Fit and Diversity: Explaining Adaptive Evolution*

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According to a prominent view of evolutionary theory, natural selection and the processes of development compete for explanatory relevance. Natural selection theory explains the evolution of biological form insofar as it is adaptive. Development is relevant to the explanation of form only insofar as it constrains the adaptation-promoting effects of selection. I argue that this view of evolutionary theory is erroneous. I outline an alternative, according to which natural selection explains adaptive evolution by appeal to the statistical structure of populations, and development explains the causes of adaptive evolution at the level of individuals. Only together can a statistical theory of selection and a mechanical theory of development explain why populations of organisms comprise individuals that are adapted to their conditions of existence.

1. Introduction. Evolutionary theory has two principal explananda, *fit* and *diversity* (Lewontin 1978). By "fit" here I mean what Darwin called "those exquisite adaptations of one part of the organisation to another part, and to the conditions of life" (Darwin [1859]1996, 114). By "diversity" I mean simply the distribution of biological form. Darwin's great insight was that fit and diversity are consequences of a single process, adaptive evolution. Darwin argued that in order to explain adaptive evolution—and hence fit and diversity—one must explain how populations come to comprise individual organisms so well adapted to their conditions of existence. So

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there are two phenomena to be explained in adaptive evolution: the structure of populations and the properties of individuals.

Natural selection stakes its place as the central, unifying concept in evolution because it demonstrates that both phenomena are the consequence of a single process. Natural selection, we are told, preferentially maintains adaptive phenotypes within populations and in doing so raises the chances that other adaptive phenotypes emerge. Selection causes populations to change over time such that they comprise increasingly well-adapted individuals.

At the same time, it appears that natural selection alone might not account completely for the distribution of biological form. The bearers of biological form are organisms and each organism faces the tribunal of the environment as a corporate entity, not as a loose aggregate of independent traits. One consequence of this is that at each stage of its development from egg to adult an organism must be an integrated, functioning whole. Another is that for any form (trait) to arise in an organism at a time, it must develop from the materials and processes at the organism's disposal at that time. The requirement of integration and the processes of development that produce it leave their distinctive traces on biological form. It seems reasonable, then, to suppose that one might appeal to the processes of development in explaining the nature and distribution of biological form.

So there are two general strategies for explaining biological form: selectional and developmental. There is little understanding, though, of the relation between them. It is usually thought that they compete for explanatory relevance. If a feature of organic form is adaptive, then its presence and prevalence will be explained by appeal to selection; the processes of development will not be particularly germane. If a feature of form is not adaptive its presence cannot be explained by appeal to natural selection; only then will developmental processes be explanatorily salient. In my view this perceived competition for explanatory relevance is an error. And a serious one; it has impeded the assimilation of a theory of development into evolutionary theory in general. My objective here is to identify the source of the error and to sketch an alternative *modus vivendi* for the theory of natural selection and a theory of development, in which selection and development are complementary not competing, explanatory strategies.

I proceed in the following way. In Section 2 (immediately to follow) I sketch out what I take to be a fairly orthodox interpretation of the Modern Synthesis theory. This is the interpretation that generates the competition for explanatory relevance between selection and development. In Sections 3 and 4, I suggest reasons for supposing that this interpretation is a mistake. In Section 3 I argue that, contrary to received opinion, natural selection theory does not identify the causes of adaptive evolution. In Sec-

tion 4, I argue that the place to look for a theory of the causes of adaptive evolution is in development, particularly in the phenomenon of developmental constraint. In Section 5, I attempt to sketch an outline of the way in which a theory of natural selection and a theory of development may complement one another in a unified explanation of adaptive evolution.

2. The Two-Force Model. The Modern Synthesis theory of evolution is composed of two components: the theory of Mendelian inheritance, and the theory of natural selection. These theories explain phenomena in different domains. The former allows us to predict and explain the transmission of *genotypes* from one generation to the next. The latter accounts for the way a population changes as a function of the different *phenotypes* of individual organisms (Lewontin 1974). A set of transformation rules is needed for mapping the phenomena of genotype space onto those in phenotype space and vice versa.[1]

Genotypes become phenotypes through the process of development (Lewontin 1992). One important objective of a theory of development is to understand the mechanics of the mapping of genotypes onto phenotypes. The "genotype-phenotype map" might be transparent or opaque. By "transparent" I mean simply that the magnitude and direction of changes in genotype space correspond closely to the magnitude and direction of changes in phenotype space. If the genotype-phenotype relation were transparent, changes to the kinds and frequencies of genotypes wrought by the processes operating over genotype space—replication, segregation, recombination, mutation, etc.—could be mapped straight on to changes in phenotype space. Changes in biological form could then be exhaustively explained by a combination of processes at the genotype level (e.g., Mendelian inheritance) plus selection operating exclusively at the level of phenotypes. The details of individual development would not matter much to the explanation of adaptive evolution.[2] On the other hand, if the genotype-phenotype map were opaque, then changes in genotype space would not translate in any simple way into changes in phenotype space. Large transitions in genotype space may correspond to small or no changes in phenotype space, while small (or no) changes in genotype space may correspond to major phenotypic differences.[3] If the genotype-phenotype map introduces changes of its own to phenotypic space, then we need to invoke (at least) two sets of causal processes, or forces, in order to explain

1. I shall be concerned here only with mapping genotypic phenomena onto phenotype space.

2. The Modern Synthesis theory was forged explicitly under the assumption of transparency (inter alia). See Hodge 1992 and Morrison 2002.

3. For examples of the way opacity might manifest itself see Oster et al. 1980.

phenotypic evolution: the force of selection and the various processes of development. I shall call this conception of the way both selection and development influence the evolution of form the "two-force model."

There is nothing in the two-force model, yet, to suggest that natural selection theory and a theory of development compete for explanatory relevance in any way that impedes their unification. There are perfectly unified multiple-force theories. The theory of classical mechanics, for example, posits multiple sorts of forces acting on bodies (e.g., gravitational and electromagnetic), but a theory of gravitation and a theory of electromagnetism are not antagonistic. The reason is that the respective effects of these forces are commensurable; they both cause bodies to accelerate. In contrast, selection and development are thought not to have common effects. Only changes in phenotype space that are the consequence of selection count as adaptations.[4] For any adaptive phenotype, the thought goes, its nature and history can be adequately explained by appeal to the process of natural selection alone (Sober 1993). Development is relevant insofar as form is not adaptive. A theory of development is needed only to the extent that natural selection is inadequate to explain the distribution of organic form. In this sense, the theory of natural selection and a theory of development compete for explanatory relevance.

2.1. Developmental Constraint. Those who seek some form of détente between selection theory and a theory of development point out that the respective effects of development and selection can be measured against one another in useful ways. The general idea is that natural selection introduces biases in form that are a function of fitness. Development introduces biases in form that are *independent* of fitness. Sometimes, the biases in the distribution of form introduced by development make those adaptations that would otherwise be caused by selection unavailable or difficult to attain. In this way, development impedes the adaptation-promoting effects of selection in much the same way that friction or drag impedes the acceleration of a body.

The canonical definition of a developmental constraint is given by John Maynard Smith et al. (1985).

> A developmental constraint is a bias in the production of variant phenotypes or a limit on phenotypic variability caused by the structure, character, composition, or dynamics of the developmental system. (Maynard Smith et al. 1985, 266)[5]

4. Indeed, this has come to be seen as a conceptual truth; it is widely held that to be an adaptation is to be the causal consequence of natural selection. For standard examples see Williams 1966, Sober 1993, and Futuyma 1997.

5. Amundson (1994 and 2001), Gould (2002, 1025), and Schwenk and Wagner (2003a) point out a significant equivocation on "developmental constraint." It is defined as a

2.2. Two Landscapes. The power of selection to cause adaptive evolution and the capacity of developmental constraint to impede it are sometimes illustrated by means of two pictorial devices, the *Adaptive Landscape* and the *Epigenetic Landscape.* The Adaptive Landscape is often used to illustrate the way fit and diversity are both causal consequences of selection.[6] We can represent the collection of genotypes in a population on a multidimensional grid, one dimension for each gene. The intersections depict whole genotypes. This multidimensional volume is usually represented as a plane, with each whole genotype assigned an altitude. Less fit individuals, those at lower altitudes, are preferentially removed from the population. As more fit variants are introduced, natural selection drives the population toward local fitness optima. Neighboring populations on either side of a fitness valley will diverge toward separate peaks. In this way, natural selection, by helping to change the constitution of a population, produces populations that are, on average, better adapted, and that diverge toward separate adaptive peaks in response to different selection pressures.

The way in which developmental constraint impedes adaptive evolution is sometimes illustrated by means of another pictorial device, the *Epigenetic Landscape* introduced by C. H. Waddington (1957 and 1960). Waddington represented the trajectory of a developing phenotypic feature, something he called a "creode," as an inclined surface. The surface is marked by a series of branching channels, like river valleys and their tributaries. We can think of the development of a creode as a ball rolling down this landscape, starting with the undifferentiated egg stage at the top and culminating in the highly differentiated adult form at the bottom. As development progresses the creode gets shunted into one channel or another, until finally it reaches its adult form. The epigenetic landscape is given its shape largely by the "epistatic" interactions among genes. Epistasis is simply the phenomenon whereby genes have effects on the products of other genes (Wade 1992). Waddington (1960) believed that these regulatory epistatic interactions among genes could exert significant control on the development of phenotype.

Waddington thought that the imagery of the Epigenetic Landscape helped clarify some of the most puzzling features of development (Gibson and Wagner 2000). One is that there are only a few different kinds of tissue types. They are discrete and stable and yet they develop from the

limit on phenotypic variation but has come to stand for a limit on the efficacy of selection.

6. The *locus classicus* of this use of the Adaptive Landscape is Simpson's 1944 account of the adaptive radiation of horses. See also Gould's (1983) discussion of Dobzhansky.

same undifferentiated, homogeneous precursors. This phenomenon is captured in the epigenetic landscape by the fact that toward the bottom of the landscape there is a limited number of channels; each is narrow and is separated from the others by steep-sided banks. Another puzzling developmental phenomenon is that even though the various creodes are sometimes highly susceptible to environmental triggers, they are also extremely robust. Tissues and organs develop successfully despite a wide range of environmental perturbations. This "environmental buffering" of trajectories is represented by the banks of the valleys. A perturbation drives the developing tissue up the banks of a channel, whence it returns (often enough) to the stable point at the bottom of the valley. A third feature of development illustrated by the landscape is that of genetic buffering. Organisms carry an enormous amount of genetic variation but despite this variability there is startling constancy in the final products of ontogeny. Waddington coined the term "*canalization*" to cover these three salient features of ontogeny.

Waddington noted that occasionally novel, stable phenotypes may be elicited when large perturbations are applied to a canalized trajectory.[7] He surmised that within a population there is latent genetic variation; some individuals have the genetic endowment for these quite different phenotypes and some do not. But these genetic differences do not show up as phenotypic differences because development is so heavily constrained by canalization.

Putting the adaptive and epigenetic landscapes together, we can see how the opacity of development—as represented by the canalized Epigenetic Landscape—might impede the power of natural selection to effect adaptive evolution, as envisaged in the Adaptive Landscape. Canalization makes unavailable certain phenotypes that might otherwise be adaptively advantageous. It is as though the canalization of development puts a cordon around certain parts of the adaptive landscape, making certain phenotypes inaccessible (in the sense of inexpressible). Were it not for canalization, individuals with the genotypes corresponding to these fenced-off areas might well have much higher fitnesses than they actually do. If these fenced-off areas of the adaptive landscape were available, selection would drive the population onto these uninhabited peaks.

If this is the appropriate interpretation of how the Epigenetic Landscape relates to the Adaptive Landscape, it seems to confirm the suspicion that all a theory of development can contribute to the Modern Synthesis is an account of the way that developmental processes impede the adaptation-promoting effects of selection. Wagner and Altenberg express the idea clearly:

7. The most striking example is the discovery that ether shock could produce the bithorax phenotype in *Drosophila* (Waddington 1957).

> For instance developmental constraints frustrate selection by restricting the phenotypic variation selection has to act upon. Adaptations would be able to evolve only to optima within the constrained space of variability. (1996, 973)

A similar role for developmental constraint is envisaged by Raff:

> The nature of the existing developmental system somehow constrains or channels acceptable change [of form in evolution], so that selection is limited in what it can achieve given some starting anatomy. (1996, 294–295)

This conception of the significance of development to adaptive evolution follows quite naturally from the two-force model of evolution. I think it is as deeply flawed as it is entrenched. It is this view of evolutionary theory that promotes the idea that a theory of development and the theory of natural selection must compete for explanatory relevance. As Schwenk and Wagner put it:

> This has led to a "dichotomous approach" in which constraint is conceptually divorced from natural selection and pitted against it in a kind of evolutionary battle for dominance over the phenotype . . . much of the constraint literature over the last 25 years has explicitly sought to explain evolutionary outcomes as *either* the result of selection *or* constraint (2003b, 1–2)

My objection to the two-force model is not that it accords too little explanatory role to development; after all it is consistent with the two-force model that developmental constraint may win the "battle for dominance over the phenotype." My objection is that it accords the wrong role to both selection and development. The two-force model takes on two commitments: that selection causes the adaptedness of individual organisms and that developmental constraint does not. I think that both these commitments are mistaken. I discuss them, in turn, in the following two sections.

3. The Force of Natural Selection. Natural selection, we are often told, is more than just the differential survival of individuals; it is the force that causes adaptive evolution. Natural selection retains the gene combinations that "fit" and it creates ones that fit better. Ernst Mayr has advanced this position, and he professes himself to be in good company:

> When natural selection acts, step by step, to improve such a complex system as the genotype, it does not operate as a purely negative force. . . . It does not confine itself to the elimination of inferior gene combinations; rather, its most important contribution is to bring superior

> gene combinations together. It acts as a positive force that pays a premium for any contribution toward an improvement, however small. For this reason profound thinkers about evolution, such as Theodosius Dobzhansky, Julian Huxley, and G. G. Simpson, have called selection "creative." (1976, 45–46)[8]

The most striking feature of this passage is its conviction that selection is *the* force that causes adaptive "improvements." Indeed, this force talk is endemic in evolutionary theorizing.[9]

Alongside this talk of selection as a force, we are told that Darwin's intellectual triumph in the discovery of natural selection was the consequence of a very simple change of perspective. Rather than asking how individuals become so well adapted to their conditions of existence, Darwin asked how populations come to comprise individuals so well adapted. Mayr (1976) famously dubbed this the shift from "typological" thinking to "population" thinking. The general idea is that the population is the unit of evolutionary change and natural selection is a theory that explains the changes in population structure. It does so by citing a specific feature of population structure: variation. As Lewontin states it:

> [Darwin] called attention to the *actual* variation among *actual* organisms as the most essential and illuminating fact of nature. Rather than regarding the variation among members of the same species as an annoying distraction, as a shimmering of the air that distorts our view of the essential object, he made that variation the cornerstone of his theory. (1974, 5)

So there are two salient features of natural selection theory: (i) it is *dynamical:* it explains by citing the actions of a force (selection), and (ii) it is *variational:* it explains by appeal to the variation within populations. These two features look odd when juxtaposed. It seems to me that if natural selection is a variational theory, it is not dynamical, and if it is dynamical, it is not variational. A dynamical theory, such as Newtonian mechanics, explains the changes in a body that are the consequence of forces impinging on it from without. It needs no ensemble-level variation. A "variational" theory, such as we have in, say, thermodynamics, explains those changes in an ensemble that are the consequence of the variation within the ensemble. It does not appeal to forces. If natural selection theory is genuinely variational, then this suggests that, contrary to Mayr's assertion, it does not explain by citing the actions of a force.

8. Endorsement of the "creative power" of selection is remarkably widespread. A sampling of such passages may be found in Dobzhansky (1980), Ayala (1970), and Neander (1995), among many other places.

9. For vivid examples see Dawkins 1986 and Sober 1984.

3.1. The Thermodynamics Analogy. Perhaps the simplest way to make the dynamical/variational tension palpable is to consider the first appearance of natural selection in the Modern Synthesis. Fisher's (1930) account of the Genetical Theory of Natural Selection theory is comprehensively variational. Notoriously, Fisher likened natural selection theory to the theory of statistical thermodynamics (Hodge 1992). Just as one explains the trajectory of a volume of gas as a function of the statistical structure of the ensemble of molecules, one also explains the trajectory of a population undergoing selection as a function of *its* statistical structure. Further pressing the analogy with thermodynamics, Fisher articulates a single law of natural selection. It has become known as Fisher's Fundamental Theorem:

> The rate of increase of fitness of any [population] at any time is equal to the additive genetic variance at that time. (1930, 36)

Immediately on stating his fundamental theorem, Fisher draws the obvious parallel between it and the Second Law of Thermodynamics:

> It will be noticed that the Fundamental Theorem . . . bears some remarkable resemblances to the Second Law of Thermodynamics. Both are properties of populations, or aggregates, true irrespective of the nature of the units which compose them; both are statistical laws; each requires of a physical system the constant increase of a measurable quantity, in the one case entropy of a physical system in the other the fitness. (1930, 36)

The analogy between entropy and average fitness is particularly strained. It just is not true that natural selection inevitably increases average fitness, either locally or globally.[10] Nevertheless, enough of the analogy between natural selection and thermodynamics survives for my purposes. It demonstrates that natural selection theory explains its phenomena in much the way that thermodynamics does.[11]

Suppose that by some contrivance a volume of gas comes to have all its fast moving molecules in the north half of its container and the slow moving molecules in the south half. This volume of gas is far from thermodynamic equilibrium. It will move from this low entropy state toward an equilibrium state of high entropy where in any arbitrarily chosen part of the container, we are as likely to find a fast-moving particle as a slow one. The statistical treatment of thermodynamics renders a strictly statistical explanation of this change. The space of states in which the entropy

10. See Levins and Lewontin 1985 and more recently Demetrius 2000. See also Depew and Weber 1995, ch. 10, and Hodge 1992.

11. See Morrison's (2002) marvellous discussion for more details.

is low is minute compared to the space of states in which the entropy is high. Consequently, changes in which entropy increases are more likely than those in which it does not.

In the same way, natural selection explains the changes in the structure of a population by appeal to differences in trait fitness. Trait fitness is strictly a statistical property of a trait type. It is the mean and variance of the fitness of individuals in a certain class, where the possession of particular heritable traits determines class membership (Gillespie 1977; Sober 2001). Trait fitness measures the likelihood of a trait type increasing its relative frequency in a population. A population not at equilibrium exhibits variation in trait fitness; some trait types (classes) are more likely to increase in size than others. In such circumstances the population will tend to undergo a change toward an equilibrium condition in which there is no variation in trait fitness. The role of the genetical theory of natural selection, like that of thermodynamics, is to identify those changes in the structure of a population that are made most likely by the statistical properties of the population, that is, its variation in mean trait fitness.

The thermodynamics analogy makes evident another feature population change: spontaneity. Where an ensemble of molecules in a gas moves from low entropy to high, the change is spontaneous in the sense that once the momenta of the particles and their distribution in a closed system are fixed, no further forces need to be added to the system to get it to move toward equilibrium. Similarly, as Fisher's analogy makes apparent, natural selection is seen as a spontaneous tendency of populations that are not at fitness equilibrium. Once the distribution of trait fitnesses is fixed, there is no need to introduce further forces to account for the change. Just as we do not need to invoke a force—an *entroprizing* force—to explain the change in structure of an ensemble of molecules we do not need to posit a *selecting* force to explain the changes that occur within a population of organisms that manifests variation in fitness. To do so would be to overpopulate the world with forces.[12]

Perhaps worse, to do so would be to misrepresent the kind of explanation offered by the genetical theory of natural selection. On this construal, natural selection explanations are statistical explanations. These are not the sorts of explanations we get from citing the actions of forces (Rosenberg 2001; Walsh, Lewens, and Ariew 2002; Matthen and Ariew 2002).

Marjorie Grene (1961) evinces an uncommon sensitivity to the statistical nature of selection theory as embodied in the Modern Synthesis. Grene says:

12. For further different arguments that natural selection theory is not a theory of forces see Matthen and Ariew 2002.

> In terms of the "strictly defined" concepts it uses, [Fisher's] fundamental theorem is a statistical device for recording and predicting population changes. Nor is the situation altered by calling such changes "genetical selection." We must still distinguish between "genetical selection," which is purely statistical, and Darwinian selection which is environment-based and causal. (1961, 30)

By "Darwinian selection" Grene means the suite of causal processes that bring about "the elimination of characters less well adapted to a particular environment in favour of those slightly better adapted to that environment" (1961, 28). Her point, I take it, is that natural selection theory, as encoded in the Modern Synthesis, may well account for the ways in which a population changes under the influence of the processes that cause differential survival and reproduction and the inheritance of traits, but it does not constitute a "causal study" of those processes.[13]

> the fundamental theorem is a guide to statistical technique [*sic*] which is overlaid on the causal relations of heredity and can be used as underpinning for the causal study of Darwinian selection; but in itself it asserts neither. (Grene 1961, 34)

One consequence of interpreting the Genetical Theory of Natural Selection as "purely statistical" is that for any change in the structure of a population undergoing selection there will be a statistical explanation provided by "genetical" selection theory and a further, distinct explanation that "asserts" the causes of differential survival and reproduction of individuals. Here again, there is an obvious analogy to the statistical interpretation of thermodynamics. For any change in the entropy of a system there will be a strictly statistical, ensemble-level explanation given by the theory of thermodynamics. There will also be a mechanical, individual-level explanation that adverts to the momenta and positions of the particles in the system and the forces acting on them. But the statistical explanation abstracts away from these. It tells us that given the structure of the ensemble, it will be expected to change in predictable ways. In both Thermodynamics Theory and the Genetical Theory of Natural Selection, then, the ensemble-level, statistical explanations and individual-level, mechanical ones are independent in the sense that the ensemble-level, statistical explanations do not tell us about the individual-level processes and the individual-level mechanical explanations do not represent the changes in the statistical structure of the ensemble.

3.2. The Thermodynamics Disanalogy. The Genetical Theory of Natural Selection and the statistical treatment of thermodynamics may be struc-

13. I thank a referee for this journal for directing me to this paper.

turally analogous, but there are important differences between the respective explanatory projects to which they applied. In explaining thermodynamical phenomena one is seldom interested in accounting for the properties of individual particles. But if Darwin's central insight is right, then explaining adaptive evolution requires more than simply accounting for changes in population structure. It requires an account of how a group of individuals severally come to possess their adaptive phenotypes. As we have seen, ensemble-level, statistical theories do not explain the etiologies of the properties of individuals. If we are looking for the individual-level causes of adaptedness in organisms, we must look elsewhere.

Here, I think, we encounter one of the two basic errors of the two-force model: it misconstrues the explanations of natural selection theory. The two-force model is predicated on the idea that in explaining adaptive evolution by appeal to natural selection theory, we are citing the force that causes it. But if the thermodynamics analogy is correct, the Genetical Theory of Natural Selection theory no more identifies the forces causing adaptive evolution than the statistical treatment of thermodynamics identifies the causes of changes in the momenta of particles. The first mistake of the two-force model is that it takes natural selection to be a force, a below-population-level cause of the *fit* of individual organisms to their conditions of existence.

4. The Genotype-Phenotype Map. The obvious rejoinder on behalf of the two-force model is that—talk of causal versus statistical theories aside—natural selection theory accounts for the origin, improvement, and maintenance of adaptive phenotypes in individuals *by* explaining changes to the structure of populations. After all, natural selection explains why trait types that contribute to individual adaptedness increase in a population. Differential retention of the fitter trait types in a population brings about the new combinations of traits that are themselves adaptive (Ayala 1970; Neander 1995). Consequently, the differential retention of trait types in a population simply entails the increase in the adaptedness of individuals in the population. The two-force model may have been wrong about the metaphysics of selection theory, but it was right about its explanatory completeness.

There are good reasons to resist this rejoinder. The increase in average fitness of genes in a population entails an increase in the average adaptedness of individuals only under certain assumptions—*inter alia* that the effects of genes on individual fitnesses are independent and additive. These amount to the assumption that the genotype-phenotype map is transparent.[14] But when the genotype-phenotype map is opaque it is far from a

14. Fisher, for example, is explicit about this. See Grene 1961 and Morrison 2002.

trivial matter to get a population of organisms that exhibits heritable variation in trait fitnesses to undergo adaptive phenotypic evolution (Kauffman 1993). A number of lines of evidence are converging on the view that whether a population of organisms undergoes adaptive evolution depends upon the details of the genotype-phenotype map. As Wagner and Altenberg put it: "Adaptation requires that genetic change be able to produce adaptive phenotypic change. Whether or not adaptive changes can be produced depends critically on the genotype-phenotype map" (1996, 968). What distinguishes those genotype-phenotype maps that have this capacity from those that don't? We have already encountered one important factor in the discussion of Waddington's Epigenetic Landscape: epistasis.

4.1. Epistasis. The significance of epistasis for adaptive evolution has been one of the central features of Stuart Kauffman's (1993 and 1995) work.[15] Kauffman presents a series of models in which a genome is represented as a large Boolean network. Each gene is a node in the network. The connections between the nodes play the part of epistatic interactions. Where the number of connections is high, each gene directly affects the consequences of many other genes. Where they are low (or zero), each gene is affected by few (or no) others. Perturbations are introduced into the networks by altering the value of one or more of the nodes. This is analogous to the introduction of one or more mutations in a genome.

The Boolean models demonstrate that where epistatic interactions are high, the networks are highly unstable and chaotic. Where epistasis is small, but nonnegligible, remarkable things occur. The introduction of a "mutation" ramifies throughout a considerable portion of the network, but eventually the network compensates and settles back into its original stable configuration. Occasionally the network will be perturbed to the extent that it settles into a new stable configuration. These low-epistasis systems demonstrate two remarkable features: (i) the maintenance of stable homeostatic configurations despite perturbations and (ii) the origination of novel stable configurations in the face of other perturbations. We might call these features "stability" and "mutability" respectively.[16] They are analogous to the capacity of organisms to preserve and to initiate phenotypes.

These models are idealized and speculative; they are also highly suggestive. They have two important, related implications for adaptive evolution; one concerns the development of individuals and the other concerns the structure of populations. At the level of individuals, the models

15. Illuminating discussions of this work can be found in Kauffman (1993 and 1995), Depew and Weber 1995, and Burian and Burian 1996.

16. I have borrowed the terminology from Schwenk and Wagner 2003b.

suggest that the capacity of an organism to develop and maintain a stable configuration in the face of some perturbations, and to generate novel stable phenotypes in response to others, is determined by a particular feature of its genotype-phenotype map—*viz.,* the degree of epistasis. At the level of populations, Kauffman (1993 and 1995) notes that adaptive evolution occurs only in populations that inhabit certain kinds of adaptive landscapes. Adaptive landscapes with many adaptive peaks in which the fitness values (altitudes) of neighboring genotypes are highly correlated are propitious for adaptive evolution. The implication of the "many adaptive peaks" requirement is that many different gene combinations may confer significant adaptive advantages on individuals that possess them. The implication of the "high correlation" requirement is that a gene may be a reasonably good predictor of an individual's fitness largely independently of the effects of other genes. Consequently, a gene that confers an adaptive advantage in one genome will not cause a catastrophic disadvantage in another, similar genome. Nor will the contribution to fitness made by a particular gene tend to be changed significantly by the introduction of a new mutation. Kauffman's models demonstrate that the degree of epistasis within individuals determines the kind of landscape that a population inhabits. Where the degree of epistasis is low but not negligible, populations inhabit smooth, highly correlated, multiple-peaked landscapes.

Taken together the models suggest that the capacity of a population of organisms to undergo adaptive evolution is determined by the capacities of its individual members to mount compensatory developmental responses to perturbations and, on occasion, to produce novel, stable adaptive phenotypes. These capacities in turn are consequences of the interactions among genes in the expression of the genotype-phenotype map. Features of the genotype-phenotype map cause both changes in population structure and the adaptedness of individuals.

4.2. Evolutionary Developmental Biology. The emergence of evolutionary developmental biology in the last fifteen years or so has illuminated the way in which the features of the genotype-phenotype map influence adaptive evolution. A number of themes emerge that together lend strong support to the idea that the capacity of a population to undergo adaptive evolution is grounded in the development of its individuals. I survey a few of these, albeit cursorily, here.

4.2.1. Regulatory Evolution. An organism has in its genome a special set of "tool-kit" or regulatory genes that control development by regulating the timing or the products of expression of other genes. These regulatory effects are simply a form of epistasis. Biologists are coming to ap-

preciate the significance of the "tool-kit" in adaptive evolution. Regulatory genes and processes can direct the development of phenotypes *despite* differences in the underlying developing tissues and despite variations in the structural genes (Stern 2000). Additionally, changes in the regulatory role of genes can produce significant changes in the structure and function of phenotypes whose development they regulate. Major morphological novelties can be the product of relatively minor changes (e.g., duplications) to the suite of regulatory genes.

Carroll et al. identify two major roles for regulatory genes in the evolution of adaptive phenotypes.

> First, conserved regulatory circuits can be recruited for new roles during the development of novel morphologies . . . . In this way, large numbers of genes may be deployed in a novel structure with just a small number of regulatory changes. Second, evolutionary changes in gene regulation can facilitate morphological diversification of a novel character. As regulatory evolution modifies the genetic interactions within a developmental program, new patterns can emerge both within and between species. (2001, 158–159).

They continue: "The recurring theme . . . is the *creative role* played by evolutionary changes in gene regulation" (2001, 167). Regulatory genes play a dual role in the adaptive evolution of phenotypes. They contribute to the maintenance of adaptive form in the face of perturbations and they generate novel adaptive phenotypes. Regulatory interactions among genes in development are crucially involved in both the stability and mutability of biological form.

Regulatory genes have their distinctive capacities because they are arranged into modules. The modularity of regulatory genes has the effect that each gene regulates the development of only very few characters at a time. This promotes not only stability within the processes regulated by the gene modules, but makes novel, stable phenotypes more likely than they would otherwise be (Stern 2000).

4.2.2. Developmental Modularity. One salient effect of genetic modularity is that it promotes the organization of development into modules. A developmental module is a suite of integrated processes that control the development of some feature of a phenotype. The key characteristics of developmental modules are the high degree of integration between the elements within a module, and the dissociation of one module from another (Bolker 2000, Gass and Bolker 2003). The modularity of development contributes to adaptive evolution in a number of ways. First, modularity stabilizes developmental processes. A developmental module can preserve the integrity of a phenotypic character despite underlying variation in structural genes, regulatory genes, and developmental processes.

In this way, the phenotype controlled by a developmental module may be buffered against the effects of mutations (Gilbert 2001; Von Dassow and Munro 1999) and other "epigenetic" influences. Second, the dissociation of developmental modules allows phenotypic features to develop independently (to a degree) from one another. Changes occurring in one module do not redound negatively on other modules. "If that were not the case, all perturbations would produce monsters or death" (Raff 1996, 334). Third, modularity promotes the generation of phenotypic novelties. Changes in the kind or timing of interactions between modules, or in the number of modules, can result in new, stable phenotypes (Raff 1996). The modularity of development confers both stability and mutability on biological form.

4.2.3. Adaptability. Stability and mutability are crucial to adaptive evolution (Schwenk and Wagner 2001, 2003b). Stability requires of an individual that its phenotype be maintained—and reproduced with reasonable fidelity—despite perturbations. Mutability requires that an organism be the kind of thing that can generate novel phenotypes *without* deleteriously affecting the rest of the organism. In fact these preconditions on the adaptive evolution of phenotypes were pointed out long ago by Lewontin (1978). According to Lewontin, adaptive evolution requires "continuity" and "quasi-independence." Continuity is the condition wherein neighboring genotypes are correlated in their fitness (see Kauffman's high-correlation condition). Quasi-independence is the condition in which "in a reasonable proportion of cases" adaptive changes in one structure do not affect others. The studies on the role of epistasis in evolution, on regulatory relations among genes in development, and on the modularity of development suggest that these conditions on adaptive evolution are secured by features of the genotype-phenotype map. The features of development—genetic and developmental modularity, the degree of epistasis—that buffer phenotypes against environmental and genetic perturbations are also those that predispose organisms to generate adaptive phenotypic novelties (Wagner and Altenberg 1996; Gilbert 2001). In fact, Newman and Mueller (2002) have recently argued that it is the stability and mutability of phenotypic development that secures these features of the genotype-phenotype map.

These properties of development ought to be familiar to us. Buffering against perturbations and the production of novel stable states are the diagnostic features of Waddington's canalization (Debat and David 2001). On anybody's account, canalization is a form of developmental constraint, "the suppression of phenotypic variation" (Wagner et al. 1997).[17] I introduced canalization in order to illustrate the way that de-

17. Here too, the degree of epistasis is significant.Wagner et al. (1997) show that the degree of epistasis is one of the factors affecting the evolution of genetic canalization.

velopmental constraint is commonly thought to impede the power of natural selection to drive adaptive evolution. But in the context of recent work on the significance of development for evolution, it appears that constraint plays a much richer, more positive role. Constraint, as manifested in the actions of regulatory genes and developmental modules, promotes the stability of adaptive phenotypes *and* it predisposes developmental systems to generate stable adaptive novelties. Stability and mutability are both causal consequences of developmental constraint. Moreover, if stability and mutability of organisms is a causal requirement for the adaptive evolution of populations, a surprising conclusion follows: Developmental constraint causes adaptive evolution.

Here I think we encounter the second major error of the two-force model. It casts developmental constraint in the wrong role. According to the two-force model, constraint is relevant to explaining adaptive evolution only insofar as it impedes the adaptation-promoting power of selection. To be sure, the presence of developmental constraints may limit the range of adaptive phenotypes that might otherwise arise. But to suppose that this is the only role of developmental constraint in adaptive evolution is to miss out on the windfall provided by recent studies of the significance of development to evolution. These studies suggest, contrary to the two-force model, that if we are looking for the causes of the adaptedness of organisms, we should look at the ways that the dynamics of development constrains phenotype. The causes of the adaptedness of individuals are to be found within the processes of development.

The traditional two-force model of evolution, then, commits two substantive errors. First, it takes natural selection to be a force that causes the fit of individuals to their conditions of existence. Second, it construes developmental constraint merely as an impediment to the power of selection. If the discussion of Section 3 is correct, however, there is no reason to think of natural selection as a force. Natural selection theory explains by citing the statistical properties of populations. It does not identify the causes of adaptive evolution. If the discussion in Section 4 is correct, developmental constraint is no mere impediment to adaptive evolution; it is its principal cause. The two-force model, despite its enormous influence, is comprehensively mistaken.

5. Natural Selection, Development, and Adaptive Evolution. The relation between the explanatory roles of selection and development needs to be re-thought in light of the dual errors of the two-force model. Indeed, some revisions of the standard model have been suggested (e.g., Wagner 2000). Some authors (e.g., Debat and David 2001; Gibson and Wagner 2000; Schwenk and Wagner 2001) suppose that selection and developmental processes both play a significant role in explaining adaptive evolution, but

that ultimately the relevant features of development are themselves consequences of selective forces. Kauffman speculates that the distribution of biological form is largely a simple consequence of the principles of self-organization and that "natural selection may be the force which pulls complex adaptive systems into [the] boundary region" (1993, 219), where the generation and maintenance of stable phenotypes through self-organization is possible. Yet others contend that the features of development exert the strongest force on the distribution of form, whereas selective forces are weak (Goodwin 1995).

These revisions have their merits. But each inherits the errors of the two-force model. In each of these proposals, selection and development are seen as causal processes, or forces, that either cause or impede adaptive evolution. In many proposals, developmental processes are accorded a significant explanatory role. But in each of these, selection is seen as a (or the) cause of the adaptedness (the fit) of individuals to their conditions of existence. Once it is supposed that selection is the cause of the adaptedness of phenotypes, some alternative causal role must be found for the processes of development in order to preserve for them some explanatory role. This results in the forces of selection and development competing for explanatory relevance. Moreover, in none of the proposals of which I am aware is developmental constraint posited as a *cause* of adaptive evolution.[18] If, as the arguments of this paper suggest, selection is not a cause of the adaptedness of individuals, and developmental constraint is, we need another conception of the division of explanatory labor between selection and development.

Recall Darwin's insight, that the fit and diversity of organic form are jointly explained by the phenomenon of adaptive evolution. Explaining adaptive evolution, in turn, involves two distinct projects (i) explaining changes in *population* structure and (ii) explaining the adaptedness of *individuals.* Natural selection theory accomplishes the first of these: populations undergo changes as a function of their statistical structure. But it does not explain the properties of individual organisms, so it does not accomplish the second project. The second project, explaining the adaptedness of individuals, requires an account of the causal processes occurring within individuals that dispose them to preserve and initiate adaptive phenotypes. My suggestion is that evolutionary developmental biology offers the prospect of just that—a theory of the causes of adaptedness within individuals. In particular, developmental constraint confers on individuals the kind of stability and mutability required for the maintenance and initiation of adaptive phenotypes.

18. Constraint is often construed as a consequence of adaptive evolution (Debat and David 2001; Schwenk and Wagner 2003b), but not as a cause.

Explaining adaptive evolution further involves synthesizing these two distinct explanatory projects. I think the synthesis is to be effected in the following way. A theory of development gives us (part of) what Marjorie Grene calls "a causal study" of adaptive evolution. Features of the genotype-phenotype map cause individuals to maintain and to generate fit-enhancing phenotypes. Populations of individuals that vary in their possession of these fit-enhancing phenotypes naturally undergo changes in their structure.

It would be an error to conclude that the theory of natural selection would be rendered otiose by a mature developmental theory of the causes of adaptation. A developmental theory of the individual-level causes of adaptive evolution would not by itself explain why biological populations in general tend to undergo predictable changes in their trait structure. That is to say, it would not identify what populations of organisms have in common such that they are capable of undergoing adaptive evolution. This is precisely the role played by the genetical theory of natural selection in the Modern Synthesis. This theory tells us that all evolving populations have in common a particular statistical property, variation in the rates of change of their heritable traits (trait fitnesses). The theory allows us to abstract away from the specific causes of change within a given population, and to generalize across all biological populations.

Natural selection theory and a theory of development do not compete for explanatory relevance. Each is (potentially) complete in its own distinct domain. Nothing about individual-level causes needs to be added to the Genetical Theory of Natural Selection in order to get it to explain why populations change in their trait structure. Similarly nothing about the structure of populations would need to be added to a complete theory of adaptive development in order for it to explain its proprietary phenomena, viz., that organisms whose developmental processes constrain form in various ways preserve and initiate adaptive phenotypes. But only together do these two kinds of theory explain Darwin's dual explanandum. A statistical theory of natural selection alone does not explain why changes in the statistical structure of populations should lead to an increase in the adaptedness of individuals. It does not tell us *how* adaptive evolution occurs. It must be complemented by a causal account of the maintenance and generation of adaptive phenotypes within individuals. Conversely, an account of the developmental causes of adaptation within individuals could not explain why *populations* of such adaptive systems tend to undergo predictable changes in their structure. Nor does it tell us why adaptive evolution should be such a pervasive feature of biological populations. That requires a theory that adverts to the structure of those populations. Taken together, these theories tell us that when a population varies with respect to the developmental processes that cause the adaptedness of in-

dividuals it will undergo changes in its structure, and those changes will be ones in which the average adaptedness of individuals increases.

In this respect, a theory of development and a theory of natural selection are related, as are Newtonian mechanics and statistical thermodynamics. The first theory in each pair identifies forces operating at the level of individuals. The second explains ensemble-level phenomena that are the consequence of the statistical structure of populations. The first theory in each pair is a theory of forces; the second is a statistical theory. These two levels of theory yield complementary explanations. Given a population of molecules in a gas, Newtonian mechanics explains why the individual molecules behave in the way they do. But the distribution of mechanical forces operating within a given ensemble of molecules does not explain what all such ensembles have in common, by dint of which they behave in predictable ways. For that we need the theory of thermodynamics. Ensembles of molecules change in ways that are explained and predicted by their statistical structure. There is no competition for explanatory relevance between the ensemble-level statistical theories and individual-level mechanical theories.

In the same way, we ought to consider that there is no competition for explanatory relevance between a developmental theory of adaptation and the Modern Synthesis theory of natural selection. The relation is one of mutual dependence. The former explains (*inter alia*) the causes of adaptive evolution operating on and within individuals; the latter explains why populations of individuals that develop in this way change in their structure. Only together do they account for the phenomenon of adaptive evolution: that populations come to comprise individuals so well adapted to their conditions of existence. Only together do they explain the fit and diversity of organic form.

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