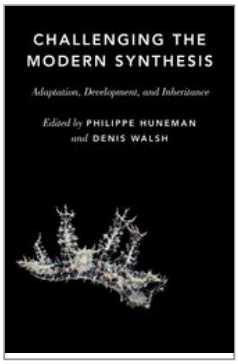

Challenging the Modern Synthesis: Adaptation, Development, and Inheritance
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“Chance Caught on the Wing”

Metaphysical Commitment or Methodological Artifact?

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Abstract and Keywords

Jacques Monod’s *Chance and Necessity* poses a paradox for modern biology: Organisms both must be and cannot be purposive systems. To resolve the paradox we must explain purpose by appeals to invariance or invariance by appeal to purpose. The methodology of modern science, however, allows no unreduced appeals to purpose. Monod traces the modern synthesis commitment to ineluctable chance back to its animadversion to teleology. He credits the pre-Socratic Atomist philosopher Democritus with holding that everything in the world is the fruit of chance and necessity. It is becoming increasingly obvious that their purposiveness is pivotal to the dynamics of evolution. This chapter outlines a ‘neo-Aristotelian’ alternative to the neo-Democritean commitments of modern synthesis biology, one that accords the purposiveness of organisms a central explanatory role in evolution.

Keywords: chance, purpose, mechanism, teleology, plasticity, gene repair, modern synthesis, Monod, Democritus, Aristotle

In his landmark book *Chance* and *Necessity: An Essay on the Natural Philosophy of Modern Biology*, Jacques Monod seeks to articulate and then to resolve what he perceives to be a paradox afflicting modern biology.[1] He calls it the “paradox of invariance,” and he leaves us in no doubt regarding its significance: “In fact, the central problem of biology lies with this very contradiction” (1971, p. 12).[2] The problem is to be found in a tension that Monod detects between scientific methodology and biological reality. On the one hand, “The cornerstone of the

scientific method is the postulate that science is objective.” Objectivity entails “the systematic denial that ‘true’ knowledge can be got at by interpreting phenomena in terms of … ‘purpose.’ ” On the other hand, “Objectivity nevertheless obliges us to recognize the teleonomic character of living organisms, to admit that in their structure they act projectively—realize and pursue a purpose” (p. 12).

“Living creatures are strange objects” (p. 17), Monod tells us. They exhibit two extreme—and contradictory—kinds of organizing principles: invariance$_1$ and purpose. They are invariant$_1$ in the sense of possessing an “ability to reproduce and to transmit *ne variateur* the **(p.240)** information corresponding to their own structure: A very rich body of information … which … is preserved in tact from one generation to the next” (p. 12). Equally, organisms are by their very natures purposive entities. Monod does not elaborate much, but at just about the same time, Ludwig von Bertalanffy made the latter point vividly:

> You cannot even think of an organism … without taking into account what variously and rather loosely is called adaptiveness, purposiveness, goal seeking and the like. (Von Bertalanffy, 1969, p. 45)

As Monod sees it, organisms are, and yet could not be, both fundamentally invariant and purposive—and that is the paradox of invariance$_1$.

In order to resolve the tension we must account for one of these evidently antithetical features of organisms in terms of the other. There are two options. We could strive to explain invariance$_1$—in particular the high fidelity of inheritance, the robustness of biological form, and the stability of DNA—as a consequence of organismal purposiveness, in this way: “Invariance is safeguarded, ontogeny guided, and evolution oriented by an initial teleonomic principle, of which all these phenomena are the purported manifestations” (Monod, 1971, p. 24). Alternatively, we could seek to ground the purposiveness of organisms by appeal to invariance$_1$. By this resolution, “all properties of living beings rest on a fundamental mechanism of molecular invariance” (Monod, 1971, p. 116). Scientific methodology—the “objectivity of science”—licenses only one of these strategies: the superordination of invariance$_1$ over purpose. So, in order to render evolutionary biology a science in good standing, we must seek to explain all its proprietary phenomena—the distribution and adaptedness of organismal form, the exquisite functional integration of organisms, their supple robustness—in terms of the preservation and transmission of invariant information.

This too raises a complication. Invariance$_1$ is stasis, but evolution is change. Moreover, *adaptive* evolution is biased change. Through the process of adaptive evolution, populations come to comprise organisms that are increasingly well suited to survival and reproduction in their conditions of existence. There must

be a constant source of new variants, and there must be a process that biases evolutionary change. We cannot suppose that the new variants that arise within organisms are biased in favor of their goals or purposes, on pain of violating the “objectivity of science.” So, the source of evolutionary novelties, Monod insists, must arise from unbiased—*chance*—alterations to the invariant$_1$ molecular structure that underlies organisms.

(p.241)

> The initial elementary events which open the way to evolution in these intensely conservative systems called living beings are microscopic, fortuitous, and utterly without relation to whatever may be their effects upon teleonomic functioning. (p. 118)

Thus, Monod’s *Chance and Necessity* elegantly traces modern synthesis biology’s commitment to ineluctable chance right back to its methodological source. The choice is between chance or purpose, and our hand is forced by the demands of scientific method, in the form of the “postulate of objectivity.” The postulate of objectivity is compulsory for scientific enquiry.

> The postulate of objectivity is consubstantial with science; it has guided the whole of its prodigious development for three centuries. There is no way to be rid of it … without departing from the domain of science itself. (1971, p. 12)

And it has no truck with purpose. Not that he is reluctant to embrace a commitment to chance:

> [Mutations] constitute the only possible source of modifications in the genetic text, itself the sole repository of the organism’s hereditary structures, *it necessarily follows that chance alone is at the source of every innovation, of all creation in the biosphere. Pure chance, absolutely free but blind, at the very root of the stupendous edifice of evolution*. (p. 112, emphasis added)

Evolution, in Monod’s resonant phrase, is “chance caught on the wing.”

My objective here is to question the commitment to ineluctable chance that, as Monod so astutely observes, is a cornerstone of modern synthesis evolutionary biology. Unlike Monod, I contend that the primacy of invariance$_1$ over purpose is not mandated on methodological grounds. Furthermore, I claim that it no longer seems plausible, even on empirical grounds. I wish to explore in outline the prospects for an evolutionary biology that prioritizes purpose over invariance$_1$. I call this “neo-Aristotelian” evolutionary biology and contrast it with the “neo-Democritean” cast of the modern synthesis. One surprising consequence of neo-Aristotelian evolutionary biology is that it relieves biology of the obligation to

accord an ineliminable role to chance. From the perspective of neo-Aristotelian biology, evolution is not fundamentally chancy; adaptive evolutionary change is inherent in the adaptiveness of organisms. What from the neo-Democritean **(p. 242)** biology is a compulsory metaphysical commitment, looks, from the neo-Aristotelian perspective, like an inapposite methodological artifact. Yet, ineluctable chance is so integral to our current neo-Democritean biology that removing that particular cornerstone threatens to undermine much of the edifice of the modern synthesis theory of evolution.

8.1. Neo-Democritean Evolutionary Biology

Monod’s project of banishing purpose from biology draws inspiration from the pre-Socratic Atomistic philosophers. The intellectual debt is acknowledged in his title. Monod credits Democritus with the claim that “everything in the world is the fruit of chance and necessity.” The Atomists, like Democritus and Empedocles, held that the fundamental units of matter are atoms of different kinds. Each kind of atom has a characteristic kind of motion. The world began in chaos—single atoms moving randomly in the void. Yet our world is clearly not chaotic. It consists of observable regularities among stable, complex, macroscopic entities. The world has become ordered because atoms encounter and combine with one another at random. Some combinations are ephemeral and some enduring. Stable combinations of atoms are differentially preserved as complex entities. These macroscopic entities behave in predictable ways because their characteristic properties are fixed—*necessitated*—by the properties and the arrangements of their constituent atoms. Ultimately, the order in the universe is the result of the chance encounters of randomly moving atoms, and the necessary consequences of those encounters (Hankinson, 1998). These same principles—chance and necessity—suffice to explain order in the biological realm.

> Empedocles, and others of his sort, consistently attempted to understand the features of mature organisms as coincidental outcomes of materially necessitated developmental processes. (Lennox, 2009, p. 357)

Monod’s choice of methodological archetype is apt. There are powerful resonances of pre-Socratic Atomism in modern synthesis evolutionary biology (Wicken, 1981). Atomism even has it own theory of the fit and diversity of organic form that, while not strictly *evolutionary*, bears a striking resemblance to our own.

> The generation of whole natured forms able to survive depended on the chance combination of fragments which were well adapted both to the needs of the whole creature and to the environment. The account of **(p. 243)** the origin of species by Empedocles is the first recorded account of the theory of natural selection. (Roux, 2005, p. 6)[3]

Modern synthesis biology takes genes or replicators to be evolutionary atoms, whose formation and whose coming together are random events. New replicators, and new combinations of replicators, arise by chance. The production of whole organisms, and their differential survival and reproduction, are causally necessary consequences of the activities of replicators. Modern synthesis biology can rightly be said, in an important sense, to be neo-Democritean.

Monod is to be commended for noticing how closely modern synthesis methodology cleaves to its Atomistic predecessor. The resemblance is so strong that it is tempting to speculate that any deficiencies of the ancestor may have been transmitted *ne variateur* to the descendant. A cogent argument against Atomism might well form the basis of a robust challenge to the modern synthesis.

The most ardent ancient opponent of pre-Socratic Atomism, especially its Democritean variant, is Aristotle. According to Aristotle, the telling deficiency of Atomism can be seen in the role that it accords to chance. The ordered features of the natural world, Aristotle contends, are not ineluctably chancy. Nature only *appears* fundamentally chancy when viewed through the polarizing lens of Atomistic methodology.

Aristotle’s argument can be found in part in *Physics* II.5 (Aristotle, 1996). He illustrates it with the story of a man “collecting subscriptions for a feast.” Our protagonist happens on a debtor, let us suppose in the marketplace, and extracts from him a payment. This encounter is strictly a matter of chance: “He actually went there for another purpose, and it was only incidentally that he got his money by going there.” So, although the encounter fulfills our agent’s purpose, it was not done *for the sake of* the agent’s purpose. The agent’s intentions or purposes form no part of the explanation of how they were fulfilled. On the other hand, if the agent *had* gone to the marketplace *in order to* collect the subscription, then the intention or purpose *would have* entered into the explanation.

This is a complex passage, but part of Aristotle’s point is that the same encounter might have occurred as a matter of chance, or for a purpose. Either way it would result in the payment of the debt. Moreover, either way it would get the same Atomistic explanation. The modern analogue would cite the various internal **(p.244)** physiological mechanisms that caused our agent’s limbs to move, various features of their shared ecological setting, various times and locations and so forth. These mechanisms and their capacity to explain their effects are completely insensitive to whether the outcome is a chance occurrence or a matter of design. If there is an important difference between the

occurrence of an event by chance or by purpose, it is invisible to Atomistic explanation.

Yet there *is* a difference between chance events and purposive events. It is most clearly seen in their respective modal profiles. Chance and purposive occurrences are robust across different ranges of counterfactual circumstances. Purposive occurrences are robust across a range of alternate background conditions and mechanisms. For instance, if this were a purposive encounter, we would expect it to be somewhat *insensitive* to initial conditions, like location. Had the debtor been somewhere else in the market, or at the bath, or at the barber, then the encounter might well have occurred anyway, only in a different place, by a different set of mechanisms. Our protagonist would be expected to have done whatever was necessary, under the circumstances, to ensure that the event occurred. In contrast, chance events are highly sensitive to initial conditions. If this were a *chance* encounter, then had the specific spatiotemporal circumstances been different, had the mechanisms been different, then in all likelihood, the event in question—the exchange of money—would not have occurred. Then again, purposive occurrences are highly *sensitive* to goals in a way that chance occurrences are not. If this were a purposive occurrence, then had the agent’s goals been different—say to collect money from someone else, to avoid his friend, to walk the dog in the country—the encounter probably would have not occurred at all.

Events that occur because they fulfill a purpose are thus subject to two distinct kinds of explanations, one that identifies the occurrence as resulting from mechanical interactions, the other that identifies it as occurring because it conduces to the fulfillment of a goal (Walsh, 2012, 2013). In contrast, a chance occurrence is subject (at best) only to a mechanistic explanation.[4] Aristotle’s claim is that restricting ourselves to only one kind of explanation leads us to miss out on at least one important kind of scientifically explainable regularity. As a result, certain perfectly explainable regularities may be erroneously dismissed as inexplicable chance occurrences.

Neo-Democritean biology—like its Atomistic precursor—is unequipped to recognize the significance of events that might occur because they fulfill purposes. **(p.245)** Evolution is exclusively to be explained in terms of chance variations and their necessary consequences. This is no mere oversight. It is borne of the conviction that Monod identifies, *viz.* that purpose has no role in scientific explanation. But here neo-Democritean evolutionary biology makes a bold empirical wager, that the pursuit of organismal purposes makes no appreciable contribution to organic evolution. It might look rather more like backing the favorite each way. After all, purpose has been all but expunged from the natural sciences since the advent of the scientific revolution, so why not bet against it. Challenging neo-Democritean biology on these grounds would require showing that natural purpose can earn its keep in biology. That, in turn, requires

two things: (1) a compelling case that purposes can figure in genuinely scientific explanations, and (2) demonstrating that such explanations are needed for the project of explaining evolution.

8.2. Natural Purpose

The principal impediment to purposive explanation in the natural sciences seems to be comprehensiveness of mechanism. If every event has a complete mechanistic explanation, mechanism leaves no unexplained residuum. There is nothing left over for purpose to explain.

Immanuel Kant’s (2000/1790) discussion of teleology is illuminating in this regard. Kant evinces an uncommon sensitivity to the need for purposive explanation in biology, but still he worries that this form of explanation is unavailable to the natural scientist. Kant reminds us that organisms are unique in the world. They are *self*-building, *self*-nourishing, *self*-reproducing entities (McLaughlin, 1990). Organisms pursue purposes that inhere in their life activities. The parts of organisms are not only appropriate to the production of the organism’s characteristic activities, they possess these properties precisely *because* of the organism’s pursuit of those activities. So, while the activities of an organism are consequences of the properties and mechanical interactions of its parts, the properties and interactions of the parts are the consequence of the activities of organisms. There is a “reciprocal causation” between organisms as a whole and the activities of the parts. In this organisms are wholly distinctive, and it is this, according to Kant, that makes them *natural purposes*.

> The definition of an organic body is that it is a body, every part of which is there for the sake of the other (reciprocally as an end, and at the same time, means)… . An organic (articulated) body is one in which each part, with its moving force, necessarily relates to the whole (to each part in its composition). (*Opus postumum*, quoted in Guyer, 2005, p. 104) **(p.246)**

> I would provisionally say that a thing exists as a natural end if it is cause and effect of itself. (Kant, 2000/1793, p. 371)

Mere nonpurposive mechanism evidently cannot wholly account for the marvelous integration, the supple adaptation, the self-regulating ability that is constitutive of organisms. We can only make sense of these magnificent features of living things by considering them as natural purposes.

The Aristotelian resonances are evident and profound.[5] The reciprocity that Kant sees in organisms as natural purposes is reflected in Aristotle’s multiplicity of explanatory modes. For Kant organisms are both causes and effects of the activities of their parts. For Aristotle, the capacities of an organism’s parts constitute the efficient cause of organismal purposiveness. And, reciprocally, the purposiveness of organisms is the final cause of the capacities of their parts. Like Kant, Aristotle stresses that the functional unity of organisms calls for

special explanation. According to Aristotle “*βιος*” (way of life) is a teleologically basic capacity that explains the integration of an organism’s various parts and activities:

> [*βιος*] accounts for the unity that integrates the many parts of an animal’s body and the many different activities those parts perform. That understanding comes from the recognition that an animal’s functional parts must make coordinated contributions to a single way of life. (Lennox, 2010, p. 350)

There is a crucial difference between Kant and Aristotle, however, on the implications of this reciprocity for a science of biological form. For Kant the need for teleology puts organisms beyond the ambit of scientific explanation. For him the paradigm of scientific explanation is Newtonian mechanism—to explain a phenomenon is to show it to be a consequence of mechanical law. But the way in which the parts of organisms, and their characteristic activities, arise out of the purposive, self-synthesizing capacities of organisms simply does not conform to the mechanistic mode of explanation.

> It is quite certain that we can never adequately come to know the organized beings and their internal possibility in accordance with merely mechanical principles of nature, let alone explain them; and indeed this is so certain that we can boldly say that it would be absurd for humans even to make such an attempt or to hope that there may yet arise a Newton **(p. 247)** who could make comprehensible even the generation of a blade of grass according to the natural laws that no intention has ordered; rather, we must absolutely deny this insight to human beings. (Kant, 2000, p. 400)

Aristotle, Kant, and Monod are responding to the same problem, the tension between the evident need to explain the features of organisms by appeal to purpose, and the presumed naturalistic stricture against doing so. Yet their respective approaches are radically different. In effect, Kant despairs of any naturalistic solution (Zammito, 2006). Monod’s attempted resolution, in contrast, is an orthodox version of modern naturalism. His strategy is simply to deny that the purposiveness of organisms has any ineliminable explanatory role to play. For Aristotle, purposive explanation is in no way antithetical to naturalism. Between them, these authors exemplify three strategies for dealing with the “paradox of invariance$_1$.” We can either repudiate naturalism (Kant), repudiate teleology (Monod), or simply deny that there is a paradox of at all (Aristotle).

The Aristotelian strategy requires us to show how purposes could actually figure in scientific explanations. A recent and compelling approach to explanation might help here. In order to explain some event X by appeal to Y, the account goes, there must be an invariance$_2$ relation between X and Y. James Woodward captures the relation between invariance$_2$ and explanation succinctly:

> On my view, the key feature that a generalization must possess if it is to figure in explanations is invariance. Invariance is a kind of robustness or stability property: a generalization is invariant if and only if it would continue to hold under some range of physical changes involving intervention… . To explain why an explanandum *Y* takes some particular value we need to identify some variable *X* and a generalization *G* linking *X* to *Y* such that, according to *G*, some range of changes in the value of *X* that are due to interventions are associated with changes in the value of *Y*. This requires that the generalization G must be invariant under some interventions on *X* that change the value of *Y*. (Woodward, 2001, p. 4)

Invariance$_2$ is just a form of robust counterfactual dependence. An explanation proceeds by demonstrating the way in which the value of the explanandum variable, *Y*, counterfactually depends on the value of the explanans variable, *X*, by providing an illuminating description of the relation (Walsh, 2012, 2013).[6]

(p.248) Aristotle’s taxonomy of efficient and final cause explanations translates easily into the modern parlance of counterfactual dependence (invariance$_2$) relations. Causes explain their effects because effects counterfactually depend on their (efficient) causes. Similarly, purposive systems manifest a different kind of counterfactual dependence: *Means counterfactually depend on their ends*. If *Y* is a system’s goal and *X* is a means to the attainment of *Y*, then intervening on *Y* (changing the goal) will occasion a change in *X* (the means). Similarly, where *Y* is a system’s goal and *X* is a means to *Y*, then changing the background conditions will occasion a change in *X*. In either case, the change in *X* will be predictable, when specified in terms of ends. The change in *X* will be such as to bring about the end, *Y*. This purposive invariance$_2$ relation has exactly the same counterfactual form as the mechanistic invariance$_2$ relation.[7]

This modernized version of Aristotelian explanation holds that if invariance$_2$ underwrites mechanistic explanation, it should licence purposive explanations too. Far from succumbing to Kant and Monod’s methodological qualms, it urges that as naturalists we should avail ourselves of purposive explanations. It remains to be seen whether this taming of purpose (to coin a phrase) provides any opportunities for modern evolutionary biology.

8.3. Plasticity and Purpose

The purposiveness of organisms is manifested in their adaptive plasticity and robustness. Plasticity is the capacity of an organism to adapt to the vagaries of its conditions of existence by controlling and implementing changes to its own structures and processes. Robustness is the capacity of organisms to maintain constant form and function across a range of circumstances (Kitano, 2004). Plasticity and robustness are two sides of the same coin (de Visser et al., 2003).

> The organism is not robust because it is built in such a manner that it does not buckle under stress. Its robustness stems from a physiology that is adaptive. It stays the same, not because it cannot change but because it compensates for change around it. The secret of the phenotype is dynamic restoration. (Kirschner & Gerhard, 2005, pp. 108–109)

Adaptive phenotypic plasticity is not an optional extra for organisms. *Organism*, as Monod, Kant, and Aristotle all agree, is a purposive category. “Phenotypic **(p. 249)** plasticity is a ubiquitous, and probably primal phenomenon of life” (Wagner, 2011, p. 216). These purposive capacities are manifest at practically all levels of organization, from gene networks, to genomes, to cells, to entire organisms (Keller, 2013). And their importance for evolution is increasingly becoming recognized.

8.3.1. Gene Networks

Genes, as we are becoming aware, play their characteristic role in development as parts of complex suites or networks with complex regulatory topologies (Davidson, 2010, Meir, von Dassow, Munro, & Odell, 2002). Ciliberti, Martin, and Wagner (2007) and Wagner (2011, 2012) have revealed some of the startling ways that gene networks contribute to evolution. These studies demonstrate that the robustness of gene networks is crucial both to the origin of evolutionary novelty and to the stability of organismal form.

The gene regulatory networks under study begin with initial input levels of gene products and a particular topology of regulatory relations between elements of the network. These networks settle down into a stable equilibrium output. This is taken to be their “phenotype.” “Mutations” are then introduced, as either quantitative or qualitative changes in gene products, or in the regulatory relations among genes in the networks.

Ciliberti et al. (2007) and Wagner (2011) map the outputs of these networks in a vast multidimensional “network space,” the axes being the values of an individual gene’s function (regulatory activities or products of transcription). Along any given axis, immediate neighbors differ from one another by a single genetic mutation. In general, in these systems there are many more genotypes than phenotypes: “This means any one phenotype typically has many genotypes that form it” (Wagner, 2011, p. 71). The space of viable phenotypes exhibits some remarkable features: it is *clustered* and *connected*. Viable networks are clustered in the sense that the genotypes that produce a particular phenotype all occupy a single small region of the state space. They are connected in the sense that every single network capable of producing the system’s characteristic phenotype can be accessed from any other through a series of single mutations without leaving the space of viable networks. Such a connected network of networks is called a “neutral network.”

Gene networks are robust. They can compensate for perturbations by producing changes in function that preserve their viable phenotype. Robustness is measured as the number (proportion) of nearest (single-mutation) neighbors in the neutral network that produce the typical phenotype. Gene networks with many viable neighbors are capable of withstanding a considerable degree of **(p. 250)** perturbation, both mutational and environmental. Wagner (2011) in fact demonstrates the enormous capacity of such systems to maintain their stability under mutational change. For gene regulatory networks of a size typically found in biological systems, two networks producing the same phenotype may share only 20% of their regulatory interactions (Wagner, 2011, p. 721). The constancy of form of organisms, the reliability of development, the transgenerational recurrence of form, are secured in large part by the adaptive robustness of gene regulatory systems.

The robustness of gene networks facilitates adaptive phenotypic evolution in at least three distinct ways. First, one and the same gene network can produce multiple phenotypes. The neutral networks that underwrite different phenotypes overlap. So, a gene network may move from one neutral network (where it produces one phenotype) to another (where it produces a novel phenotype) with no genetic change. Second, typically a gene network will have novel phenotypes as its near neighbors, so phenotypic novelties may be produced as an adaptive response of networks to minor mutations. In these ways, plasticity allows a gene network to innovate.[8] Novel phenotypes may be initiated by mutations or environmental changes. Third, robustness acts as an evolutionary capacitor. A gene network that can maintain its stability across a range perturbations has the capacity to access a large range of novel phenotypes (Wagner, 2012).

One startling feature of the adaptiveness of gene networks is that the production of novelties is biased. To be adaptively plastic *just is* to produce compensatory changes to perturbations precisely because those changes are conducive to the viability of the system. That goes as much for compensations that maintain phenotypic stability as for compensatory changes that produce new phenotypes. The new phenotypes that these systems generate tend to be stable and viable.

8.3.2. Reactive Genomes

Research in developmental genetics over the last 20 years or so has issued in a gradual shift in emphasis from discrete genes as bearers of phenotypic information to genomes, as complex, reactive systems:

> The transition that concerns us has involved genomes rather than genes being treated as real, and systems of interacting macromolecules rather **(p.251)** than sets of discrete particles becoming the assumed underlying objects of research. (Barnes & Dupré, 2008, p. 8)

Reactive genomes are not repositories of phenotypic information—or “programs”—as such. Instead they are open systems that exploit the resources of their genetic, cellular, extracellular, and environmental circumstances in the production of stable, viable forms. Just as genes influence biological form, biological form affects the functioning of genes. “The passage of information is not simply one-way, from genes to function. There is two-way interaction” (Noble, 2006, p. 50).

Genomes are complex, adaptive systems. They respond to genetic, organismic, and environmental cues. They buffer the organism against perturbations.

> At the very least, new perceptions of the genome require us to rework our understanding of the relation between genes, genomes and genetics… . It has turned our understanding of the basic role of the genome on its head, transforming it from an executive suite of directorial instructions to an exquisitely sensitive and reactive system that enables cells to regulate gene expression in response to their immediate environment. (Keller, 2013, p. 3)

We cannot understand the way that genes, cells, whole organisms, or environments contribute to the development of an organism unless we understand how genomes react to these influences. The reactive, adaptive dynamics of genomes is the hallmark of a goal-directed, purposive system. Organismal purposes, then are reflected in the dynamics of genomes.

8.3.3. Developmental Plasticity

There are architectural requirements for complex systems to be evolvable, which essentially requires the system to be robust against environmental and genetic perturbations (Kitano, 2004, p. 829). As discussed previously, phenotypic plasticity is “the ability of an organism to react to an environmental input with a change in form, state, movement or activity” (West Eberhard, 2003, p. 37). It might be added that it also consists in the capacity to respond to an *internal* input in the same way. An organism has the capacity to produce a wide range of phenotypes, according to the circumstances.

> Through its ancient repertoire of core processes, the current phenotype of the animal determines the kind, amount and viability of phenotypic **(p. 252)** variation the animal can produce … the range of possible anatomical and physiological relations is enormous. (Gerhard & Kirschner, 2007, p. 8588)

As in the case of gene networks and genomes, plasticity allows organisms to maintain stability in the face of genetic and environmental perturbations. But it also permits them to innovate; often enough they produce novel, stable phenotypes when perturbed.

> Adaptive phenotypic adjustments to potentially disruptive effects of the novel input exaggerate and accommodate the phenotypic change. *Without genetic change*. (West-Eberhard, 2005, p. 613, emphasis in original)

The production of a novel phenotype has potentially catastrophically disruptive effects on the organism as a whole. But here again, the plasticity of development buffers the organism against these. This adjustment to phenotypic change is known as “phenotypic accommodation,” and it is crucial to development: “Phenotypic accommodation reduces the amount of functional disruption occasioned by developmental novelty” (West-Eberhard, 2003, p. 147).

Accommodation, it turns out, is a prerequisite for adaptive evolution (Schlichting & Moczek 2010). The evolution of complex adaptations requires a significant degree of orchestration between an organism’s various systems. For example, the increase in the strength of a striated muscle carries with it demands not just on the muscular system but the associated skeletal, nervous, circulatory, and integumentary systems (Sterelny, 2009). Each system responds adaptively to its changed circumstances, in a way that accommodates the new structure and its altered function. If each system had to wait for a fortuitous mutation in order to produce the appropriate accommodation, complex evolutionary adaptations might never arise.

> In contrast to the rapid response produced by plasticity, if the production of newly favored phenotypes requires new mutations, the waiting time for such mutations can be prohibitively long and the probability of subsequent loss through drift can be high. (Pfennig et al., 2007, pp. 459–460)

Recent developmental biology, then, appears to suggest that the adaptive plasticity of organisms positively contributes to the origin and evolution of organismal form.

> These considerations all point to the importance of adaptive plasticity for the adaptive evolution of organismal form. Plasticity consists in the capacity to respond adaptively to genetic, epigenetic, and environmental **(p.253)** influences, in the production of viable, robust living things. This plasticity is evident at every level of biological organization (Keller, 2013).

Plasticity is merely a manifestation of organismal purposiveness. The purposiveness that makes organisms organisms appears to be a necessary precondition of adaptive evolution.

8.4. Purpose and Genetic Stability

Monod’s solution to the “paradox of life,” as we saw, was to ground organismal purposiveness in the unchanging (invariant$_1$) structure of the “gene code.” “All properties of living beings rest on a fundamental mechanism of molecular invariance” (p. 116). This strategy presupposes an asymmetry; whereas the

constancy of genes can explain the purposiveness of organisms, the purposiveness of organisms could not possibly explain the constancy of genes. The explanatory asymmetry is predicated on the conviction that there is no way that the structure of DNA could be changed in response to the needs of an organism.

> There exists no conceivable mechanism whereby any instruction or piece of information could be transferred to DNA… . Hence the entire system is totally, intensely conservative, locked into itself, utterly impervious to any “hints” from the outside world. (Monod, 1971, p. 110)

Indeed, the idea that the structure of the genome is stable and impervious to the influences of the organism is a bedrock commitment of the modern synthesis.

Yet, there is mounting empirical evidence that the structural integrity of DNA is not an evolutionary primitive. It is safeguarded by the supple adaptiveness of organisms. The DNA damage repair (DDR) system offers vivid evidence of the ability of organisms to secure the invariance$_1$ of their DNA. An organism’s DNA is subject to a battery of endogenous and exogenous insults. These cause lesions —structural alterations—at an estimated rate in humans on the order of 150,000/cell/day (Ciccia & Elledge, 2010). These are by no means neutral or benign.

> These lesions can block genome replication and transcription, and if they are not repaired or are repaired incorrectly, they lead to mutations or wider-scale genome aberrations that threaten cell or organism viability. (Jackson & Bartek, 2009, p. 1071)

Cells (organisms) have evolved elaborate systems for detecting and repairing DNA damage. The systems comprise various mechanisms for determining the **(p.254)** nature and severity of the lesion, and marshaling the appropriate response. The response may involve mitigating the consequences to the organism of DNA damage through the disruption of mitosis, or through apoptosis (Branzei & Foiani, 2008), or it may consist in altering the structure of damaged DNA, restoring its former nucleotide sequence.

> The DDR system is a highly responsive, adaptive system. The DNA damage response (DDR) is a signal transduction pathway that senses DNA damage and replication stress and sets in motion a choreographed response to protect the cell and ameliorate the threat to the organism. (Ciccia & Elledge, 2010, p. 180)

The famed stability of the genome, then, is not so much due to its being inherently “intensely conservative,” as Monod would have us believe, but to the fact that it is actively monitored and repaired by a highly adaptive goal-directed

system that detects errors and implements the appropriate cascade of responses, in ways that safeguard the organism’s viability.

There is, then, no particular asymmetry between gene structure and organismal purpose. The structural stability of genes is involved in securing the purposiveness of organisms, but conversely, the purposiveness of organisms is involved in securing the structural stability of genes.

> The stability of gene structure thus appears not as a starting point but as an end-product—as the result of a highly orchestrated dynamic process requiring the participation of a large number of enzymes organized into complex metabolic networks that regulate and ensure both the stability of the DNA molecule and its fidelity in replications. (Keller, 2000, p. 31)

We need to appeal to the purposiveness of organisms to explain the invariant$_1$ structure of DNA. This is an example of the very relation between “purpose” and “invariance” that Monod sought to proscribe. That being so, the envisaged reduction of the purposiveness of organisms to the invariant$_1$ structure of DNA is not viable. Invariance$_1$ is neither conceptually nor causally prior to organismal purposiveness.

8.5. Neo-Aristotelian Evolutionary Biology

Adaptive evolution requires three things: (1) the constancy of form—organismal form must be stable, and reliably generated from generation to generation; (2) a source of novelties; and (3) adaptively biased change. The case for a **(p.255)** neo-Aristotelian evolutionary biology can be made by demonstrating that organismal purposiveness is crucially involved in each of these phases.

8.5.1. The Constancy of Form

According to the neo-Democritean modern synthesis theory, form is reliably reproduced and passed on from generation to generation because the processes that produce individual organisms—replication and development—are fundamentally conservative (Lewens, 2009). However, a consideration of the importance of adaptive plasticity in securing the inheritance of phenotypes suggests that the intergenerational stability of phenotypes across a huge range of internal and external perturbations is underwritten by the capacity of organisms to make adaptive adjustments to the vagaries of genome and environment. Moreover, form is reliably inherited, reliably produced, and reliably maintained, not because the processes that occur within organisms are inherently *conservative*, but because they are *adaptive*.

If inheritance of form requires the stability of the genotype, that too is to be explained, to a significant degree, by the adaptive, reactive capacities of organisms. The integrity of the genome is secured by adaptive responses to DNA damage.

8.5.2. Novel Variants

Neo-Democritean biology holds that the only source of evolutionary novelty is genetic mutation. Typically, mutations have little or no effect on phenotype. However, even on those occasions when phenotypic novelties are *initiated* by mutations, the new phenotypes are themselves the consequence of the reactive response of the genome and the organisms’ various developmental systems to the perturbations. It appears that phenotypic novelties are principally caused by the adaptive response of organisms to perturbations of any sort, genetic, epigenetic, or environmental.

> Responsive phenotype structure is the primary source of novel phenotypes. And it matters little from a developmental point of view whether the recurrent change we call a phenotypic novelty is induced by a mutation or by a factor in the environment. (West-Eberhard, 2003, p. 503)

Novel, stable phenotypes could not result from perturbations of any sort were it not for the responsiveness of organisms, their genomes, and their gene regulatory networks. The principal cause of novel phenotypes is the plasticity of the **(p.256)** organism, whether or not those novelties are *initiated* by mutation or by environmental change.

8.5.3. Adaptively Biased Change

Neo-Democriteanism is committed to the view that the source of phenotypic novelties is adaptively unbiased—mutations are random and “utterly without relation to whatever may be their effects upon teleonomic functioning” (Monod, 1971, p. 118). But, if novelties arise from the reactive response of organisms to perturbations, this claim might require some reconsideration. Phenotypic accommodation enables organisms to mitigate the potentially disruptive consequences of novel phenotypes. Robust gene regulatory networks make the alterations they do to their activities because these alterations preserve the proper functioning (the phenotypic output) of the system. So, while mutations may be random and indifferent to organismal well-functioning, the way in which organisms generate novelties in response to them is not.

It would appear, then, that contra the convictions of Neo-Democritean modern synthesis biology, the purposiveness of organisms—as manifested in their robust adaptive plasticity—is not available to figure in genuine scientific explanations of biological phenomena, it is crucially implicated in each of the factors required for adaptive evolution: the constancy of form, the production of novel heritable variants, and the adaptive bias in form. A neo-Aristotelian evolutionary biology would take seriously the significant role that organismal purposes play in securing the conditions necessary for adaptive evolution.

8.6. Chance on the Wing

The purposiveness of organisms is an empirically observable phenomenon, as Monod himself concedes. Yet, the “postulate of objectivity” prohibits explanations that advert to those purposes. In asserting the postulate of objectivity Monod is reaffirming one of the most fundamental methodological commitments of modern synthesis evolutionary biology. The methodological commitment, in turn, generates what I described earlier as the great empirical wager of the modern synthesis, namely, that organismal purposes make no substantive contribution to the process of evolution. It is becoming increasingly evident that the wager will not pay out, for two reasons. First, the stricture against purposive explanation is not well motivated on methodological grounds. Purposive explanation is available to the natural sciences. Secondly, it is unsustainable on empirical grounds. Organismal purposes make an observable difference to evolution. Advances in biology increasingly suggest that purpose is indispensable to **(p.257)** a comprehensive understanding of evolution. The reason is simple, but wholly contrary to modern synthesis orthodoxy: Evolution is adaptive because organisms are purposive.

One of the consequences of the proscription against purpose is that it forces on biology a commitment to the ineluctable role of chance in evolution.

> It necessarily follows that chance alone is at the source of every innovation, of all creation in the biosphere. Pure chance, absolutely free but blind, at the very root of the stupendous edifice of evolution. (Monod, 1971, p. 112)

From the “neo-Aristotelian” perspective, the modern synthesis commitment to chance looks ill-conceived. The “source of every innovation” is not random mutation, but the reactive, adaptive response of an organism’s myriad systems to influences from genes, cells, tissues, and environments. The influences may be chancy—mutations really are indifferent to an organism’s viability—but the responses are not. They are biased by the capacity of organisms to buffer themselves against perturbations, to adapt, to compensate, to orchestrate, to accommodate, to innovate. These are manifestations of organismal purposes. As a consequence, adaptive evolution is not ineluctably chancy. It is inherent in the purposiveness of organisms. If our scientific methodology fails to countenance purpose, then it renders us incapable of explaining these perfectly real, empirical regularities. The upshot is that what ought to be explicable is dismissed as the whim of capricious chance: “pure chance, absolutely free but blind.” This is a mistake. Evolution merely *appears* ineluctably chancy if we are blind to the role of purpose. From the perspective of a neo-Aristotelian evolutionary biology, the modern synthesis conviction that evolution is “chance caught on the wing” does not deserve its status as a bedrock metaphysical commitment. It is an unfortunate methodological artifact.

8.7. Conclusion

Since its inception in the early 20th century, the modern synthesis theory of evolution has been guided by a methodology that explicitly prohibits explanations of phenomena in the natural world that appeal to the fulfillment of goals or purposes. This is hardly surprising. The natural sciences comprehensively eschew purposes. Nevertheless, it sits uneasily with the obvious fact that *organism* is a purposive category. Increasingly, it is becoming apparent that the purposiveness of organisms, as manifest in the robust, reactive, adaptive plasticity of their various systems, from gene networks to entire organisms, is pivotal to the process of evolution. Yet, we **(p.258)** still have an evolutionary biology of genes and chance, rather than of organisms and purpose.

The principal impediment to an understanding of the role of organisms in evolution, in my view, is not empirical but methodological, or perhaps more importantly historical. The stricture against natural purpose, Monod’s “postulate of objectivity,” is one of the great legacies of the scientific revolution. The scientific revolution, of course, was not principally motivated by the needs of biology, much less by an understanding of *evolution*. There is no guarantee that its methods are adequate to the task. Monod’s methodological orthodoxy is just one example of a puzzling syndrome evident throughout the growth of the modern synthesis. When 16th-century methodology rubs up against 20th- and 21st-century biology, philosophers and biologists alike have overwhelmingly chosen to side with the former. I think we should feel no compunction about throwing our lot in with the latter.

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Notes:

(1.) Monod’s term is a “flagrant epistemological contradiction.”

(2.) Unfortunately, the term “invariance” and its cognates are used in two drastically different ways in the literature drawn on in this paper—awkward. Where the use of these terms cannot be avoided, and the context does not wholly disambiguate, I shall index them: “invariance$_1$” denotes Monod’s conception of invariance, and “invariance$_2$” denotes Woodward’s.

(3.) Dawkins’s (1989) scenario of assembling the best rowing eight by randomly assigning combinations of rowers to a boat, and then selecting the fastest is a nice analogy. It too is meant to illustrate how the random aggregation of parts produces well-functioning ordered, differentially persisting aggregations.

(4.) Some chance occurrences, like radioactive decay, may have no mechanistic explanation.

(5.) Ginsborg (2004) offers a lovely discussion.

(6.) Invariance is necessary, but not sufficient, for explanation. In the case of purposive explanations, one must also provide a description of how the means conduces to the end (Walsh, 2012, 2013).

(7.) Except it is time reversed. The earlier event counterfactually depends on the later. Note, this does *not* imply backward causation.

(8.) Andreas Wagner (2014) calls this capacity of biological systems to innovate “innovability.” It is manifest at practically all levels of biological organization—gene networks, genomes, cells, tissues.