6; Fodor 1975, pp. 9–26). A first step in understanding the mechanisms of how genes work is to discover that some genes are made of DNA, and to do so is to effect a partial token-token reduction of genetics to molecular biology. In the case of the lactose operon, very little can be understood of how the i , p , and o genes function until some token-token reduction is achieved. Explanation of why i^+ is dominant to i^- (in merozygotes) is not

possible purely within genetics. Dickson (1975) confirms these points. A mechanism is proposed in that paper for how the *p* and *o* genes function. The fact that some *p* and *o* genes were discovered to have certain base sequences is important evidence for the model.

There are at least two problems with token-token reduction. First is the difficulty of individuating concepts in *S1* and *S2*, and in comparing them. There is no guarantee they will be commensurable. My reply is that certainly this is a legitimate worry, calling for additional philosophical analysis. However that may be, in the case of genetics and molecular biology, this problem has in fact been solved well enough that we can with great confidence say that genes are on chromosomes, that genes are made of DNA, that the *p* gene of the lactose operon has a certain DNA base sequence, and so on.

A second difficulty with token-token reduction is that it may not appear to capture the generality of the reduction which seems preanalytically to be taking place. We know not only that some genes are on chromosomes but that almost all of them are. I would point out that reduction functions, such as those above, which use existential quantifiers can achieve a great deal of generality by being applied to many distinct individuals. For example, in humans most (but not all) males have exactly two sex chromosomes, *X* and *Y*. This fact can be represented in the form of (1), above, where *Z* is the property of being a human male and *A* is the property of having one *X* and one *Y* chromosome. Generality is achieved by pointing out that in fact very many distinct individuals share these two properties. If the arguments presented above on type-type reduction are correct, nothing stronger can be achieved in the connection between genetics and molecular biology, at least for some of the main concepts in the two sciences.

If the argument of this section is correct, understanding the mechanisms of events in *S2* is facilitated by token-token reduction of *S2* to *S1*. But token-token reduction is possible without type-type reduction. Indeed, a complete token-token reduction of *S2* to *S1* is possible without any type-type reduction. Such a reduction would greatly illuminate *S2*. One wonders what more ought be expected of reduction.

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