Reductionism in a Historical Science*

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1. Introduction. Reductionism is a metaphysical thesis, a claim about explanations, and a research program. The metaphysical thesis reductionists advance (and antireductionists accept) is that all facts, including all biological facts, are fixed by the physical and chemical facts; there are no nonphysical events, states, or processes, and so biological events, states and processes are "nothing but" physical ones. The research program can be framed as a methodological prescription which follows from the claim about explanations. Antireductionism does not dispute reductionism's metaphysical claim, but rejects the explanatory claim and so the methodological moral. To a first approximation what reductionists and antireductionists disagree about is whether explanations in functional biology can be or need to be explained or completed or perhaps replaced by explanations in terms of molecular biology.[1] And this disagreement over the adequacy of explanations in functional biology drives a significant methodological disagreement with consequence for the research program of biology.

The reason is simple: if the aim of science is explanation and explanations in functional biology are adequate, complete, and correct, then the

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1. Let us distinguish functional biology and molecular biology. Functional biology is the study of phenomena under their functional kind-descriptions: for example, organism, organ, tissue, cell, organelle, gene. Molecular biology is the study of certain classes of organic macromolecules. As I shall show below, this distinction is not entirely satisfactory, for many of the kinds identified in molecular biology are also individuated functionally. What makes a kind functional is that its instances are the products of an evolutionary etiology: a history of random variation and natural selection. Since natural selection operates at the macromolecular level some of its kinds will be functional too. But the functional/molecular distinction is convenient one which reflects wide-spread beliefs about a real division in the life sciences.

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methodological prescription that we must search for molecular completions, corrections, or foundations of these functional explanations in molecular processes will be unwarranted. Consequently, molecular biology need not be the inevitable foundation for every compartment of functional biology. If the aim of science is explanation, and functional explanations are either false or incomplete and molecular explanations either (more) correct or (more) complete, then biology must act on the methodological prescription that we should seek macromolecular explanations. If at its explanatory base, all biology is molecular biology, then all biologists, or at least all those who seek complete and correct explanations, will have eventually to be molecular biologists.[2]

Biologists are unlikely to be interested in philosophical disputes about the nature of explanation. Regrettably, they will have to be, if they wish to decide intelligently about whether to embrace a reductionist or nonreductionist methodology. For the dispute between reductionists and antireductionists turns very largely on the nature of scientific explanation. If there is no consensus on the nature of explanation, there will be no way to adjudicate the dispute between reductionism and antireductionism.

Matters used to be clearer, as a bit of the history of philosophy of biology will show.

2. What Was Reductionism? For the record, let us recall how philosophers supposed reduction was to proceed, and some of the qualifications added to the original model in order to bring it into contact with the history of science. Reduction is an inter-theoretical relation between theories. In the Anglo-Saxon *locus classicus*, Ernest Nagel's *Structure of Science* (1961), reduction is characterized by the deductive derivation of the laws of the reduced theory from the laws of the reducing theory. The deductive derivation requires that the reduced theory share meanings with the terms of the reducing theory. Though often stated explicitly, this second requirement is actually redundant as valid deductive derivation presupposes univocality of the language in which the theories are expressed. However, as exponents of reduction noted, the most difficult and creative part of a reduction is establishing these connections of meaning, i.e. formulating "bridge principles", "bi-lateral reduction sentences", "coordinating definitions." Thus it was worth stating the second requirement explicitly.

In posing the question above I use the past tense advisedly. For reduc-

2. Some antireductionists might wish to saddle reductionism with the indefensible thesis that all biology is molecular biology, that molecular biology provides not only the explanans (what does the explaining), but also uncovers all the facts to be explained (the explananda). This is not reductionism, for it affords no role to functional biology. It is some kind of eliminativism no reductionist has ever advocated.

tionism, as a doctrine received from the logical positivists and their post-positivist empiricist successors, is a dead letter, at least in biology. An account of why this is so for physical science is relegated to a footnote.[3]

3. Early on in discussions of reduction Kenneth Shaffner (1964) observed that reduced theories are usually less accurate and less complete in various ways than reducing theories, and therefore incompatible with them in predictions and explanations. Accordingly, following Shaffner, the requirement was explicitly added that the reduced theory needs to be "corrected" before its derivation from the reducing theory can be effected. This raised a problem which became non-trivial in the fallout from Thomas Kuhn's *Structure of Scientific Revolutions* (1961), and Paul Feyerabend's "Reduction, Empiricism and Laws" (1964). It became evident in these works that "correction" sometimes resulted in an entirely new theory, whose derivation from the reducing theory showed nothing about the relation between the original pair. Feyerabend's examples were Aristotelian mechanics, Newtonian mechanics, and Relativistic mechanics, whose respective crucial terms, 'impetus' and 'inertia', 'absolute mass' and 'relativistic mass' could not be connected in the way reduction required.

No one has ever succeeded in providing the distinction that reductionism required between 'corrections' and 'replacements.' Thus, it was difficult to distinguish reduction from replacement in the crucial cases that really interested students of reduction. This was a matter of importance because of reductionism's implicit account of scientific change as increasing approximation to more fundamental truths. It was also Shaffner who coined the term "layer-cake reduction" to reflect the notion that synchronically less fundamental theories are to be explained by reduction to more fundamental theories—at the basement level some unification of quantum mechanics and the general theory of relativity, above these physical and organic chemistry, then molecular biology and functional biology, at the higher levels psychology, economics, and sociology. Synchronic reduction is supposed to be explanatory because on the account of explanation associated with reduction, the Deductive-Nomological (D-N) model, explanation was logical deduction, and the explanation of laws required the deduction of laws from other laws. Synchronic reduction is mereological explanation, in which the behavior of more composite items described in reduced theories is explained by derivation from the behavior of their components by the reducing theory. Thus, reduction is a form of explanation. Diachronic reduction usually involves the succession of more general theories which reduce less general ones, by showing them to be special cases which neglect some variables, fail to measure coefficients, or set parameters at restricted values. As the history of science proceeds from the less general theory to the more general, the mechanism of progress is the reduction of theories. But if there is no way to distinguish reduction from replacement, then the incommensurablity of replacing theories makes both the progressive diachronic and synchronic accounts of inter-theoretical relations impossible ideals.

More fundamentally, reductionism as a thesis about inter-theoretical explanation was undermined by the eclipse of the Deductive-Nomological model. Once philosophers of science began to doubt whether deduction from laws was sufficient or necessary for explanation, the conclusion that inter-theoretical explanation need take the form of reduction was weakened.

Finally, reductionism is closely tied to the so-called syntactic approach to theories, an approach which treats theories as axiomatic systems expressed in natural or artificial languages. Indeed, "closely tied" may be an understatement, since deduction is a syntactic affair, and is a necessary component of reduction. Once philosophers of science began to take the semantic approach to theories seriously, the very possibility of re-

To the general philosophical difficulties which the post-positivist account of reduction faced, biology provided further distinct obstacles. To begin with, as Hull first noted (1973), the required "bridge principles" between the concept of gene as it figures in population biology and as it figures in molecular biology could not be effected. And none of the ways philosophers contrived to preserve the truth of the claim that the gene is nothing but a (set of) string(s) of nucleic acid bases succeeded in providing the systematic link between these two 'types' required by a reduction. There is of course no trouble identifying 'tokens' of the population biologist's genes with 'tokens' of the molecular biologist's genes. But token-identities won't suffice for reduction, even if they are enough for physicalism to be true.

The second problem facing reductionism in biology is the absence of laws, either at the level of the reducing theory or the reduced theory. If there aren't any laws in either theory, there is no scope for reduction at all. Understanding the reason why there are no laws anywhere within the various subdisciplines of biology (i.e., beyond the theory of natural selection) is not only essential for understanding why post-positivist reductions are impossible, but equally essential to understanding the problems of antireductionism, and vital for framing any alternative notion of either thesis.

That (with one set of exceptions to be discussed in section 4 below) there are no laws in biology is now widely recognized among philosophers of biology, although some philosophers have responded to this realization by redefining the concept of 'law' so that some biological statements may continue to be so-called. (Sober 1993; Lange 1995)

The absence of laws in biology reflects some fundamental and ineliminable facts about the biological realm and the scientific study of that realm. To begin with, individuation of types in biology is almost always via causal role, and in particular via function. For instance to call something a wing, or a fin, or a gene is to identify it in terms of its function. But biological functions are naturally-selected effects. That is, Larry Wright's (1976) analysis of function as etiological is correct in broad outline. And natural selection for adaptations—i.e., environmentally appropriate effects—is blind to differences in physical structure that have the same or roughly similar effects.

Natural selection "chooses" variants by *some of their effects*, those

duction became moot. For the semantic approach treats theories as families of models, and models as implicit definitions, about which the only empirical question is whether they are applicable to phenomena. For reduction to obtain among semantically characterized models requires an entirely different conception of reduction, and whether such a conception would capture anything of interest about inter-theoretical relations is questionable.

which fortuitously enhance survival and reproduction. When natural selection encourages variants to become packaged together into larger units, the adaptations become functions. Selection for adaptation and function kicks in at a relatively low level in the organization of matter. As soon as molecules develop the disposition chemically, thermodynamically, or catalytically to encourage the production of more tokens of their own kind, natural selection comes into force. To employ vocabulary due to Dawkins (1983) and Hull (1989), at this point in the aggregation of matter, replicators and interactors (or vehicles) first appear. As a result of purely physical processes some molecules become replicators—template or catalyze or otherwise encourage the production of copies of themselves—and these molecules interact with the environment so that changes in them (mutations) will result in changes in their rates of replication in their environments. Among such replicating and interacting molecules, there are frequently to be found multiple *physically distinct* structures with some (nearly) identical rates of replication, different combinations of different types of atoms and molecules that are about equally likely to foster the appearance of more tokens of the types they instantiate. This structural diversity explains why no simple identification of molecular genes with the genes of population genetics, of the sort post-positivist reduction requires, is possible More generally, the reason there are no laws in biology is thus the same reason there are no bridge-principles of the sort post-positivist reduction requires (one might have expected this consequence: bridge principles are supposed to be laws).

It is the nature of any mechanism that selects for effects, that *it cannot discriminate between differing structures with identical effects.* And functional equivalence combined with structural difference will always increase as physical combinations become larger and more physically differentiated from one another. Moreover, perfect functional *equivalence* isn't necessary. Mere functional similarity will do. Since selection for function is blind to differences in structure, there will be no laws in any science which, like biology, individuates kinds by selected effects, that is by functions. A law in functional biology will have to link a functional kind either with an other functional kind, for example, "all butterfly wings have eyespots" or a structural kind, "all eyespots are composed of proteins". But neither of these statements can be a strict law, because of the blindness of natural selection (which forms functional kinds) to structure (which will therefore heterogeneously realize functional kinds). The details of this argument are relegated to a footnote.[4]

4. To see why there can be no strict laws in biology consider the form of a generalization about all *F*s, where *F* is a functional term, like gene, or wing, or belief, or clock, or prison, or money, or subsistence farming. The generalization will take the form $(x)[Fx \rightarrow Gx]$, a law about *F*s and *G*s. Gx will itself be either a structural predicate or

Any science in which kinds are individuated by causal role will have few if any exceptionless laws. But of course, many will ague that neither biology nor reduction requires strict laws. Non-strict *ceteris paribus* laws will suffice. But there are no non-strict laws in biology either. The reason is that what makes for the allegedly *ceteris paribus* claims of physics does not obtain in biology. In physics there are a finite (indeed small) number of forces—mechanical, electromagnetic, thermodynamic—which all work together to produce actual outcomes we seek to explain. To the extent a text-book generalization of mechanics, like $F = gm_1m_2/d^2$, is silent on these other forces, it is not a completely true description of physical processes, but rather a *ceteris paribus* law. There may perhaps be what Cartwright (1983) calls "super-laws", which include the finite number of forces actually operative in nature. These will in effect be strict laws. But in biology the role of natural selection does not limit the number of interfering forces that would turn a *ceteris paribus* law into a "super" or strict law. The reason is to be found in the role of the environment in setting adaptational or design problems for evolving lineages to solve. At a relatively early stage in evolution these design-problems take on the reflexive character of what Dawkins and others have called "arms races," dynamic strategic competitions in which every move generates a counter-move so that conditions are never constant and *ceteris* is never *paribus.*

Ever since Darwin's focus on artificial selection it has been recognized that in the evolution of some species, other species constitute the selective

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a functional one. Either it will pick out *G*s by some physical attribute common to them, or *Gx* will pick out *G*s by descriptions of one of the causes or effects that everything in the extension of *Gx* possesses. But there is no physical feature common all items in the extension of *Fx: Fx*s constitute a physically heterogeneous class since the members have been selected for their effects. So *G* cannot be a structural predicate. Of course some structural feature may be shared by all of the members of *F*. But it will not be a biologically interesting one. Rather it will be a property shared with many other things, like mass or electrical resistance—properties which have little or no explanatory role with respect to the behavior of members of the extension of *Fx*. For example the exceptionlessly true generalization that "all mammals are composed of confined quarks" does relate a structural property, quark confinement, to a functional one, mammality, but is not a law of biological interest.

The existence of a functional property different from *F* that all items in the extension of the functional predicate *Fx* share must be highly improbable. If *Fx* is a functional kind then, owing to the blindness of selection to structure, the members of the extension of *Fx* are physically diverse. As such, any two *F*s have non-identical (and usually quite different) sets of effects. Without a further effect common to *F*s, selection for effects cannot produce another selected effect; it cannot uniformly select all members of *F* for some further adaptation. Thus, there is no further functional kind for all *F*'s to share in common.

Whether functional or structural, there will be no predicate *Gx* that is linked in a strict law to *Fx*.

force channeling their genetic changes. The interaction of predator and prey manifest the same relationship. Since the importance of frequency-dependent selection became apparent, it has been recognized that an interbreeding population can be an environmental force influencing its own evolutionary course.

Competition for limited resources is endemic to the biosphere. Any variation in a gene, individual, line of descent, or species which enhances fitness in such a relentlessly competitive environment will be selected for. Any response to such a variation within the genetic repertoire of the competitor gene, individual, lineage, or species, will in turn be selected for by the spread of the first variation, and *so on.* One system's new solution to a design problem is another system's new design problem. If the "space" of adaptational "moves" and "counter-moves" is very large, and the time available for trying out these stratagems is long enough, every regularity in biology about functional kinds will be falsified (or turned into a stipulation) eventually.

What this means of course is that any functional generalization in biology will be a *ceteris paribus* generalization in which, over evolutionary time scales, the number of exceptions will mount until its subject becomes extinct. Take a simple example, such as "butterflies have eyespots." The explanation for why they do is that eyespots distract birds from butterflies' more vulnerable and more nutritious parts, and provide camouflage when they give the appearance of the eyes of owls that prey on birds. This strategy for survival can be expected in the long run to put a premium on the development of ocular adaptations among birds, say the power to discriminate owl eyes from eyespots, that foil this stratagem for butterflies. This in turn will lead either to the extinction of eyespot butterflies or the development of still another adaptation to reduce predation by birds, say the development of an unappetizing taste, or shift in color to the markings of a butterfly that already tastes bad to birds. And in turn this stratagem will lead to a counter-stroke by the bird lineage. The fantastic variety of adaptational stratagems uncovered by biologists suggests that there is a vast space of available adaptive strategies among competing species, and that large regions of it are already occupied. The upshot is that to the extent that general laws must be timeless truths to which empirical generalizations approximate as we fill in their *ceteris paribus* clauses, no such laws are attainable in biology because we can never fill in these clauses.

Notice this result obtains as much for molecular biology as it does for functional biology. Because the kinds of molecular biology are also functional, even at the level of the biochemical, natural selection's persistent exploration of adaptational space makes for lawlessness at the level of macromolecules as well. Consider three examples of generalizations in molecular biology once held to be strict laws and now found to have

exceptions: all enzymes are proteins; hereditary information is carried only by nucleic acids; the central dogma of molecular genetics: DNA is transcribed to RNA and RNA is translated to protein. It turns out that RNA catalyzes its own self-splicing, that Prions (proteins responsible for Mad Cow Disease) carry hereditary information, and the retroviruses carry their own hereditary material in RNA and transcribe it to DNA. These exceptions to the relevant generalizations emerged through the operation of natural selection—finding strategies in adaptational space that advantage one or another unit of selection in the face of stratagems employed by others.

If there are no laws in biology then biological theories cannot be related to one another in ways that satisfy the post-positivist conception of reduction. For the bridge principles that this formulation of reduction requires are laws, and the derivations it consists in require laws. Without recourse to laws, reductionism must be rejected or reformulated.

3. What Was Antireductionism? If antireductionism were merely the denial that post-positivist reduction obtains among theories in biology, it would be obviously true (in part for reasons outlined in Footnote 2). But antireductionism is not merely a negative claim. It is the thesis that a) there are generalizations at the level of functional biology, b) these generalizations are explanatory, c) there are no further generalizations outside of functional biology which explain the generalizations of functional biology, and d) there are no further generalizations outside functional biology which explain better, more completely, or more fully, what the generalizations of functional biology explain.

All four components of antireductionism are daunted by at least some of the same problems that vex reductionism: the lack of laws in functional biology and the problems facing a nomic subsumption-account of explanation. If there are no laws and/or explanation is not a matter of subsumption, then antireductionism is false too. But besides the false presuppositions antireductionism may share with reductionism, it has distinct problems of its own.

To see the distinctive problems for antireductionism, consider a paradigm of putative irreducible functional explanation advanced by an antireductionist, Philip Kitcher. The explanadum is

(G) Genes on different chromosomes, or sufficiently far apart on the same chromosome, assort independently. (Kitcher 1999, 199)

The antireductionist proffers an explanans for (G), which we shall call (PS):

(PS) Consider the following kind of process, a *PS*-process (for *pairing*

and *separation*). There are some basic entities that come in pairs. For each pair, there is a correspondence relation between the parts of one member of the pair and the parts of the other member. At the first stage of the process, the entities are placed in an *arena.* While they are in the arena, they can exchange segments, so that the parts of one member of a pair are replaced by the corresponding parts of the other members, and conversely. After exactly one round of exchanges, one and only one member of each pair is drawn from the arena and placed in the *winners box*.

In any PS-process, the chances that small segments that belong to members of different pairs or that are sufficiently far apart on members of the same pair will be found in the winners box are independent of one another. (G) holds because the distribution of chromosomes to gametes at meiosis is a PS-process.

This I submit is a full explanation of (G), and explanation that prescinds entirely from the stuff that genes are made of. (Kitcher 1999, 199–200)

Leave aside for the moment the claim that (PS) is a full explanation of (G), and consider why, according to the antireductionist, no molecular explanation of (PS) is possible.

The reason is basically the same story we learned above about why the kinds of functional biology cannot be identified with those of molecular biology. Because the same functional role can be realized by a diversity of structures, and because natural selection encourages this diversity, the full macromolecular explanation for (PS) or for (G) will have to advert to a range of physical systems that realize independent assortment in many different ways. These different ways will be an unmanageable disjunction of alternatives so great that we will not be able to recognize what they have in common, if indeed they do have something in common beyond the fact that each of them will generate (G). Even though we all agree that (G) obtains in virtue only of macromolecular facts, nevertheless, we can see that because of their number and heterogeneity these facts will not explain (PS), still less supplant (PS)'s explanation of (G), or for that matter supplant (G)'s explanation of particular cases of genetic recombination. This is supposed to vindicate antireductionism's theses that functional explanations are complete and that functional generalizations cannot be explained by non-functional ones, nor replaced by them.

But this argument leaves several hostages to fortune. Begin with (G). If the argument of the previous section is right, (G) is not a law at all, but the report of a conjunction of particular facts about a spatiotemporally restricted kind, "chromosomes" of which there are only a finite number extant over a limited time period at one spatio-temporal region (the

Earth). Accordingly, (G) is not something which we can expect to be reduced to the laws of a more fundamental theory, and the failure to do so constitutes no argument against reductionism classically conceived, nor is the absence or impossibility of such a reduction much of an argument *for* antireductionism.

The antireductionist may counter that regardless of whether (G) is a generalization, it has explanatory power and therefore is a fit test-case for reduction. This however raises the real problem which daunts antireductionism. Antireductionism requires an account of explanation to vindicate its claims. Biologists certainly do accord explanatory power to (G). But how does (G) explain? And the same questions are raised by the other components of the antireductionist's claims. Thus, what certifies (PS)—the account of PS-processes given above—as explanatory? What prevents the vast disjunction of macromolecular accounts of the underlying mechanism of meiosis from explaining (PS), or for that matter from explaining (G) and indeed whatever it is that (G) explains?

There is one tempting answer, which I shall label, "explanatory Protagoreanism," the thesis that "some human or other is the measure of all putative explanations, of those which do explain and those which do not." Thus, consider the question of why a macromolecular explanation of (PS) is not on the cards? One answer is presumably that it is beyond the cognitive powers of any human contemplating the vast disjunction of differing macromolecular processes each of which gives rise to meiosis, to recognize that conjoined they constitute an explanation of (PS). Or similarly, it is beyond the competence of biologists to recognize how each of these macromolecular processes gives rise to (G). This is explanatory Protagoreanism. That the disjunction of this set of macromolecular processes implements PS-processes and thus brings about (PS) and (G) does not seem to be at issue. Only someone who denied the thesis of physicalism—that the physical facts fix all the biological facts—could deny the causal relevance of this vast motley of disparate macromolecular processes to the existence of (PS) and the truth of (G).

In fact, there is something that the vast disjunction of macromolecular realizations of (PS) have in common that would enable the conjunction of them fully to explain (PS) to someone with a good enough memory for details. Each was selected for because each implements a PS process and PS processes are adaptive in the local environment of the Earth from about the onset of the sexually reproducing species to their extinction. Since selection for implementing PS processes is blind to differences in macromolecular structures with the same or similar effects, there may turn out to be nothing else completely common and peculiar to all macromolecular implementations of meiosis besides their being selected for implementing PS processes. But this will be a reason to deny that the conjunction of all

these macromolecular implementations explain (PS) and/or (G), only on a Protagorean theory of explanation.

Antireductionists who adopt what is called an erotetic account of explanation, in preference to a unification account, a causal account, or the traditional D-N account of explanation, will feel the attractions of explanatory Protagoreanism. For the erotetic account of explanations treats them as answers to "why questions" posed about a particular occurrence or state of affairs, which are adequate (i.e. explanatory) to the degree they are appropriate to the background information of those who pose the why-question and to the degree that the putative explanation excludes competing occurrences or states of affairs from obtaining. Since it may be that we never know enough for a macromolecular answer to the question "Why does (G) obtain?" no macromolecular explanation of why (G) obtains will be possible. Similarly, we may never know enough for a macromolecular explanation of (PS) to be an answer to our question "Why do PS processes occur?" But this seems a hollow victory for antireductionism, even if we grant the tendentious claim that we will never know enough for such explanations to succeed. What is worse, it relegates antireductionism to the status of a claim about biologists, not about biology. Such philosophical limitations on our epistemic powers have been repeatedly breached in the history of science.

Antireductionists wedded to alternative, non-erotetic accounts of explanation, cannot adopt the gambit of a Protagorean theory of explanation in any case. They will need a different argument for the claim that neither (G) nor (PS) can be explained by its macromolecular supervenience base, and for the claim that (PS) does explain (G) and (G) does explain individual cases of recombination. One argument such antireductionists might offer for the former claim rests on a metaphysical thesis: that there are no disjunctive properties or that if there are, such properties have no causal powers. Here is how the argument might proceed: The vast motley of alternative macromolecular mechanisms that realize (PS) have nothing in common. There is no property—and in particular no property with the causal power to bring about the truth of (G)—which they have in common. Physicalism (which all antireductionists party to this debate embrace) assures us that whenever PS obtains, some physical process, call it P_i, obtains. Thus we can construct the identity (or at least the biconditional) that

$$(R)\ PS = P_1 \vee P_2 \vee \ \ldots\ \vee P_i \vee \ \ldots\ \vee P_m$$

where m is the number, a very large number, of all the ways macromolecular processes can realize PS processes.

The Protagorean theory of explanation tells us that (R) is not explanatory roughly because it's too long a sentence for people to keep in their

heads. A causal theory of explanation might rule out R as explaining PS on the ground that the disjunction, $P_1 \vee P_2 \vee \ldots \vee P_i \vee \ldots \vee P_m$, is not the *full* cause. This might be either because it was incomplete—there is always the possibility of still another macromolecular realization of PS arising—or because disjunctive properties just aren't causes, have no causal powers, perhaps aren't really properties at all. A unificationist theory of explanation (or for that matter a D-N account) might hold that since the disjunction cannot be completed, it will not effect deductive unifications or systematizations. Thus (PS) and (G) are the best and most complete explanations biology can aspire to. Antireductionist versions of all three theories, the causal, the unificationist, and the Protagorean need the disjunction in (R) to remain uncompleted in order to head off a reductionist explanation of (PS) and/or (G).

Consider the first alternative, that (R) is not complete, either because some disjuncts haven't occurred yet or perhaps because there are an indefinite number of possible macromolecular implementations for (PS). This in fact seems to me to be true, just in virtue of the fact that natural selection is continually searching the space of alternative adaptations and counter-adaptations, and that threats to the integrity and effectiveness of meiosis might in the future result in new macromolecular implementations of (PS) being selected for. But this is no concession to antireductionism. It is part of an argument that neither (PS) nor (G) report an explanatory generalization, that they are in fact temporarily true claims about local conditions on the Earth.

On the second alternative, (R) can be completed in principle, perhaps because there are only a finite number of ways of realizing a (PS) process. But the disjunction is not a causal or a real property at all. Therefore it cannot figure in an explanation of either (PS) or (G). There are several problems with such an argument. First, the disjuncts in the disjunction of $P_1 \vee P_2 \vee \ldots \vee P_i \vee \ldots \vee P_m$, do seem to have at least one or perhaps even two relevant properties in common: each was selected for implementing (PS) and causally brings about the truth of (G). Second, we need to distinguish predicates in languages from properties in objects. It might well be that in the language employed to express biological theory, the only predicate we employ that is true of every P_i is a disjunctive one, but it does not follow that the property picked out by the disjunctive predicate is a disjunctive property. Philosophy long ago learned to distinguish things from the terms we hit upon to describe them.

Arguing against the causal efficacy of some disjunctive properties, Sober has held that "disjunctive properties will appear to be causally efficacious only to the degree that their disjuncts strike us as subsuming similar sorts of possible causal processes" (Sober 1984, 94). Suppose we drop out the qualifications "will appear to be" and "strike us" as unsuited to

a question about whether disjunctive properties really are casually efficacious as opposed to seeming "to us" to be causally efficacious. If we adopt this principle, the question at issue becomes one of whether the disjunction of P_1 v P_2 v . . . v P_i v . . . v P_m subsumes similar sorts of causal processes, to which the answer seems to be that the disjunction shares in common the feature of having been selected for resulting in the same outcome—PS processes. Thus, the disjunctive predicate names a causal property, a natural kind. Antireductionists are hard pressed to deny the truth and the explanatory power of (R).[5]

Besides its problems in undermining putative macromolecular explanations of (PS), (G) and what (G) explains, antireductionism faces some problems in substantiating its claims that (PS) explains (G) and (G) explains individual cases of genetic recombination. The problems, of course, stem from the fact that neither (PS) nor (G) are laws, and therefore an account is owing of how statements like these can explain. This in fact is a problem that any revision of a thesis of reductionism must come to grips with as well. So perhaps we should turn to this problem directly. And then reformulate and reassess both reductionism and antireductionism as explanatory theses in its light.

4. Biology Is History (All the Way Down). The upshot is not simply that there are no laws, ergo neither reductionism nor antireductionism about laws is tenable in biology. The entire character of biology as a discipline reflects the considerations which make laws impossible (with an exception now to be noted). Functional kinds have etiologies that reflect natural selection operating on local conditions, and natural selection is constantly changing local conditions. This makes biology an essentially historical discipline. Any reformulation of the thesis of reductionism or of antireductionism will have to reflect this fact about the discipline if it is to have a ghost of a chance of illuminating the structure of biology or motivating a research program.

Evolution is a mechanism—blind variation and natural selection—that can operate everywhere and always throughout the universe. It obtains whenever tokens of matter have become complex enough to foster their own replication and variation so that selection for effects can take hold. Recent experiments in chemical synthesis suggest that this may not be an uncommon phenomenon.[6] Macromolecules are the initial replicators and

5. It will not escape readers that similar problems vex the antireductionist position in the philosophy of psychology. Thus, writers such as Fodor (1975, 9–25), who seek to underwrite the autonomy of intentional psychology from neuroscience also require an account of explanation to establish their claims. Mere invocation of the supervenience of the predicates of the "special sciences" is by itself insufficient.

6. See, for example, A. E. Winter 1996.

also the initial interactors or vehicles (though they are eventually selected for "building" larger interactors or vehicles: chromosomes, cells, tissues, organs, bodies, etc.).

However we express the mechanism of natural selection, its general principles operate exceptionlessly everywhere replicators and their vehicles appear. The principles of the theory of natural selection are the only real laws in biology.[7] Beyond the bare theory of natural selection itself, the rest of biology is a set of subdisciplines historically conditioned by the operation of natural selection on local circumstances during the history of the Earth. The functional individuation of biological kinds reflects the vagaries and vicissitudes of natural selection, since biological kinds are the result of selection over variation in order to solve design problems set by the environment. Possible solutions to the same problem are multiple and one biological system's solution sets a competing biological system's next design problem. Therefore, each system's environment varies over time in a way that makes all putative biological "generalizations" about these systems into historically limited descriptions of local patterns. Any subdiscipline of biology—from paleontology to developmental biology to population biology to physiology or molecular biology—can uncover at best historically conditioned patterns, owing to the fact that a) its kind vocabulary picks out items generated by a historical process, and b) its "generalizations" will always be overtaken by evolutionary events. Some of these "generalizations" will describe long-term and wide-spread historical patterns, such as the ubiquity of nucleic acid as the hereditary material; others of them will be local and transitory, such as the description of the primary sequence of the latest AZT-resistant mutation of the AIDS virus.

The apparent generalizations of functional biology are really spatiotemporally restricted statements about trends and the co-occurrence of finite sets of events, states, and processes. Beyond those laws which Darwin uncovered, there are no other generalizations about biological systems to be uncovered, at least none that connect kinds under biological—that is, functional—descriptions.

Biological explanation is historical explanation, in which the implicit laws are the principles of natural selection. This will be true even in molecular biology. To cite a favorite example of mine[8], the explanation of why DNA contains thymine while messenger mRNA, transfer tRNA, and

7. See Rosenberg (2001) for a set of sustained arguments for this claim, and against the accounts of putative biological laws and other principles offered by Sober 1993, Lange 1995, Kitcher 1993, and others, to supplement or supplant these laws in biological explanation.

8. First elaborated in Rosenberg 1985, Chapter 3.

ribosomal rRNA contain uracil is a thorough-going historical one. Long ago on earth DNA won the selective race for best available solution to the problem of high fidelity information storage; meanwhile RNA was selected for low cost information transmission and protein synthesis. Uracil is cheaper to synthesize than thymine, because thymine has a methyl group that uracil lacks. Cytosine spontaneously deaminates to uracil. Replication of DNA with uracil produced by deamination results in a point mutation in the conjugate DNA strand, since cytosine pairs with guanine while uracil and thymine both pair with adenine. A repair mechanism evolutionarily available to DNA removes uracils and replaces them with cytosines to prevent this point mutation. The methyl group on thymine molecules in DNA blocks the operation of this repair mechanism when it attempts to remove thymines. Employing this relatively costly molecule was a cheaper and/or more attainable adaptation than DNA's evolving a repair mechanism that could distinguish uracils that are not the result of cytosine deamination from those which are the result of deamination. So it was selected for. Meanwhile, the spontaneous deamination of cytosine to uracil on one out of hundreds or thousands of RNA molecules engaged in protein synthesis will disable it, but result only in a negligible reduction in the production of the protein it would otherwise build. Ergo, natural selection for economic RNA transcription resulted in RNAs employing uracil instead of thymine.

Notice how the explanation works: first, we have two "generalizations": DNA contains thymine, RNA contains uracil. They are not laws but in fact statements about local conditions on the Earth. After all, DNA can be synthesized with uracil in it and RNA can be synthesized with thymine. Second, the explanation for each appeals to natural selection for solving a design problem set by the environment. Third, tRNA, mRNA, and the various rRNAs are functional kinds, and they have their function as a result of selection over variation. Fourth, we can expect that in nature's relentless search for adaptations and counteradaptations, the retroviruses, in which hereditary information is carried by RNA, may come to have their RNAs composed of thymine instead of uracil if and when it becomes disadvantageous for retroviruses to maximize their rates of mutation. At this point of course the original generalizations will, like other descriptions of historical patterns, cease to obtain, but we will have an evolutionary explanation for why they do so, and we will be able to retain our original explanation for why these generalizations obtained about the composition of DNA and RNA during the period and in the places where they did so. In these respects, explanation in molecular biology is completely typical of explanation at all higher levels of biological organization. It advances historical explanation-sketches in which the principles of the theory of natural selection figure as implicit laws.

5. Reductionism in a Historical Science. In biology neither reductionism nor antireductionism can be theses about the explanation of laws, except perhaps about the explanation of the laws of natural selection. I say perhaps, because there may be parties to this dispute that will not grant nomological status to any principles of the theory of natural selection, and so cannot dispute whether there are any laws of this theory to be explained by more fundamental ones. (Brandon 1990, for example) It is not obvious among philosophers of biology that there are such laws of natural selection. But I have assumed as much above. Moreover, I have assumed that the laws of natural selection obtain just in virtue of chemical and physical regularities, since all it takes for replicators and interactors to be possible is that these physical laws obtain. Reductionists should welcome the addition of laws of natural selection to the explanatory store of a reductionistic approach to biology. On the other hand, it would be an easy vindication of antireductionism if such laws were not themselves accepted as physical principles explainable without remainder as the result of physical processes. For, as we have seen, every part of biology relies on natural selection to give content its functional individuation. If the generalizations of natural selection are irreducible, so is *all* of biology, including all of molecular biology—the part of biology to which reductionists propose to reduce the rest.

If reductionism is to be given a chance of being right, we must give it natural selection as at least a component of biology's reduction base in physical science.

Reductionism will have to be a thesis about the explanation of historical facts, some more general than others, but all of them ultimately the contingent results of general laws of natural selection operating on boundary conditions. Reductionism needs to claim that the only way to explain one historical fact is by appeal to other historical facts, plus some laws or other. If there are no laws in biology beyond the principles of the theory of natural selection, then the explanation of one historical fact by appeal to another will have to appeal to these laws and if necessary to other laws drawn from physical science. This might be viewed as a vindication of some form of reductionism, understood as the claim that explanations of biological phenomena are ultimately to be given by appeal to the operation of non-biological laws drawn from physical science. But it will be a hollow vindication of reductionism. There must be more to reductionism that the claim that evolutionary explanation is physical explanation.

To see what more there must be to reductionism, recall the distinction between two different kinds of explanatory tasks in biology: proximate and ultimate explanation. (Mayr 1981) Thus, the question "Why do butterflies have eye-spots?" may be the request for an adaptationalist explanation that accords a function in camouflage, for instance to the eye-spot

on butterfly wings, or it may be the request for an explanation of why at a certain point in development eye-spots appear on individual butterfly wings and remain there throughout their individual lives. The former explanation is an ultimate one, the latter a proximate one. Reductionism must be a thesis about both sorts of explanation. In fact, I shall suggest that reductionism is the radical thesis that ultimate explanations must give way to proximate ones and that these latter will be molecular explanations.

To expound its thesis about explanations, reductionism adduces another distinction among explanations. It is a distinction well known in the philosophy of history, a division of philosophy whose relevance to biology may now be apparent. The distinction is between what William Dray (1957) called "how-possibly explanations" and "why-necessary explanations." A why-necessary explanation effectively rebuts a presumption that the explanandum need not have happened, "by showing in the light of certain considerations (perhaps laws as well as facts), it had to happen." (Dray 1957, 161) How-possibly explanations show how something could have happened, by adducing facts which show that there is after all no good reason for supposing it could not have happened. "The essential feature of explaining how-possibly is . . . that it is given in the face of a certain sort of puzzlement." (165) The appeal to puzzlement makes it clear that Dray was sympathetic to erotetic models of explanation. Indeed, he went on to say: "These two kinds [of explanation] are logically independent in the sense that they have different tasks to perform. They are answers to different questions." (162) But Dray recognized an important asymmetrical relationship between them.

> It may be argued that although, in answer to a "how-possibly" question, all that need be mentioned is the presence of some previously unsuspected necessary condition of what happened . . . nevertheless, this does not amount to a full explanation of what happened. In so far as the explanation stops short of indicating sufficient conditions, it will be said to be . . . an incomplete explanation, which can only be completed by transforming it into an appropriate answer to the corresponding 'Why?'
>
> . . . Having given a how-possibly answer it always makes sense to go on to demand a why-necessary one, *whereas this relationship does not hold in the opposite direction.* (Dray 1957, 168; emphasis added)

Of course Dray's concern was human history, but the claims carry over into natural history. They enable us to see how reductionism might be vindicated, among biologists at least, as ultimate how-possibly explanation gives way to proximate why-necessary explanation. Let us see how.

Consider the ultimate explanation for eyespots in the species *Precis coenia*. To begin with, notice there is no scope for explaining the law that

butterflies have eye-spots, or patterns that may include eye spots, scalloped color patterns, or edge-bands. There is no such law to be explained.[9] There are however historical facts to be explained.

The ultimate explanation has it that eyespots on butterfly and moth wings have been selected for over a long course of evolutionary history. On some butterflies these spots attract the attention and focus the attacks of predators onto parts of the butterfly less vulnerable to injury. Such spots are more likely to be torn off than more vulnerable parts of the body, and this loss does the moth or butterfly little damage, while allowing it to escape. On other butterflies, and especially moths, wings and eye spots have also been selected for taking the appearance of an owl's head, brows, and eyes. Since the owl is a predator of those birds which consume butterflies and moths, this adaptation provides particularly effective camouflage.

Here past events help to explain current events via implicit principles of natural selection. Such ultimate explanations have been famously criticized as "just-so" stories, allegedly too easy to frame and too difficult to test. (Gould and Lewontin 1979) Though its importance has been exaggerated, there is certainly something to this charge. Just because the available data show that eyespots are wide-spread does not guarantee that they are adaptive now. Even if they are adaptive now, this is by itself insufficient grounds to claim they were selected because they were the best available adaptation for camouflage, as opposed to some other function or for that matter that they were not selected at all but are mere "spandrels," or traits riding piggy-back on some other means of predator avoidance or some other adaptive trait.

Reductionists will reply to this criticism that adaptationalist ultimate explanations of functional traits are "how-possibly" explanations, and the "just-so-story" charge laid against ultimate explanation on these grounds mistakes incompleteness (and perhaps fallibility) for untestability. The reductionist has no difficulty with the ultimate functional how-possibly explanation, as far as it goes. For its methodological role is partly to show how high fitness could in principle be the result of purely non-purposive processes, and partly to set the research agenda which seeks to provide why-necessary explanations, which cash in the promissory notes offered by the how-possibly explanation. But the reductionist shares with others suspicious of ultimate explanation a cognition of its severe limitations: its silence about crucial links in the causal chains to which it adverts.

9. Perhaps the best explanation for why there is no law here is to be found in Lange 1995; see also Lange 2000. Lange however employs his analysis to shows why biologists treat a statement of the form "the S is (or has) T" as a law even though it is admittedly neither purely qualitative nor counterfactual supporting.

The how-possibly explanation leaves unexplained several biologically pressing issues, ones which are implicit in most well-informed requests for an ultimate explanation. These are the question of which alternative adaptive strategies were available to various lineages of organisms, and which were not, and the further question of how the feedback from adaptedness of functional traits—like the eyespot—to their greater subsequent representation in descendants was actually effected. Silence on the causal details of how the feedback loops operate from fortuitous adaptedness of traits in one or more distantly past generations to improved adaptation and ultimately an approach to constrained locally optimal design, is the most disturbing lacuna in how-possibly explanations. Dissatisfaction with such explanations, as voiced by those suspicious of the theory of natural selection and those amazed by the degree of apparent optimality of natural design, as well as the religious, all stem from a widely shared prescientific commitment to complete causal chains, along with the denial of action at a distance, and of backward causation. Long before Darwin, or Paley for that matter, Spinoza diagnosed the problem of purposive or goal directed explanation as that it "reverses the order of nature," and makes the cause the effect. Natural selection replaces goal-directed processes. But natural selection at the functional level is silent on the crucial links in the causal chain that convert the appearance of goal-directedness into the reality of efficient causation. Therefore, explanations that appeal to it sometimes appear to be purposive or give hostages to fortune, by leaving too many links in their causal chains unspecified. Darwin's search for a theory of heredity reflected his own recognition of this fact.

The charge that adaptational explanations are unfalsifiable or otherwise scientifically deficient reflects the persistent claim by advocates of the adequacy of ultimate explanations that their silence on these details is not problematic.

Only a macromolecular account of the process could answer these questions. Such an account would itself also be an adaptational explanation. It would identify strategies available for adaptation, by identifing the genes (or other macromolecular replicators) which determined the characteristics of Lepidopterans' evolutionary ancestors, and which provide the only stock of phenotypes (leaf color camouflage, spot-camouflage, or other forms of Batesian mimicry, repellant taste to predators, Mullerian mimicry of bad tasting species, etc.) on which selection can operate to move along pathways to alternative predation-avoiding outcomes. The reductionist's why-necessary explanation would show how the extended phenotypes of these genes competed and how the genes which generated the eyespot eventually became predominant, i.e. were selected for.

In other words, the reductionist holds that a) every functional ultimate explanation is a how-possibly explanation, and b) there is a genic and

biochemical pathway selection process underlying the functional how-possibly explanation. As we shall see below, reduction turns the merely how-possible scenario of the functional ultimate explanation in to a why-necessary proximate explanation of a historical pattern. Note that the reductionist's full explanation is still a historical explanation in which further historical facts—about genes and pathways—are added, and are connected together by the same principles of natural selection that are invoked by the ultimate functional how-possibly explanation. But the links in the causal chain of natural selection are filled in to show how past adaptations were available for and shaped into today's functions.

Antireductionists will differ from reductionists not on the facts but on whether the initial explanation was merely an incomplete one or just a how-possibly explanation. Antireductionists will agree that the macromolecular genetic and biochemical pathways are causally necessary to the truth of the purely functional ultimate explanation. But they don't complete an otherwise incomplete explanation. They are merely further "facets of [the] situation that molecular research might illuminate." (Kitcher 1999, 199). The original ultimate answer to the question "Why do butterflies have eyespots?" does provide a complete explanatory answer to a question. Accordingly, how-possibly explanations are perfectly acceptable ones, or else the ultimate explanation in question is something more than a mere how-possibly explanation.

Who is right here?

On an erotetic view, how-possibly and why-necessary explanations may be accepted as reflecting differing questions expressed by the same words. The reductionist may admit that there are contexts of inquiry in which how-possible answers to questions satisfy explanatory needs. But the reductionist will insist that in the context of advanced biological inquiry, as opposed say to secondary school biology instruction, for example, the how-possibly question either does not arise, or having arisen in a past stage of inquiry, no longer does. How-possibly questions do not arise where the phenomena to be explained are not adaptations at all, for instance constraints, or spandrels, and the only assurance that in fact how-possibly explanations make true claims is provided by a why-necessary explanation that cashes in their promissory notes by establishing the adaptive origins of the functional traits in molecular genetics. This will become clearer as we examine proximate explanation in biology.

Consider the proximate explanation from the developmental biology of butterfly wings and their eyespots. Suppose we observe the development of a particular butterfly wing, or for that matter suppose we observe the development of the wing in all the butterflies of the buckeye species, *Precis coenia*. Almost all will show the same sequence of stages beginning with a wing imaginal disk eventuating in a wing with such spots, and a few will

show a sequence eventuating in an abnormal wing or one without the characteristic eyespot, maladapted to the butterfly's environment. Rarely one may show a novel wing or markings fortuitously better adapted to the environment than the wings of the vast majority of members of its species.

Let's consider only the first case. We notice in one buckeye caterpillar (or all but a handful) that during development an eyespot appears on the otherwise unmarked and uniform epithelium of the emerging butterfly wing. If we seek an explanation of the sequence in one butterfly, the general statement that all members of its species develop eyespots is unhelpful. First because examining enough butterflies in the species shows it is false. Second, even with an implicit ceteris paribus clause, or a probabilistic qualification, we know the "generalization" simply describes a distributed historical fact about some organisms on this planet around the present time and for several million years in both directions. One historical fact cannot by itself explain another, especially not if its existence *entails* the existence of the fact to be explained. That all normal wings develop eyespots does not explain why one does. Most non-molecular generalizations in developmental biology are of this kind. That is, they may summarize sequences of events in the lives of organisms of a species or for that matter in organisms of higher taxa than species.[10] But, the reductionist will argue, they proximately explain nothing. They cannot, owning to their character as implicit descriptions of historical (i.e. particular, implicitly dated) events, states, conditions, processes, or patterns.

How is the pattern of eyespot development in fact proximally explained? Having identified a series of genes which control wing development in *Drosophila*, biologists then discovered homologies between these genes and genes expressed in butterfly development, and that whereas in the fruit fly they control wing formation, in the butterfly they also control

10. Here is an example of typical generalizations in developmental biology from Wolpert (1997, 320):

> Both leg and wing discs [in *Drosophila*] are divided by a compartmental boundary that separates them into anterior and posterior developmental region. In the wing disc, a second compartment boundary between the dorsal and ventral regions develops during the second larval instar. When the wings form at metamorphosis, the future ventral surface folds under the dorsal surface in the distal region to form the double layered insect wing.

Despite its singular tone, this is a general claim about all (normal) *Drosophila* leg and wing imaginal discs. And it is a purely descriptive account of events in a temporal process recurring in all (normal) *Drosophila* larva. For purposes of proximate explanation of why a double layer of cells is formed in any one imaginal disc, this statement is no help; it simply notes that this happens in them all, or that it does so in order to eventually form the wing.

pigmentation. The details are complex but following out a few of them shows us something important about how proximate why-necessary explanation can cash in the promissory notes of how-possibly explanation and in principle reduce ultimate explanations to proximate ones.

In the fruit fly, the wing imaginal disk is first formed as a result of the expression of the gene *wingless* (so called because its deletion results in no wing imaginal disk and no wing) which acts as a position signal to cells directing specialization into the wing disc-structure. Subsequently, the homeotic selector gene *apterous* is switched on and produces apterous protein only in the dorsal compartment of the imaginal disk. This controls formation of the dorsal (top) side of the wing and activates two genes, *fringe* and *serrate,* which form the wing margin or edge. These effects were discovered by preventing dorsal expression of *apertous,* which results in the appearance of ventral (bottom) cells on the dorsal wing, with a margin between them and other (nonectopic) dorsal cells. Still another gene, *distal-less*, establishes the fruit fly's wing tip. Its expression in the center of the (flat) wing imaginal disk specifies the proximo-distal (closer to body/further from body) axis of wing development.[11]

Once these details were elucidated in *Drosophila,* it became possible to determine the expression of homologous genes in other species, in particular in *Precis coenia*. To begin with, nucleic acid sequencing showed that genes with substantially the same sequences were to be found in both species. In the butterfly these homologous genes were shown to also organize and regulate the development of the wing, though in some different ways. For instance, in the fruit fly *wingless* organizes the pattern of wing margins between dorsal and ventral surfaces, restricts the expression of *apterous* to dorsal surfaces, and partly controls the proximo-distal axis where *distal-less* is expressed. In the butterfly, *wingless* is expressed in all the peripheral cells in the imaginal disk which will not become parts of the wing, where it programs their death. (Nijhout 1994, 45) *Apterous* controls the development of ventral wing surfaces in both fruit flies and butterflies, but the cells in which it is expressed in the *Drosophila* imaginal disk are opposite those in which the gene is expressed in *Precis* imaginal disks. As Nijhout describes the experimental results:

> The most interesting patterns of expression are those of *Distal-less*. In *Drosophila Distal-less* marks the embryonic primordium of imaginal disks and is also expressed in the portions of the larval disk that

11. The implicit naming convention for many genes is that a gene is named for the phenotypic result of its deletion or malfunction. Thus *wingless* builds wings. Note that genes are individuated functionally and evolutionarily. *Wingless* is so called because of those of its effects which were selected by the environment to provide wings. Similarly for *distal-less*.

> will form the most apical [wing-tip] structures . . . . In *Precis* larval disks, *Distal-less* marks the center of a presumptive eyespot in the wing color pattern. The cells at this center act as inducers or organizers for development of the eyespot: if these cells are killed, no eyespot develops. If they are excised, and transplanted elsewhere on the wing, they induce an eyespot to develop at an ectopic location around the site of implantation . . . . the pattern of *Distal-less* expression in *Precis* disks changes dramatically in the course of the last larval instar [stage of development]. It begins as broad wedge shaped patters centered between wing veins. These wedges gradually narrow to lines, and a small circular pattern of expression develops at the apex of each line.
>
> What remains to be explained is why only a single circle of *Distal-less* expression eventually stabilizes on the larval wing disks. (Nijhout 1994, 45)

In effect, the research program in developmental molecular biology is to identify genes expressed in development, and then to undertake experiments—particularly ectopic gene-expression experiments—which explain the long established observational "regularities" reported in traditional developmental biology. The explanantia uncovered are always "singular" boundary conditions insofar as the explananda are spatiotemporally limited patterns, to which there are always exceptions of many different kinds. The reductionistic program in developmental molecular biology is to first explain the wider patterns, and then explain the exceptions—"defects of development" (if they are not already understood from the various ectopic and gene deletion experiments employed to formulate the why-necessary explanation for the major pattern).[12]

12. Is there an alternative to the reductionist's why-necessary explanation in terms of the switching on and off of a variety of genes which control the emergence and activity of cells of certain types at the eyespots? Some antireductionists seek such an alternative in explanatory generalizations that cut across the diverse macromolecular programs that realize development. For example, Kitcher identifies certain mathematical models as regularities important to 'growth and form' (consciously echoing D'Arcy Thompson) in development. These regularities suggest a multilevel process, one in which levels above the macromolecular really are explanatory. In particular Kitcher cites the work of J. D. Murray (1989).

J. D. Murray elaborated a set of simultaneous differential equations reflecting relationships between the rates of diffusion of pigments on the skin and the surface areas of the skin. By varying the ratio of skin surface to diffusion rates, Murray's equations can generate patterns of spots, stripes, and other markings in a variety of mammals. As Kitcher has pointed out (1999, 204), Murray's system of equations together with some assumptions about the ratio of surface area to diffusion rates of pigments imply that there are no striped animals with spotted tails—an apparently well established observational regularity. Though Kitcher does not mention it, Murray goes on to develop another system of differential equations, for the relation between surface area and pigment that produces eyespots on butterfly wings. What is of interest in the present

This program is by no means complete and the reductionist's why-necessary explanations are not yet in. But they are obviously coming. In providing them, the reductionist also pays the promissory notes of the ultimate how-possibly explanations biologists proffer. Recall that the ultimate how-possibly explanation of the eyespot appeals to its predator-distraction and camouflage properties, but is silent on why this adaptation emerged instead of some other way of avoiding predation, and so is vulnerable to question, and invulnerable to test. Developmental molecular biology can answer questions about adaptation by making adaptation's historical claims about lines of descent open to test.

The developmental molecular biologists, S. B. Carroll and colleagues, who reported the beginnings of the proximal explanation sketched above, eventually turned their attention to elucidating the ultimate explanation. They write:

> The eyespots on butterfly wings are a recently derived evolutionary novelty that arose in a subset of the Lepidoptera and play an impor-

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debate is Murray's assessment of the explanatory power of these mathematical models—sets of differential equations together with restrictions on the ratios among their variables:

> Here we shall describe and analyze a possible model mechanism for wing pattern proposed by Murray (1981). As in [mammalian coat color], a major feature of the model is the crucial dependence of the pattern on the geometry and scale of the wing when the pattern is laid down. Although the diversity of wing patterns might indicate that several mechanisms are required, among other things we shall show here how seemingly different patterns can be generated by the same mechanism. (1989, 450–451)

Murray concludes:

> The simple model proposed in this section can clearly generate some of the major pattern elements observed in lepidopteran wings. As we keep reiterating in this book, it is not sufficient to say that such a mechanism is that which necessarily occurs . . . . From the material discussed in detail in [another chapter of Murray's book] we could also generate such patterns by appropriately manipulating a reaction diffusion system capable of diffusion driven pattern generation. What is required at this stage if such a model is indeed that which operates, is an estimate of parameter values and how they might be varied under controlled experimental conditions.
>
> . . . It is most likely that several independent mechanisms are operating, possibly at different stages, to produce diverse patterns on butterfly wings. . . .
>
> . . . Perhaps we should turn the pattern formation question around and ask: "What patterns cannot be formed by such simple mechanisms?" (465)

Murray treats his sets of simultaneous equations not as generalizations with independent explanatory power, but as parts of a how-possibly explanation which needs to be cashed in by developments that convert it into a why-necessary explanation or supplant it with such an explanation.

In the period after Murray first produced his models, molecular biology has provided more and more of the proximate why-necessary explanations the reductionist demands for the historical facts about butterfly eyespots.

> tant role in predator avoidance. The production of the eyespot pattern is controlled by a developmental organizer called the focus, which induces the surrounding cells to synthesize specific pigments. The evolution of the developmental mechanisms that establish focus was therefore the key to the origin of butterfly eyespots. (Keys, Carroll, et al. 1999, 532)

What Carroll's team discovered is that the genes and the entire regulatory pathway that integrates them and which control anterior/posterior wing development in *Drosophila* (or its common ancestor with butterflies) have been recruited and modified to develop the eyespot focus. This discovery of the "facility with which new developmental functions can evolve . . . . within extant structures" (534) would have been impossible without the successful why-necessary answer to the proximate question of developmental biology.

Besides the genes noted above, there is another, *Hedgehog,* whose expression is of particular importance in the initial division of the *Drosophila* wing imaginal disk into anterior and posterior segments. As in the fruit fly, in *Precis* the *Hedgehog* gene is expressed in all cells of the posterior compartment of the wing, but its rate of expression is even higher in the cells that surround the foci of the eyespot. In *Drosophila*, *Hedgehog*'s control over anterior/posterior differentiation appears to be the result of a feedback system at the anterior/posterior boundary involving four other gene products, and in particular one, *engrailed*, which represses another, *cubitus interruptus* (hereafter '*ci*' for short), in the fruit fly's posterior compartment. This same feedback loop is to be found in the butterfly wing posterior compartment, except that here the *engrailed* gene's products do not repress *ci* expression in the anterior compartment of the wing. The expression of *engrailed*'s and *ci*'s gene-products together results in the development of the focus of the eyespot. One piece of evidence that switching on the *Hedgehog-engrailed-ci* gene system produces the eyespot comes from the discovery that in those few butterflies with eyespots in the anterior wing compartment, *engrailed* and *ci* are also expressed in the anterior compartment at the eyespot foci (but not elsewhere in the anterior compartment). "Thus, the expression of the *Hedgehog* signaling pathway and *engrailed* is associated with the development of all eyespot foci and has become independent of the [anterior/posterior] restrictions [that are found in *Drosophila*]." (Keys, Carroll, et al. 1999, 534)

Further experiments and comparative analysis enabled Carroll and coworkers to elucidate the causal order of the changes in the *Hedgehog* pathway as it shifts from wing-production in *Drosophila* (or its ancestor) to focus production in *Precis* eyespot development. "The similarly between the induction of *engrailed* by *Hedgehog* at the [anterior/posterior] bound-

ary [of both fruit fly and butterfly wings, where it produces the intervein tissue in wings] and in eyespot development suggests that during eyespot evolution, the *Hedgehog*-dependent regulatory circuit that establishes foci was recruited from the circuit that acts along the Anterior/Posterior boundary of the wing." (Keys, Carroll, et al. 1999, 534)

Of course, the full why-necessary proximate explanation for any particular butterfly's eyespots is not yet in, nor is the full why-necessary proximate explanation for the development of *Drosophila's* (or its ancestor's) wing. But once they are in, the transformation of the ultimate explanation of why butterflies have eyespots on their wings into a proximate explanation can begin. This fuller explanation will still rely on natural selection. But it will be one in which the alternative available strategies are understood and the constraints specified, the time and place and nature of mutations narrowed; in which adaptations are unarguably identifiable properties of genes—their immediate or mediate gene products (in Dawkin's terms, their extended phenotypes); and in which the feedback loops and causal chains will be fully detailed. The scope for doubt, skepticism, questions, and methodological critique that ultimate explanations are open to will be much reduced.

6. Methodological Morals: Reductionism and the Return of the Gene. At the outset I claimed that reductionism is a methodological dictum that follows from biology's commitment to provide explanations. This claim can now be made more explicit, even against the background of an erotetic theory of which explanations are adequate and when. Every one should agree that biology is obliged to provide why-necessary explanations for historical events and patterns of events. The latter-day reductionist holds that such why-necessary explanations can only be provided by adverting to the macromolecular states, processes, events, and patterns that these non-molecular historical events and patterns supervene on. Any explanation that does not do so cannot claim to be an adequate, complete why-necessary explanation.

The reductionist does not claim that biological research or the explanations it eventuates in can dispense with functional language or adaptationism. Much of the vocabulary of molecular biology is thoroughly functional. As I have noted, the reductionist needs the theory of natural selection to make out the case for reduction. Nor is reductionism the claim that all research in biology must be "bottom up" instead of "top down" research. So far from advocating the absurd notion that molecular biology can give us all of biology, the reductionist's thesis is that we need to identify the patterns at higher levels because they are the explananda that molecular biology provides the explanantia for. What the reductionist asserts is that functional biology's explanantia are always molecular biol-

ogy's explananda, that molecular biology does not merely provide "explanatory extensions" (Kitcher 1984) of functional biological explanations. It deepens and completes them, when it does not supplant them.

So, why isn't everyone a reductionist, why indeed, is antireductionism the ruling orthodoxy among philosophers of biology and even among biologists? Because, in the words of one antireductionist, reductionism's alleged "mistake consists in the loss of understanding through immersion in detail, with concomitant failure to represent generalities that are important to 'growth and form'." (Kitcher, p. 206, invoking D'Arcy Thompson's expression) The reductionist rejects the claim that there is a loss of biological understanding in satisfying reductionism's demands on explanation, and denies that there are real generalities to be represented or explained. In biology there is only natural history—the product of the laws of natural selection operating on macromolecular initial conditions.

Rejecting the claim that natural selection is always at bottom genic or some other sort of macromolecular selection, another antireductionist argues that reductionism adds nothing to the predictive power of functional biology:

> A predictive theory needs to focus on fitness differences that can occur anywhere in the biological hierarchy. Multilevel selection theory [which denies reduction of selection to genic selection] offers a precise framework for identifying these differences [at the levels of populations, groups, individuals, as well as genes] . . . . and for measuring their relative strengths. Selfish gene theory requires all these same distinctions. But its central concept of genes as replicators offers no help. All the hard work is left for the . . . vehicles [populations, groups, individuals]. (Sober and Wilson 1998, 93–94)

Reductionism accepts that selection obtains at higher levels, and that for predictive purposes, focus on these levels often suffices. But the reductionist insists that the genes, and proteins they produce, do in fact offer irreplaceable "help." Sometimes, indeed for a long period in the natural history of the earth, they were the only vehicles of selection, and they are still the "bottleneck" through which selection among other vehicles is channeled. Without them, the causal credentials and indeed the explanatory power of predictively useful claims in functional biology are open to challenge, and with them, explanatory force is vouched safe, while predictive power may be increased.

In "The Return of the Gene" two antireductionists, Kitcher and Sterelny, argue for a thesis they call "pluralist genic selectionism": the thesis that there is more than one maximally adequate representation of a selective process, that for any given selective process, this set of maximally adequate representations will sometimes include a description in terms of

individual selection, at other times kin selection, sometimes group selection, or even species selection. But, they argue, every set of (equally) maximally adequate representations for any one process will always include at least one representation attributing causal efficacy to genic properties. (Kitcher and Sterelny 1998, 171) They distinguish this thesis from one they call hierarchical monist selectionism: the thesis that selection can operate independently at many different levels of organization—the gene, the individual, the group, the species, etc.—and " that for each process there is one kind of adequate representation [not many, hence the monism], but that processes are diverse in the kinds of representations they demand [hence the hierarchy]." (173)

Antireductionism requires the truth of hierarchical monist selectionism. But reductionists can accept pluralist genic selectionism. To see this, consider whether the adequacy of genic selection descriptions in every selective process is an accident, or has an explanation. Kitcher and Sterelny explicitly reject the explanation that claims genic selection is "the (really) real causal story." (171) Instead, "the virtue of the genic point of view, on the pluralist account is not that it alone gets the causal structure right, but that it is always available." (172) But why is it always available? What seems like the right explanation of the universal appropriateness of the genic representation is not that it is the whole of the (really) real causal story in every case of selection, but that it is an indispensable *part* of the (really) real causal story in every case of selection. The only way to deny this is to claim that explanations of selective processes that do not advert to genes are complete, adequate, and correct. And this is hierarchical monist selectionism, according to which the genes add nothing: all the work is done by the vehicles of selection.

Insofar as science seeks to complete this (really) real explanation for historical events and patterns on this planet, it needs to pursue a reductionistic research program. That is, biology can nowhere remain satisfied with how-possibly ultimate explanations, it must seek why-necessary proximate explanations, and it must seek these explanations in the interaction of macromolecules.

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