What Darwin Got Wrong
Update for the paperback edition: Replies to our critics

1. On our book and its reception

The hardcover edition of this book was first published early in 2010. It was intended to raise two objections to the Theory of Natural Selection (TNS) and to explore their connections to each other and to familiar questions about evolution. First, we claimed that TNS is committed to an untenable externalism: Like Skinner, Darwin held that paradigm explanations of biological (and psychological) structure should invoke relations between organism and their ecologies. But, whereas Skinner's externalism was largely motivated by his methodological commitment to behaviorism, Darwin's was quite different; Darwin held that externalism is the price one pays for adaptationism: only an externalist theory could explain why the features of a creature's phenotype are so often well-adapted to the features of its ecology. The explanation on offer is that phenotypes are shaped by the ecological features to which they are adapted. We suggested, by contrast, that the appearance of adaptation is in large part illusory. The reason a creature's phenotype seems well-adapted to its ecology is that *by definition*, an "ecological feature" is one to which the fitness of phenotypic traits is sensitive; and a "phenotypic trait" is *by definition*, one that effects a creature's fitness in relation to its ecology. We aren't, of course, the first to suspect that there are vicious circularities lurking at the heart of TNS. But we have tried to make them explicit, and to document a variety of recent empirical findings that strongly suggest the crucial role of *endogenous* variables in the evolution of phenotypes. About half of our book is devoted to doing so.

The second problem we raised for TNS has, to our knowledge, hardly been noticed elsewhere in the literature: the tension between its treatment of *selection* and its treatment of *selection-for*. TNS holds, in effect, that though what get *selected* are kinds of creatures (kinds of creatures are what flourish, or fail to, in a given ecology), what creatures get *selected-for* are certain of their phenotypic traits (viz those phenotypic traits that cause their fitness.) Problems arise because, unlike selection, selection-for is a paradigmatically *intensional* concept: it is perfectly possible that there should be selection-for one, but not the other, of two coextensive phenotypic traits. The intensionality of selection-for is duly inherited by a variety of other notions that are interdefined with it, and to which TNS is committed. These include, in particular, the notion of a phenotypic trait itself (since one but not the other of coextensive phenotypic traits may be selected-for). This we suggest, is the logical consideration from which the notorious problems about "arches and spandrels" eventually arise. We argue that because selection-for is intensional and selection is not, TNS can't, even in principle, decide which of its traits is selected for when a kind of creature is selected. This should hardly be surprising; there is an exactly parallel situation in cognitive psychology, where the intensionality of the "propositional attitudes" - beliefs, desires, and the like - offers a prima facie objection to the naturalizability of "Representational" theories of mind. That there is this previously widely ignored analogy between the (putative) intensionality of mental processes and the (putative) intensionality of evolutionary processes is one of the things that make the present issues philosophically interesting.

Our claim is that, given coextensive phenotypic traits, TNS can't distinguish ones that are causally active from ones that aren't. Many of the objections that have been raised against us seem unable to discriminate this claim from such quite different ones that we didn't and don't endorse, such as: when traits are coextensive, there is no fact of the matter about which is a cause of fitness; or, when traits are coextensive, there is no way to tell which of them is a cause of fitness; or when traits are coextensive Science cannot determine which is a cause of fitness…etc. Such views are, we think, preposterous on the face of them; we wouldn't be caught dead holding them. To the contrary, it is precisely because there *is* a fact of the matter about which phenotypic traits cause fitness, and because there is no principled reason why such facts should be inaccessible to empirical inquiry, that the failure of TNS to explain what distinguishes causally active traits from mere correlates of causally of active traits, shows that something is seriously wrong with TNS.

We were, on balance, very pleased the way our book turned out. It seemed to us quite plausible, in the light of the considerations it raised, that TNS is simply untenable and that, insofar as current evolutionary theory presupposes it, current evolutionary theory is due for a thorough reconsideration. We thought of this as a real scientific advance; the next best thing to finding out what one ought to believe is finding out what one ought not. We didn't exactly expect to be awarded a tickertape parade, of course; but we were looking forward to at least a few warm congratulations. In the event, however, the book was received very badly. Almost (though not quite) all the reviews were hostile and some were hysterical. Our arguments and our conclusion were both widely and wildly misrepresented. Many suspected that we are covert Theists, committed to undermining the foundations of the Scientific World View (of which they took themselves to be the anointed custodians). Others reproached us for having opinions on issues that are proprietary to members of the Guild of Professional Biologists. The blogs, in particular, were ablaze with anonymous contumely. Well, what did we expect? Hadn't we heard there's a Culture War on?

Some of the objections we've seen strike us as too silly to bother refuting. Others deserve serious replies. The latter should be addressed at length; They will be in future publications. But there is a number of criticisms that can be replied to succinctly; hence the present Update. We propose to quote, and rebut, a scattering of short passages from reviews of our book. Hope springs eternal, so we're told. We hope, at a minimum, to clear the ground for more extended discussions. We still believe in the possibility of a rational, informed, interdisciplinary, consideration of what's wrong with the conceptual architecture of TNS.

2. When some biologists (indirectly) agree with us

Several reviewers have suggested that we don't know enough about biology to criticize a theory that so many biologists hold dear. The implication is: only someone improperly educated could say the sort of things we do. But we don't think our critics are well-advised to insist on our lack of credentials. For one thing, several of them aren't biologists either. For another, it's a self-defeating line of argument; do they hold that only theologians are licensed to discuss the existence of God?

Everybody makes mistakes; even biologists; even biologists who agree with one another; even great biologists like Darwin. If you think somebody has made a mistake, then it's a good thing for you to say so, so that s/he (or you) can be corrected. Surely that is common ground among scientists, philosophers, and everybody else who cares about distinguishing the true from the false. The parochial is the enemy of the true, and should not be encouraged. But we won't go on about this; it's a little embarrassing even to have to mention it. Instead, we report verbatim some recent passages by fully qualified evolutionary biologists, each of whom has earned a Ph.D from an accredited institution of higher learning, and all of whom are explicit in maintaining that neo-Darwinism (the new synthesis) is gone.

"In the post-genomic era, all major tenets of the modern synthesis have been, if not outright overturned, replaced by a new and incomparably more complex vision of the key aspects of evolution. ***So, not to mince words, the modern synthesis is gone.*** *What comes next? […] a postmodern state […]. Above all, such a state is characterized by the pluralism of processes and patterns in evolution that defy straightforward generalization".* (our emphasis)
Eugene V. Koonin (Senior Investigator, National Institutes of Health) (2009 a).

"Evolutionary-genomic studies show that natural selection is only one of the forces that shape genome evolution and ***is not quantitatively dominant, whereas non-adaptive processes are much more prominent than previously suspected****".* (our emphasis)
Koonin, E. V. (2009 b).

"*Although 2009 will be marked by a plethora of celebrations on the subject of evolution, most of the attention is being bestowed on the personalities and historical circumstances surrounding the theory of natural selection, as if this and its synthesis with genetics in the first decades of the 20th century marks the culmination of the theory of evolution. It does not.*" ……
"*Dogmatic thinking has prevailed all too often in our account, with disastrous consequences for the progress of the fields of microbiology, molecular biology, and the study of the evolutionary process. It led to the stagnant and scientifically invalid notion of the prokaryote; it led to the redefinition of the problem of the gene; and through* ***a slavish adherence to the modern evolutionary synthesis****, it led to a premature declaration of victory in the struggle to understand the evolutionary process. …… The study of evolution is poised to cast off a century of dogma and to become a true science, fully integrated with discoveries that owe their roots to microbiology and molecular biology. It is time for biology to put its past behind and begin rethinking the discipline's future.* ***It can no longer afford to keep the study of evolution within the narrow confines of the so-called modern evolutionary synthesis****."*
(o.e.)
Carl R. Woese (Microbiologist, University of Illinois, winner of the 2000 National Medal of Science) and Nigel Goldenfeld (Professor of Physics at the University of Illinois at Urbana-Champaign and Head of the Biocomplexity Group at the University's Institute for Genomic Biology) (2009).

"Despite elaborate Neo-Darwinist mathematical models that focus on inherited variation in animals, evidence continues to mount that the branches of "the tree of life" do not just bifurcate. They do not simply diverge by gradual accumulation of random mutations. Rather lineages converge, as the result of gene transfers, mergers, fusions, partnerships, anastomoses and other forms of alliance. The most accurate modern taxonomies recognize that

Archaebacteria and Eubacteria have become subkingdoms of the prokaryotes whereas all nucleated organisms (eukaryotes) evolved symbiogenetically."
Lynn Margulis (Distinguished University Professor of Geosciences at the University of Massachusetts, winner of the 1999 Presidential Medal of Science) and Michael J. Chapman (Marine Biological Laboratory, Woods Hole, MA) (2010),

*"**There is a growing appreciation among evolutionary biologists** that the rate and tempo of molecular evolution might often be altered at or near the time of speciation, i.e. that **speciation is in some way a special time for genes.** Molecular phylogenies frequently reveal increased rates of genetic evolution associated with speciation and other lines of investigation suggest that various types of abrupt genomic disruption can play an important role in promoting speciation via reproductive isolation. **These phenomena are in conflict with the gradual view of molecular evolution that is implicit in much of our thinking about speciation and in the tools of modern biology.** This raises the prospect of studying the molecular evolutionary consequences of speciation per se and studying the footprint of speciation as an active force in promoting genetic divergence. …. **Speciation might often owe more to ephemeral and essentially arbitrary events that cause reproductive isolation than to the gradual and regular tug of natural selection that draws a species into a new niche.**"* (o.e.)
Chris Venditti (Evolutionary Biologist, The University of Reading UK) and Mark Pagel (Microbiologist, The University of Reading UK) (2009)

In summary: We have seen how several of the recent discoveries in biology that our book recounts lead some biologists to explicit non-Darwinian conclusions. Samir Okasha (2010) pushes them aside saying (correctly) that *"they simply concern aspects of biology about which traditional neo-Darwinism didn't have much to say"*. But our point about these biological mechanisms is not that the neo-Darwinists don't attend to them; but rather the marginalization of TNS that they suggest. It seems that most of the action may well be in a different part of town.

3. Replies to critiques from biologists

What follows are brief replies to criticisms that some of our biologist reviewers have made and that we think are radically wrong-headed; they don't exhaust the list, but they are typical.

3.1 Nothing new

A frequent critique we have received is that all the non-selectionist factors and processes summarized in Part One of our book have been known to evolutionary biologists for a long time and are all perfectly compatible with the Theory of Natural Selection (TNS). This is wrong on two counts: First, because we have based that part of our book mostly on articles published in the last 5 years in specialized biology journals, and (rightly) presented as innovative by their authors; Second, because it is very hard to reconcile these discoveries with TNS, as several authors say explicitly (see the quotes above and more in our book) and almost all of them at least implicitly.

In particular, our critics say that the existence of internal constraints on possible phenotypic variation is obvious and has been acknowledged to be so for decades, indeed by Darwin himself. We have doubts about this. Although we are no experts of Darwin's publications, those who are say what follows: (see also note 2 to pp. 20-24)

"*There can be no direction imposed on evolution by factors internal to the organisms, because the variation upon which selection acts is random in the sense that it is composed of many different and apparently purposeless modifications of structure. The environment determines which shall live and reproduce, and which shall die, thus defining the direction in which the population evolves."* (Bowler, 2003, pp. 10–11).

One more qualified quote, by the bio-physicist and bio-mathematician Stuart Kauffman, a pioneer in the search of physical and self-organizational components of biological structures and evolution, a scientist highly regarded by Richard Lewontin and the late Stephen Jay Gould (see Chapter 5):

"*A curious, logically unnecessary, but influential feature of Darwin's thinking was that the variation within one species which paved the way for emergence of well-marked varieties constituting two species* ***was an indefinite range****. The idea that variations could occur* ***in virtually any direction****, an idea which dominates in Darwin's work despite attention to correlations among traits under selection, has had important conceptual consequence. It follows that* ***selection alone can discriminate which new variants will be found in later generations****. Here is one root of our current idea that selection is the sole source of order in the biological world*". (Kauffman 1993. Page 6) (emphasis ours)

3.2 Two wrong analogies

We like good analogies, but there are limits. The ones we're about to quote seem to us beyond the pale; the kind of far-fetched arguments that responsible scientists should avoid.

"Thus, the authors argue, there cannot be a universal theory of natural selection, for no general relationship of phenotype to fitness can be specified. But the same might be said of many other research programs. For example, the effect of an enzyme is highly context-dependent, so Fodor and Piattelli-Palmarini presumably would not expect any successful theory in biochemistry". Douglas Futuyma (2010, page 692))

The net effect of an enzyme is to catalyze (that is drastically accelerate) a chemical reaction. This action depends on factors such as temperature, acidity, concentration of the substrate and of other chemical participants (co-enzymes, inhibitors). The influence of each of these factors is well understood and separable in principle. Indeed there are general laws of enzymology, such as the Michaelis-Menten equation of enzyme kinetics. These processes take place at one well specified level, that of molecular reactions, where the panorama is totally different from the highly composite one of the genotype to phenotype relation, where we have multiple levels (from Angstroms to yards), and multiple kinds of dynamics. In our book we summarize more than a dozen of these processes; the likelihood of unifying all of them under one theory is negligible. The analogy with enzymology is, therefore, totally fallacious.

The next one is due to Jerry Coyne:

"Clearly, F&P are confusing our ability to understand how a process operates with whether it operates. It's like saying that because we don't understand how gravity works, things don't fall." ... "Our inability to understand all the details [of natural selection] is hardly a reason to claim

that natural selection doesn't work."
(Coyne 2010)

We are not only scientific realists, but scientific hyper-realists. Nothing like the above ever crossed our minds. We will go back to the analogy with the law of gravity in a moment, in our reply to Elliott Sober. Let's concentrate here on just one point. It's one thing to lament our failure to understand some or other natural process which we nevertheless have good reasons to believe occurs. It's quite another to offer principled reasons why some or other theory of such a process isn't viable. Our book is concerned with the latter in the case of the theory of natural selection. Coyne needs to rebut these arguments. He doesn't.

We never said that NS does not operate in the wild because it's so hard for us to understand how it works. We say that general explanations based on natural selection are necessarily based on correlations (between the presence of a trait and greater reproductive potential), not causes. Detailed, very heterogeneous explanations of the selection for individual traits, in individual species, in their particular environments, can sometimes reveal causal factors. There is a radical difference, on which we insist in our book and in this update. The analogy with gravity is untenable. Gravity is the cause of the falling of bodies, not a correlation.

3.3 Merging evolution and Natural Selection

In his review, and in his recent book, Coyne regularly fails to distinguish arguments about evolution and arguments about natural selection. For example, Coyne and Dawkins both discuss at length the circuitous and devious geometry of the laryngeal nerve in mammals, which connects organs only a few inches apart, but runs from the head to the heart, looping around the aorta and then doubling back up to the neck (Coyne points out that, in the giraffe, this detour involves about fifteen feet of superfluous nerve). Then follows an account of how this oddity occurred via progressive transformations from older species of the anatomy of the organs, something we have no reason to question. Dawkins and Coyne take such cases to argue against evolution by "intelligent design", and so they do. They are, however, thoroughly irrelevant to the issues that our book is concerned with, which is whether the mechanism of evolution is Natural Selection. But then, these data and arguments in favor of the evolutionary descent of species are transmuted into data and arguments in favor of the theory of natural selection. Questioning TNS is considered identical with questioning evolution as such. This conflation leads Coyne to say:

"Their [our:JF&MPP] claim to have nullified 150 years of science, and one of humanity's proudest intellectual achievements, with some verbal legerdemain, is not only breathtakingly arrogant but willfully ignorant of modern biology".

Enraged at having failed to hit the target he intended, Coyne proceeds to loose his shafts at a venture.

We repeat: We have no doubts about the reality of evolution, or, more specifically, about the descent and radiation of species from preexisting ancestors; and we entirely accept that topological and functional transformations of internal organs

offer persuasive evidence in its favor. What we seriously doubt is the power of natural selection to explain how it happens.

3.4 The argument from the success of artificial selection

Here's another argument of Coyne's:

"If there really were so many constraints on selection, and if development really were so complex and tightly interconnected that organisms could not respond to natural selection, then why would artificial selection be so effective at changing animals and plants?"

First of all, we do not say that "organisms could not respond to natural selection". What we say is that there are innumerably many different ways of responding, depending on the phenotype, the species and the environment, defying a unitary theory. Moreover, to the best of our knowledge, artificial selection has never managed to produce new species, something that natural selection is supposed to have done many times. So, even artificial selection is effective only up to a point. Numerous sub-species have been obtained, by means of repeated selective cross-breeding, aiming at specific phenotypes (better wool, more milk, stronger muscles etc.). In our book (page 62 and note 2 page 210) we stress that these desired traits were invariably accompanied by a number of others (curly tails, floppy ears, piebald color etc.). These other traits are free riders that were obviously not selected for. The lesson here is that, in cases of artificial selection, it's straightforward to decide which trait was selected for and which one came fortuitously, because we can ask the human agents involved, or make an educated guess. The burden of our book is that, on one hand, the distinction between traits that are selected for is essential to distinguishing causes of fitness from free riders; and, on the other hand, this distinction can't be drawn in cases where there *isn't* a breeder (including, in particular, cases of selection in the wild).

3.5 Missing heritability

Coyne makes the following accusations:

"*Beyond distorting the scientific literature, F&P make a number of claims that are simply silly. I mention just one: "The textbook cases of Mendelian inheritance, in spite of their great historical and didactic importance, are more the exception than the rule." This came as a surprise to me. In fact, cases of Mendelian inheritance (the random assortment of parental genes into sperm and eggs) are the rule; if they weren't, genetic counseling would be useless. Statements like this typify the authors' attitude toward science throughout their book: they seize on some new wrinkle in the scientific literature, like a rare gene that doesn't behave according to Mendel's rules, and interpret it as a revolution that nullifies all of mainstream biology. This lack of grounding is often seen in work by science journalists who make their living touting "revolutionary" new findings, but it is inexcusable in a supposedly serious book written by academics.".*

We are not surprised that this came as a surprise to Coyne. Indeed genetic counsel is often (not always, but often) useless, for instance, when well characterized frequent mutations in over 20 genes explain just 3% or 5% of genetic risk. The case of the "missing heritability of complex diseases" is not a "wrinkle", as Coyne would have us believe. Witness the manifesto by this title published in Nature (October 8 2009, Vol

461, pp. 747-753) by 27 leading human geneticists lamenting the situation, and the following summary by one of the authors, David Goldstein (Richard and Pat Johnson Distinguished University Professor, Director, Center for Human Genome Variation, Duke University) in the New England Journal of Medicine on April 23 2009:

"*20 gene variants account for 3 percent in the variation of risk susceptibility to type 2 diabetes….If common variants are responsible for most genetic components of type 2 diabetes, height, and similar traits, then genetics will provide relatively little guidance about the biology of these conditions, because most genes are "height genes" or "type 2 diabetes genes…News are as bleak as they could be."*

These are not the irresponsible scientific journalists to whom Coyne compares us. A quote will say it all. Another of those authors, Leonard Kruglyak (Professor of Ecology and Evolutionary Biology at Princeton University) in Nature: Vol 456, 6 November 2008, p. 21 says:

"It's a possibility that there's something we just don't fundamentally understand, that it's so different from what we're thinking about that we're not thinking about it yet".

Kruglyak refers to the genotype-phenotype relation for complex diseases, but the same can be said, we think, for complex traits more generally. We suggest that Coyne absorbs these facts, stops pontificating and pays attention, not to us, but to these colleagues of his.

Coyne concludes: *"In the end, F&P's contrarian efforts are all belied by the world of Richard Dawkins--the flourishing field of modern evolutionary biology, where natural selection remains the only explanation for the wondrous adaptive complexity of organisms."*
Please underline: "*natural selection remains the only explanation"* for later reference.

3.6 Catching phenotypes

We conclude our replies concerning biology with a critique voiced both by Douglas Futuyma and Jerry Coyne:

"The ludicrous analogy with which Fodor and Piattelli-Palmarini end: "organisms 'catch' their phenotypes from their ecologies in something like the way that they catch their colds from their ecologies." (Futuyma)

"After much demurring, they [i.e us JF&MPP] float the idea that "organisms 'catch' their phenotypes from their ecologies in something like the way that they catch their colds from their ecologies." Although this "explanation" links evolution to ecology, it's completely meaningless. How did ancestral whales catch their flukes and flippers from the water? How did ancestral birds catch their wings from the air? F&P don't say". (Coyne)

Actually, we don't think that whales catch their flukes from the water. This discussion is, of course, awash in metaphors on both sides, and the thing about metaphors is that if you don't treat them with a dollop of subtlety, they are likely to bite you. Darwin's metaphor is: "Natural selection is like breeding". We think it invites failures to notice the difference between breeding-for (which is intensional) and selection (which is not). Our metaphor is: "the processes that mediate coming down with a phenotypic trait are like the ones that

mediate coming down with a cold"; the point is that both depend on massive dynamic interactions between a host's endogenous properties and properties in its environment; and quite likely the details of such interactions are highly idiosyncratic from case to case. That's why nobody in his right mind thinks there could be a general theory of catching diseases. Why, then do biologists think there could be a general theory of the evolution of phenotypes?

4 Replies to critiques of the conceptual situation (Part 2 of the book)

4.1 Explanations and definitions

The crucial sentence in Peter Godfrey-Smith's review of WDGW (London Review of Books) is:

"if one [but not the other of two linked traits] is causing increased reproductive success, it is [sic] being selected for, in the sense that matters to evolutionary theory."

A number of other reviewers have made much the same suggestion, but it won't do. The theory of natural selection claims that a trait's having been selected for causing reproductive success *explains* why a creature has it. But then it can't also claim that "in the sense that matters" "a trait was selected for" *means* that it is a cause of reproductive success. For, if it did mean that, then the theory of natural selection would reduce to *a trait's being a cause of reproductive success explains its being a cause of reproductive success* which explains nothing (and isn't true).

This is all old news; because John's being a bachelor *is* his being an unmarried man, John's being a bachelor doesn't *explain* his being an unmarried man. Psychologists who hoped to defend the "law of effect" by saying that it is *true by definition*, that reinforcement alters response strength, made much the same mistake that Godfrey-Smith does.

Likewise, Elliott Sober says,

"the distinction between selection-for and `free riding' is nothing other than the distinction between cause and correlations."

Later on he says that

"there is selection for trait T in a population if and only if trait T causes organisms to have reproductive success in the population".

This, he claims, is a *definition* of "selection-for": it's true by definition that the trait that is a cause of increased fitness is selected-for but the other is not. However, as we just saw, that can't be right. The very heart of TNS is the thesis that, in the paradigm cases, traits are selected-for *because* they are causes of fitness; that is, differences of their effects on fitness *explain* why some traits are selected-for and others aren't. But if that's so, then the connection between being selected-for and being a cause of fitness can't be *definitional.* The dialectics here precisely parallels arguments that philosophers of mind offered in '50s against the claim that, in paradigm cases, the relation between behavior

and mental states is "criterial" (in effect, definitional). If it's *conceptually necessary* that you raise your arm when you want to, then the cause of your raising your arm can't be your wanting to raise it. It took fifty years for philosophy to get over this. Must we now have it yet again? Something really ***is*** seriously wrong with the theory of natural selection, and stipulating that it is true by definition won't fix it.

4.2 The intensionality of selection-for

Elliott Sober has what seems to us to be a distorted view of the present polemical situation.

"FP really do maintain that there cannot be natural selection for one but not the other of two traits that are locally coextensive. However, in Fodor and Sober (2010) Fodor denies that the book says this."

What Sober says that the book says is that there can't be a *causal theory* of "selection-for." But the book doesn't say what Sober says it does. What it does say is that the Theory of Natural Selection can't provide an account of natural selection (because it's a causal theory and selecting-for is an intensional relation). So the book proposes a dilemma: either there is no such thing as natural selection, or, if there is, the Theory of Natural Selection misdescribes it.

4.3 Can linked properties be distinct in causal role?

Here's what Ned Block and Philip Kitcher (hereinafter BK) think is one of our two main errors.

"Their [e.g. our, Fodor and Piattelli-Palmarini's] specific charge is that, with respect to correlated traits in organisms - traits that come packaged together - there is no fact of the matter about which of the correlated traits causes increased reproductive success".

BK then speculate that we endorse the "very ambitious" claim that when traits are correlated, there can be no fact of the matter about which trait causes what. But, of course, we don't believe, still less make, either of these claims. In fact, we think that it's preposterous on the face of it. Indeed, if the causal powers of linked traits can't be distinguished, it would not be an argument against the Theory of Natural Selection that it fails to distinguish them. We therefore spent a whole chapter (Ch. 7) discussing a number of ways in which the causal roles of confounded variables can be, and routinely are, assessed. The most obvious of these is J. S. Mill's "method of differences": run an experiment in which one but not the other of the putative causes is suppressed. If you still get the effect, then it must be the variable you *didn't* suppress that's doing the causing. People (scientists very definitely included) do this sort of thing all the time, and with great success. All this is familiar from Phil. 101. Do Block and Kitcher really believe that, old and battle-weary as we are, could have written a book that gets that wrong?

The question whether there is a fact of the matter about which variable is the cause, or about whether this fact of the matter is epistemically accessible, really must not be confused with whether Natural Selection, as Darwin understands it, is able to distinguish causes from their local confounds. For reasons the book details, we think it

can't. To repeat: One can work out what caused what in all sorts of ways: use Mill's method; or take the system of causes and effects apart and find out what mechanisms operate inside it; or ask the guy who built it (if somebody did) how it works… and on and on and on. But Natural Selection can't do any of these things. It can't look inside, and it can't run experiments, and it can't contrive theories, and it can't consult the intentions of the builder. All natural selection can do is recognize *correlations* between phenotypic traits and fitness. And that doesn't help because, by assumption, if either of the confounded traits is correlated with fitness, so too is the other, and to the same extent.

Samir Okasha, in his review, commits much the same misreading of our book: He accuses us of denying the distinction between causes of fitness and free-riders. But our view is *neither* that it is impossible to deconfound causes of fitness from free-riders nor that there is no such distinction. What we do think (and what we do think our book shows) is that Darwin's theory can't, even in principle, specify a mechanism by which selection could reliably distinguish causes of fitness from correlates of causes of fitness. To a first approximation, this is because TNS recognizes only exogenous variables as selectors, and the only (relevant) fact to which such variables are sensitive, according to TNS, is the strength of the correlations between phenotypic changes and changes of fitness. And, of course, correlation doesn't imply causation. Indeed it *patently* doesn't imply causation when the correlation in question is identical for both of the candidate causes; as it is by assumption, in the case where phenotypic traits are linked.

To repeat: It is beside the point that scientists in the laboratory often can deconfound linked causes; scientists have minds and the process of evolution does not. Indeed, it is the prima facie connection between intensional states and mental states that makes the intensionality of "select for" a problem for naturalizing TNS; a point in respect of which WDGW is vehement.

For a while it bothered us that many of our critics should have so blatantly misread what we wrote. But we have a theory: It's that the neo-Darwinian community is so blindly committed to TNS that they allow themselves to reason as follows (implicitly, to be sure): (1) This book says that TNS can't distinguish causes of fitness from correlates of causes of fitness. But, it goes without saying that: (2) TNS is certainly true and everybody knows that it is. So: (3) if the authors claim that TNS can't distinguish causes from correlates, that must be because they think that there is no such distinction. So (4) I shall write a review accusing them of thinking that. But if that is indeed how our critics are reasoning, we protest that it's more than a tad question-begging.

4.4 Laws of evolution

A short summary of the second half of the book might go like this: TNS needs selection-for to be intensional, but offers no suggestion of how it could be. But, as we remarked above, if there are laws of evolution (nomologically necessary empirical generalizations to which evolutionary processes conform) it might be from those that the intensionality of select-for derives. So it matters to the present question whether there are such laws. The bad news, according to WDGW, is that there aren't. This is. Indeed, one of the cases in which WDGW agrees with what we take to be the consensus view among biologists. Nobody doubts, of course, that evolution is law-governed; after all, the laws of physics apply to everything. The present issue is whether there are *biological* laws of evolution; that is, laws of evolution that are defined over *biological* kinds (such as, for example,

laws about evolution defined over ecological properties *so described* and their effects on fitness *so described.*) Missing this point has lead to all sorts of confusion including, notably, the suggestion that if there are no laws of evolution, determinism and/or mechanism are ipso facto undermined.

Well, Elliott Sober thinks we're wrong about that. Actually what he says is not that are such laws, but that we haven't shown that there aren't. And indeed we haven't. Since the issue is entirely empirical, there's no question of demonstrative arguments on either side. There are, however, straws in the wind, and we think they're blowing our way.

Here are two reasons for doubting that there are laws of evolution. The first is that there seem to be no examples of such laws. That is easily explained on the assumption that, in fact, there are no such laws. The second is that, if there were laws of evolution, they would have to be horrendously complicated. A long tradition of modeling evolution has indentified at least the following factors, among others: effective population size, density-dependent selection, drift with or without selection, migration, gene flow and horizontal transmission, the diffusion of neutral mutations, mutational bias, biased gene conversion, differentials in fertility, sexual selection, variable sex ratios, the overlap of fertile generations, the fixation of deleterious alleles, phenotypic plasticity, and various kinds of epistasis (gene-gene interactions). Sober says (rightly) that complexity isn't, in and of itself, an argument against the putative laws. But the kind of complexity that laws of evolution would require is, we think, without precedent in the other sciences. First of all, laws of evolution would have to take into consideration interactions at vastly heterogeneous levels: molecule to molecule, gene to gene, gene to cell, cell to cell, developmental module to developmental module, tissue to tissue, organism to organisms of the same species, organism to organisms of different species, and all these to the local ecology. The heterogeneity concerns both sheer size (from Angstroms to miles) and the conceptualization of the relevant kinds. His failure to understand this is part and parcel of Sober's mishandling of one of his own examples:

"The gravitational force now acting on the earth depends on the mass of the sun, the moon, and of everything else. It does not follow that there are no laws of gravity, only that the laws need to have numerous placeholders…. The fact that an effect has numerous complexly interacting causes does not show that there are no laws about this complex cause/effect relation".

Well, of course there are laws of gravity; principally that the gravitational force between objects varies directly with their total mass and inversely with the square of their distance. Notice, however, that this law is quite simple; in particular, it has no 'place holders' for the sun, the moon, the Earth or anything else except the masses and distances of the objects involved. That's why the law of gravity would be unaffected even if there weren't the sun, the moon, or the earth.

What goes on when explanations appeal to laws is something like this: there are variables for relevant properties of things that fall under the laws; and there are specifications of the "initial conditions" in some domain to which the laws apply. Neither the moon nor its mass gets mentioned by the laws of gravity; but both *do* get mentioned in specifying the conditions that obtain when the theory of gravity is used to predict the gravitational force between (eg.) the moon and the earth. In consequence, the laws of gravity have very many fewer "place holders" than there are things in the

universe to which they apply. We won't argue for this view; but please take our word for it that a lot depends on getting it straight.

So now the question arises whether this picture is plausible for the (putative) evolutionary laws of trait fixation. We think it pretty clearly isn't; *not,* however, because there are very many creatures to which the laws would have to apply, and very many environmental features with which such creatures may interact. Rather, it's because of the awesome *heterogeneity* of levels and kinds we have mentioned, and of the ways in which interactions of creatures with their environment depend on what kind of creature it is and what kind of environment it is interacting with. As we saw two paragraphs back, laws don't need place-holders for each thing that falls under them, but they do need placeholders for each *kind* of thing that falls under them.

To make the point slightly differently, there are typically many kinds of creatures that can share an environment, and many kinds of environments that creatures can share. (We're told that more than ten thousand species share Central Park). That being so, the putative laws that determine fitness as a function of such interactions would have to be complicated in precisely the way that the laws of gravity are not: They would need "place holders" for each of the *kind* of creatures that they apply to and for each *kind* of environment that the creatures can interact with. And, to repeat, though the number of things a law applies to doesn't determine how many placeholders it needs, how many *kinds* of things it applies to does. Given all that, could there be such laws about how creature/environment interactions determine fitness? In principle, sure there could. But are there such laws? We think the probability is asymptotically close to nil. The kind of complexity that *does* tell against a putative law is the kind that proliferates kinds beyond necessity.

There are other things Sober's review says that we think are wrong; for example, we think it's wrong about whether truths about individual events support counterfactuals (except for the dreary counterfactual that if exactly the same thing were to happen again, all else being equal, exactly the same effects would ensue.) But, for present purposes, we're content to leave it here.

4.4 TNS versus sufficient reason

David Papineau, in his review says:

"If Fodor and Piattelli-Palmarini are right, polar bears don't have white fur because it confers advantages in the Arctic; we don't have eyes because they help us to see; and in general there is no tendency for natural selection to preserve adaptive traits".

Could we really be denying that the reason polar bears are white is that being white hides them in the snow? No. Part of the story about why polar bears are white is surely that there were many causal chains in which white polar bears got missed by their predators (and/or were able to sneak up on their prey) more regularly than polar bears that were less white. On our view, tracing such causal chains is what natural history does for a living. But a theory of Fs doesn't consist of an enumeration of causal chains in which Fs are involved. *A theory of Fs is an account of what Fs have in common as such.* Accordingly, a theory of trait evolution is an account of what instances of trait evolution have in common as such. (Notice, in passing, that "as such" is intensional). So what does TNS say about what instances of trait evolution have in common as such? What, for example,

does it say about what the evolution of four chambered hearts in mammals, and of long necks in giraffes, and of web spinning in spiders and of bipedal gait in us have in common *qua* instances of trait evolution? Just this*: In every such case there has to be something about the creatures (or about their ecology, or both) such that those of the creatures that were F flourished more than otherwise similar creatures that were not F.* Well of course there has to be. That follows just from the "principle of sufficient reason" according to which if something is F, there must be something that caused it to be F; and, of course, whatever the "something" is, it has to be either internal to the organism or external to the organism. There's no place else that it could be. On our view *there is no theory of evolution.* All there is, is natural history.

Speaking of the adaptive function of the eye (as Papineau urges us to do) a species of jellyfish (the cubozoan jellyfish, *Tridpedalia cystophora* discovered in the waters near Puerto Rico) has 24 globular eyes in 6 groups of 4 (called rhopalia), very similar to our vertebrate eyes, but no brain to collect the images, no optic nerve, and the lenses can only form images behind the retina. No adaptive explanation is in sight, though the genetic and developmental mechanisms responsible for this feast of structure without function are well understood.

4.5 <u>On mathematical models</u>

Samir Okasha and other reviewers hope to vindicate TNS by appealing to the "paradigm" (sic) explanatory power of mathematical models of natural selection. We are fully aware of the long and illustrious tradition of mathematical theory of natural selection and, more generally, of evolution; from the Hardy-Weinberg law of equilibrium between allele frequencies (1908)) to the works of Ronald Fisher, J. B. S Haldane and Sewall Wright (1924-1937*)* to George R. Price's theorem (1970, 1972) all the way to the present day (for thorough expositions see Provine 1971/2001 and Rice 2004). However, as a leading historian of mathematical evolutionary theories says:

"They [Fisher, Haldane, Wright, Hogben, Chetverikov and other mathematical modelists] all disagreed, often intensely, with each other about actual processes of evolution in nature, <u>even when their models were mathematically equivalent.</u>" (William B. Provine 1988, p. 56) (our emphasis)

Several critiques of the plausibility of many such models have been raised by qualified biologists including, just to name a few, Carl Woese, Andre'Ariew and Richard Lewontin, Richard Michod and even Massimo Pigliucci, who is by no means in sympathy with our view of TNS. In particular, Carl Woese, in a recent interview with Marc Buchanan for the "New Scientist", says:

"Biology built up a facade of mathematics around the juxtaposition of Mendelian genetics with Darwinism, and as a result it neglected to study the most important problem in science - the nature of the evolutionary process."
(Buchanan 2010)
And it is again beside the point that scientists are quite often successful in constructing models of such phenomena as the evolution of sex ratios in a population; or of how actual foraging strategies approximate ideal foraging strategies; etc. The point is that such

models aren't causal explanations; they don't do - they don't even purport to do - what so many proponents of TNS claim that it does: explicate the causal mechanism of evolution. The most strenuous defenders of the modern synthesis state explicitly that, although causal inference is desirable, mathematically, all that is required is correlation. In general, mathematical models can only be as good as the idealizations on which they are based. In the words of a leading expert and author of a comprehensive technical treatise:

"It is in the nature of model building that our models often hinge on assumptions that we know are not exactly true. What is interesting about [two such] assumptions – monomorphic populations in which variant strategies appear one at a time and populations that respond quickly to environmental changes – is that they are contradictory. A population cannot quickly evolve to a new equilibrium unless it has a substantial amount of heritable variation. If evolution always had to wait for a new variant to arise by mutation, it would be a very slow process, especially if each new mutation differed from the previous state by only a small amount. Thus, when one of these assumptions is a good approximation, the other one ceases to be."
(Sean H. Rice, 2004, Page 289)

Mathematical model building can make explicit the consequences of certain idealizations, but it doesn't even purport to *reveal* the causal mechanisms that sustain the phenomena; whereas our worry about TNS is that no causal mechanism could do what it claims that the process of selection-for does.

Conclusion

We continue to believe that there's a lot that Darwin Got Wrong. We continue to believe that the issues implied by the externalism of his account of selection, and by his failure to notice the intensionality of selection-for, are in need of thorough and careful consideration. Thus far, the critical responses to our attempts have not been edifying; mostly a howl of reflexive Darwinism, with very little attention paid either to the structure of the arguments or to their repercussions. But we're told that hope springs eternal. Our hope, at a minimum, is to have cleared the ground for calmer and much more responsible polemics. We still believe in the possibility of a rational, interdisciplinary, discussion of the empirical warrant and the conceptual architecture of TNS. But we must admit that we don't believe in it now as much as we did a year ago.

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