Adaptationist Reasoning and Applied Population Genetics
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0. Introduction

In what follows, I consider some applications of population genetics to natural populations and their relationship to adaptationist reasoning. My main goal is to bring out how the inferential structure of both kinds of reasoning contains many of the same elements. There is one crucial difference between the two, however: researchers applying population genetics provide *evidence* that the causal scenarios that ultimately explain the dynamics of their target populations are real. It is toward the goal of establishing the truth of a causal scenario that nearly all the quantitative techniques characteristic of applied population genetics, but absent from adaptationist reasoning, are directed.

The most interesting connection between adaptationist reasoning and applied population genetics is the role played by considerations of the plausibility of the causal scenarios that function as the ultimate explainers in both kinds of reasoning. Plausibility considerations serve a critical function: they rule out alternative explanations that are compatible with the known facts about the evolution of a population. Plausibility considerations play this role in *both* applied population genetics and adaptationist reasoning. While researchers applying population genetics make a considerable effort to provide evidence that the causal scenarios that explain the dynamics of their populations are correct, they explicitly invoke plausibility considerations too. There is nothing wrong with allowing considerations of plausibility to contribute to the justification of an evolutionary explanation; rather one must simply refuse to rely upon *nothing but* plausibility. Because researchers applying population genetics work so hard to provide evidence that the causal scenarios they attribute to their populations are correct, and because adaptationist reasoning and applied population genetics are otherwise very similar, what is wrong with adaptationist reasoning is its reliance upon nothing more than plausibility to justify the imputation of a causal scenario to a population.

1. Adaptationist Reasoning

By "adaptationist reasoning" I mean to refer to the foremost pattern of argument associated with the adaptationist program, as criticized by Gould and Lewontin in their famous paper, "The Spandrels of San Marco and the Panglossian Paradigm: a critique of the adaptationist program" (1979). That pattern of reasoning is the generation of plausible evolutionary scenarios that are meant to function as explanations of designed-appearing organic structures. In their critique, Gould and Lewontin make compelling rhetorical use of a collection of bouts of adaptationist reasoning, ones that involved plausible-sounding explanations that were abandoned in favor of different explanations:

> Zig-zag commissures of clams and brachiopods, once widely regarded as devices for strengthening the shell, become sieves for restricting particles above a given size. A suite of external structures (horns, antlers, tusks) once viewed as weapons against predators, become symbols of intraspecific competition among males. The Eskimo face, once depicted as 'cold engineered'

> becomes an adaptation to generate and withstand large masticatory forces. (Gould and Lewontin 1979, 586)

The adaptationist accounts that get discarded and replaced by others consist in a series of failures. Adaptationist reasoning is unreliable.

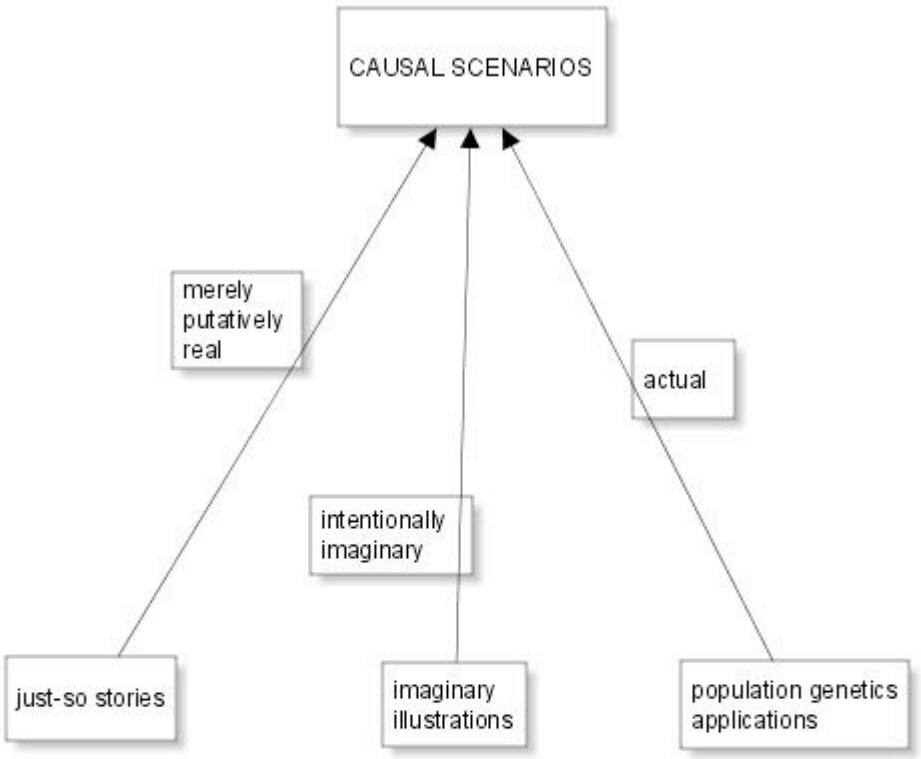
So doing so on purpose, let's mean by "adaptationist reasoning" a bad thing. Adaptationist reasoning is a specific use imaginary scenarios, the use of them to establish conclusions about the causes of evolution in ancestral populations. Adaptationist reasoning does not always yield false conclusions; indeed, the replacements for the discarded adaptationist explanations that Gould and Lewontin list "may all be right" (Gould and Lewontin 1979, 586). Rather, adaptationist reasoning cannot *reliably* be used to establish true conclusions about evolution.

By defining adaptationist reasoning in the way that I have, I allow the use of imaginary scenarios to function in the way that Darwin used them (Lennox 1991, 226-230). Darwin uses his imaginary illustrations to provide accounts of how features of organisms that appear designed *could* have spread throughout a population by natural selection; Darwin's imaginary illustrations show that some structure is *explicable* in terms of natural selection. Adaptationist reasoning, on the other hand, is the use of imaginary scenarios not to show how some population *could* have evolved, but how it *did* evolve.

2. Causal scenarios

It is clear from Darwin's use of imaginary scenarios that the form of explanation being offered here is causal explanation. For instance, Darwin's scenario leading to the evolution of nectar production by flowering plants postulates a series of causally concatenated events that result in the production of especially vigorous seedlings by nectar-releasing plants (Darwin [1859]1988, 92). It is clear from Gould and Lewontin's writings that they are interested in causal explanation too. One aspect of the adaptationist program is the suppression of alternative non-selectionist causal explanations of phenotypic traits. Gould and Lewontin want to undercut the privileging of a specific sort of causal explanation of itemized phenotypic traits, an account that presents them as having spread through a population because of their conduciveness to the reproduction of their bearers. Indeed, "the good fit of organisms to their environment can occur at three hierarchical levels with different causes" (Gould and Lewontin 1979, 592).

Darwin's imaginary illustrations are determinate versions of causal scenarios, specifically ones that are imaginary. The instances of adaptationist reasoning that come under fire from Gould and Lewontin are instances causal scenarios too, but this time putatively real ones. We will see that population geneticists generate causal scenarios too.



3. An inferential model of adaptationist reasoning

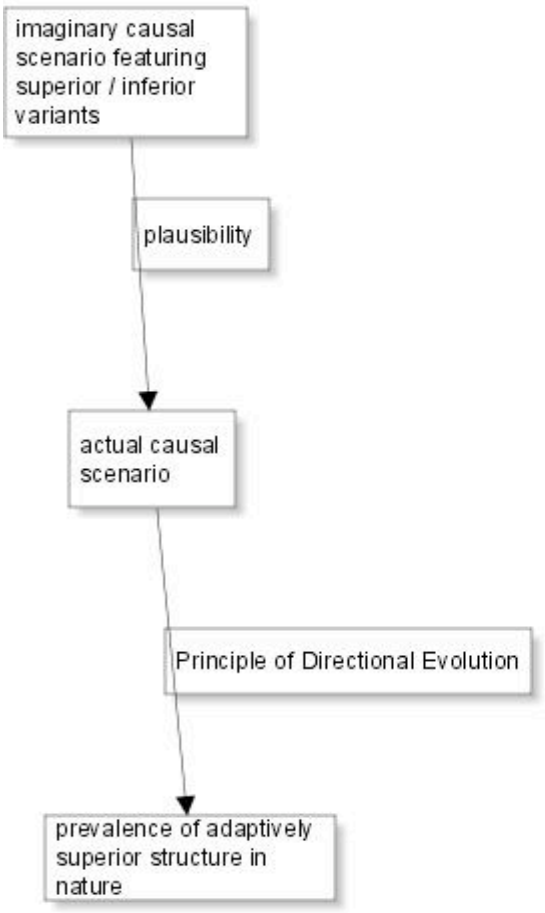
We have seen that the conclusions we get from adaptationist reasoning are conclusions about what the evolution of ancestral populations. Now I want to consider what we need in order to engage in adaptationist reasoning, and what we are supposed to get out of it. Generating explanatory causal scenarios through adaptationist reasoning involves a slim set of requirements:

1) knowledge of a designed-appearing structure,
2) an imagination, and
3) a sense of the reasonableness, the plausibility, of explanations.

Given that everyone encounters designed-appearing structures, and imaginability is hardly a constraint at all, plausibility is the only constraint on adaptationist reasoning with any bite. However, that a casual scenario is plausible is not good enough grounds to believe that it gets at historical evolution as it really occurred. There are simply too many plausible scenarios that could account for some phenotypic trait for plausibility to constrain evolutionists' adaptationist reasoning sufficiently strongly to make that reasoning reliable (Gould and Lewontin 1979, 588). The shortfall of adaptationist reasoning, it's unreliability, is a result of the paucity of the constraints on its deployment.

Yet despite its weakness, plausibility remains a real constraint on adaptationist reasoning. You would not be playing by the rules if you offered an absurd conjectural explanation for some designed-appearing structure, something deliberately far-fetched and weird, say involving aliens, even if the scenario would explain the target phenotype were it to have occurred. The problem with adaptationist reasoning is that plausibility is *too weak* a constraint to ensure reliability, not that plausibility is no constraint whatsoever.

So there are really two inferences involved in adaptationist reasoning. One is from an imaginary scenario, by way of its plausibility, to its reality; the other is an explanatory inference from the scenario to the ubiquity in a population of a designed-appearing structure.



The problem with adaptationist reasoning occurs in the first step only. The exploitation of the second inferential connection, the one lying between a causally described population and an evolutionary result, is not the problem. It is perfectly reasonable to suppose that structures conducive to increased relative reproduction rates will spread throughout a population. I'll call that the *principle of directional evolution*. The principle is no doubt defeasible; there are a number of ways in which variations might fail to spread despite their conduciveness to the reproduction of their bearers. But the problem with adaptationist reasoning is not that it relies on a defeasible principle of inference; the problem is that it does not license you to believe you have a grip on an actual causal scenario in the first place, since, as we saw already, the inference from plausibility to reality is a bad one.

4. Two studies in Applied Population Genetics

I now turn to consider a couple of cases of applied population genetics. These cases are not intended to be especially representative of how population genetics is applied to natural populations undergoing selection. At least, I took no precautions to ensure that they were. My goal is draw a connection between how adaptationist reasoning functions and how the deployment of formal population genetics over natural populations *can* work. That said, I consider three cases of applied population genetics among many more that function in a roughly similar fashion (Gigord, Macnair, and Smithson 2001; Smith 1993; Sandoval 1994; Prout and Savolainen 1996; Benkman 1996; Mitton 1997; Rainey and Travisano 1998; Stahl et al. 1999; Mousseau, Sinervo, and Endler 2000; Suiter, Bänziger, and Dean 2003; Tian et al. 2003; Hedrick 2003; Hoekstra, Drumm, and Nachmann 2004).

The first case of applied population genetics I will consider concerns a population of cichlid fish living in the African Rift Valley (Hori 1993). *Perissodus microlepis* approaches prey from behind to snatch several scales. The mouths of these predators are angled either to the left or to the right, making some left-handed (dextral) and others right-handed (sinistral). Michio Hori proposes that frequency-dependent selection is the mechanism that maintains this polymorphism. His causal scenario explaining the polymorphism is as follows. The cichlids have relatively poor hunting success, succeeding in snatching scales only about one-fifth of the time they attempt to do so, and this is because their prey are aware of them and guard against their assaults. The more frequent predator morph, whether dextral or sinistral, is at a disadvantage because the prey guard their left or right flank more vigilantly when that flank is the subject of a greater number of assaults. Increased guarding by prey leads to fewer successful assaults by the more common predator, which leads to predictable downstream consequences for their relative rates of survival and reproduction. Accordingly, selection for rare types keeps both morphs at roughly intermediate relative frequencies in the population.

The second case of applied population genetics that I will consider is that of Schemske and Bierzychudek's intensive study of the desert perennial *Linanthus parryae* (Schemske and Bierzychudek 2001; Turelli, Schemske, and Bierzychudek 2001). These authors seek to oppose their interpretation of the dynamics of that population to one they attribute to Sewall Wright, who explained the dynamics of the blue and white *Linnathus* morphs as governed primarily by drift in a hierarchically structured population. Schemske and Bierzychudek implicate differential response on the part of the morphs to different levels of precipitation in maintaining the polymorphism in the population. The less widespread blue morph produces more descendants during drier seasons, while the white morph produces more in high rainfall years. Each morph is favored at different times owing to the interaction between the genotypes and a varying environmental influence, leading to a persistent polymorphism in the population.

Applied population genetics involves main inferences as well. SHOW PICTURE. The first connects empirical evidence to a definite causal scenario, such as the ones just rehearsed for the fish and flower populations. The second inference connects that causal scenario to the long-run dynamics of the population. Our researchers expect their populations to remain polymorphic, and the connection between the persistence of the polymorphisms and the causal scenarios the researchers propose is secured by population genetics equations. I discuss the connection between causal scenarios and long-run dynamics provided population genetics equations next, and then consider how population geneticists use empirical evidence to justify the causal scenarios they impute to their populations.

5. Generic Equations

Philosophers of biology are liable to write that relative fitness coefficients in population genetics models must be assigned to population members on the basis of relative reproduction rates, and they do so with an eye to explaining the notion of fitness (Horan 1994; Ariew and Matthen 2002; Stephens 2004). The use of relative reproduction rates to estimate relative fitness variables is not the most common technique that population geneticists have used to fix values for fitness variables (Manly 1985, 46), but even if it were the *only* technique available, its use would not be enough to explain the deployment of relative fitness variables in population genetics. This is because population genetics features a great many different equations featuring different types of entities weighted by different relative fitness coefficients or relative fitness functions.

I will call the various equations used by population geneticists to make inferences about population dynamics *generic equations*. Many different generic equations can lead to a single sort of dynamics; there are a great many equations that can yield stable polymorphisms, for instance. So the use of estimators for relative fitness variables would have to be coupled with some other understanding that would license the deployment of one generic equation for the system rather than another. What the case studies I consider show is that the understanding one needs in order to fix upon an appropriate generic equation for a population is *causal* understanding of that system.

None of the researchers I surveyed applied population genetics by deploying equations with values for their variables that had been fixed on the basis of statistical assessments. Indeed, population geneticists rarely engage in estimations of all the parameters in a model (Turelli, Schemske, and Bierzychudek 2001, 1294). Rather than making exact inferences about the dynamics of their populations using equations with fixed values, the researchers surveyed here instead make use of *analytic results* yielded by mathematically tractable equations.

Analytic inferences provide a means of explaining the long-run dynamics of real populations even when statistical estimations of the values of some or all of the variables in the equations have not been made. So not only are estimations of relative fitness based on relative reproduction rates atypical of applied population genetics, estimations of relative fitness are sometimes entirely superfluous. Generic equations may link causal scenarios to population dynamics without the researchers having to estimate relative fitness values at all. Researchers who appeal to analytic results of population genetics equations explain the dynamics of their target populations as a *sort* of dynamics that is liable to result from a causal scenario, typically a persistent polymorphism of one sort or another. This parallels adaptationist reasoning in which a sort of dynamics, directional evolution eventuating in fixation of a trait, is taken to result from a putative causal scenario.

Of the researchers I discuss, only Schemske and Bierzychudek make any effort to estimate values for the relative fitness variables in the generic equation they deploy, and they only do this because they seek to show that their *Linanthus* population is subject to a selection regime that yields a protected polymorphism under a limited range of conditions. A polymorphism results under the sort of temporally variable selection regime they consider only when the relative fitness parameters of their equation fall within a specific range values, so they need to assess relative fitness parameters to check whether the relative fitness values of their *Linanthus* morphs fall within that range.

5. Evidence for Causal scenarios

Now let us consider the first inferential connection exploited by population genetics researchers, the one connecting empirical evidence concerning a natural population and a definite causal scenario. Almost all the experimental and statistical work done by the researchers considered here is done to justify their attributions of specific causal scenarios to their populations, very little is done to assess values for variables. Because they are imputing causal scenarios to populations, and we lack a general rule for ascertaining when causal relationships hold, the population geneticists considered here are forced, as is everyone else who tries to establish the existence causal relationships, to deploy *evidential reasoning* to justify their attributions of specific causal scenarios. The researchers must trace out the implications of causal relations were they to obtain, and then test to see whether those implications really are borne out in their populations. There are two main tools of evidential reasoning in population genetics; researchers run statistical tests and they do experiments.

5.1. Statistical tests

Hori runs a number of statistical tests to justify the causal scenario he envisions as what explains the dynamics of his fish population. Because the scales from the right and left flanks of the cichlid prey appear different under the microscope, Hori can affirm, on the basis of the examination of stomach contents, that predator morphs only consume scales from one flank of their prey. Hori collects statistics on the characteristics of broods and the adults guarding them and is thereby able to discern that the variation is heritable, and probably the result of a Mendelian single locus two-allele system. He is also is able to track the oscillation in frequency of the two morphs through their lifecycle by sampling the fish population, and further establishes that smaller younger individuals typically show the opposite bias in relative frequency to that seen among adults, providing evidence that the rare types have more reproductive success. In one way or another, these statistical tests serve to justify the causal scenario Hori attributes to his population; none of the statistical tests serve to fix values for variables.

Schemske and Bierzychudek run a gamut of statistical tests too, far more than can be reviewed here. But one can get a sense of the rigor of their research from a few of these. They monitor the dynamics of three sub-populations of *Linanthus* morphs and sample them to collect statistics on relative and absolute seed and flower production, which they use to measure relative fitness. They correlate the morphs flower and seed production to seasonal rainfall over the course of eleven years to show how rainfall impacts the fecundity of both types of morph. They go on to research weather patterns for the area to see whether the precipitation pattern over the course of their research is representative for the region in general. They also assess the longevity of the seed bank in their population and attempt to establish whether either morph persists longer within it.

5.2. Experiments

One rarely hears about experimentation in connection with population genetics, but the researchers I consider do several experiments. Schemske and Bierzychudek test for differential mortality of their *Linanthus* morphs in the lab (Schemske and Bierzychudek 2001, 1273). They also use laboratory populations to secure the link between protracted rainfall and germination.

Hori also conducts an experiment; he uses the prey fish of the cichlids as lures on fishing lines to confirm that the cichlids only ever attack one flank of their prey (Hori 1993, 218). These experiments simply do not make sense except as ones designed to provide evidence that the causal scenario the researchers attribute to their populations is correct.

6. Plausibility and Ruling out Alternative Explanations

Experiments, along with statistical tests, contribute to the establishment of a causal scenario as what explains the dynamics of a target population. But the researchers considered here entertain the possibility that the evidence they find for their causal scenarios has other explanations. In some cases, the possibility of alternative explanations prompts the researchers to do additional tests. For instance, Schemske and Bierzychudek count how many times pollinators visit the different morphs in an effort to locate a bias in the attractiveness of the flowers to pollinators. But plausibility retains a role to play in applications of population genetics of the sort considered here, too. It is used as grounds for the elimination of rival explanations *without* these having been shown to be incompatible with the evidence.

The rejection of alternative explanations on plausibility grounds is probably an inevitable aspect of causal reasoning: we ask juries to convict defendants when it is beyond a reasonable doubt that they committed the crime with which they have been charged; we do not require that there are absolutely no explanations of the evidence that has been marshaled by the prosecution that do not implicate the defendant in the misdeed. Hori makes a few remarks that amount to appeals to plausibility. He assumes that the survival rate of the two phenotypes does not differ for reasons other than the frequency-dependent selection mechanism he picks out. He also asserts that "pleiotropic effects seem most unlikely" (Hori 1993, 218). Equally, he assumes that the size of the cichlids is indicative of their relative age (Hori 1993, 218).

Schemske and Bierzychudek make many appeals to plausibility. They are frank about the limitations of their tests of alternative mechanisms of selection involving differential pollination or differential water-use efficiency (Schemske and Bierzychudek 2001, 1279). They explicitly recognize that difficulties may arise from the fact that they measure only the female component of fitness (Schemske and Bierzychudek 2001, 1272). They acknowledge further that "there are countless other possible pleiotropic effects on flower color that may explain the fitness differences of the two morphs" (Schemske and Bierzychudek 2001, 1279), as well as a variety of potential interactions not represented in their generic equation (Turelli, Schemske, and Bierzychudek 2001, 1295). While the researchers point out these limitations of their research, they do not offer any hard evidence to rule out the possibility that they have misrepresented what is going on in their *Linanthus* population because of the limitations they rehearse.

So along with the experimental and statistical evidence that researchers use to underwrite their affirmation of a specific explanatory causal scenario, we should add plausibility considerations too. Plausibility considerations are used by the researchers to rule out alternative causal scenarios that are compatible with the evidence they garner from their statistical tests and their experiments, for instance explanations that would consign the phenotypic variations that the researchers claim make a *causal* impact on relative reproduction rates to the role of developmental side effects of genes with other causal effects, where it is these *other* unknown and unmeasured causal consequences of the pleiotropic genes that are what is really driving population dynamics. Because causal scenarios can only be affirmed on the basis of limited evidence, evidence that it always possible in principle to explain in other ways, researchers must

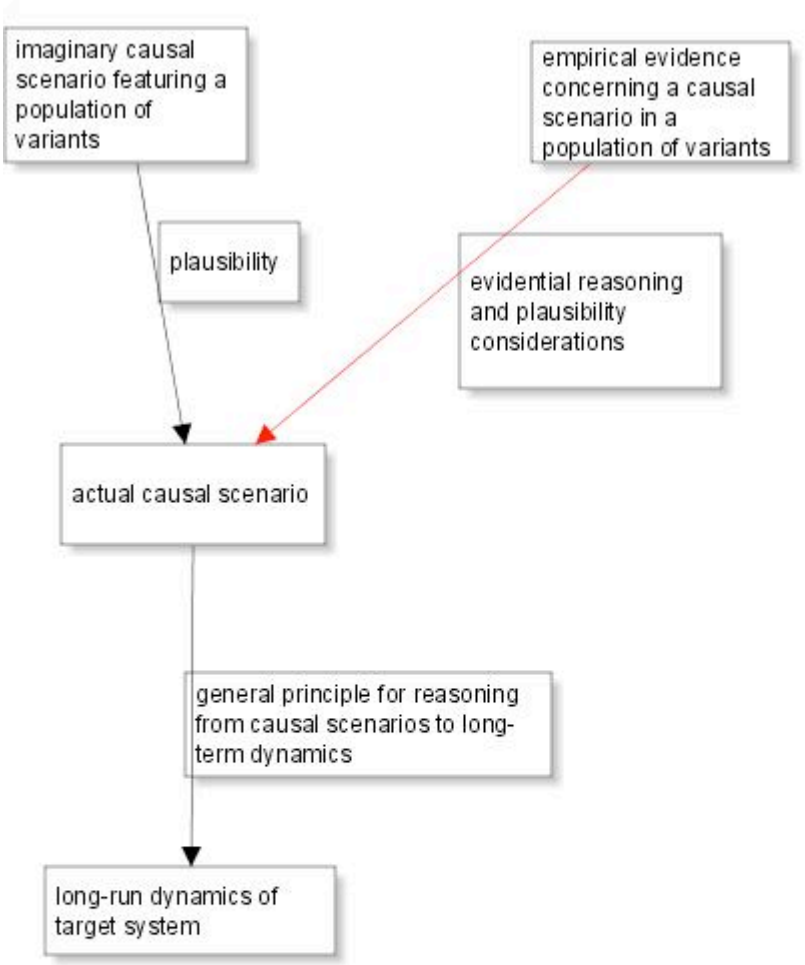
ultimately rule out alternative explanations of their evidence as *implausible*, or at least less plausible than the causal scenarios they offer.

8. Causal Scenarios and Generic Equations

While the researchers invest considerable effort in establishing a causal scenario that would account for the dynamics of their target populations, the link between the causal facts the researchers establish about their populations and the generic equations which they use to explain their dynamics is more or less taken for granted. The real challenge is to establish the causal scenario; that some generic equation adequately expresses that causal scenario in mathematical terms is treated as a straightforward matter.

For instance, Hori appeals to a generic equation featuring frequency-dependent relative fitness functions. However, he does not even bother to write it down. Hori simply asserts that were his causal scenario true, the population would reach an evolutionarily stable state by frequency-dependent selection, and he cites a theoretical discussion featuring frequency-dependent selection favoring rare types as an example of an evolutionary stable strategy (Cresswell and Sayre 1991). Turelli, Schemske and Bierzychudek draw analytic conclusions concerning their population from a generic equation suitable for temporally variable selection in a population with a seed bank. Schemske and Bierzychudek's generic equation does not fit the population exactly; the researchers note a variety of ways in which the mathematics simplifies the actual causal situation (Turelli, Schemske, and Bierzychudek 2001, 1295).

It is worth noting that despite the off-handed attitude on the part of the researchers to the the quantitative representations of their systems, the researchers' explanations of their populations' dynamics depend crucially on formal mathematical reasoning in population genetics. It is because of abstract investigations of frequency-dependent selection models that we know that frequency-dependent selection is the sort of thing that can explain a persistent polymorphism. The importance of formal results to Schemske and Bierzychudek is even more dramatic. According to John Gillespie, Sewall Wright made a mathematical error in his analysis of temporally variable selection and reached what later proved to be a false conclusion, that temporal fluctuations in fitness could not yield a stable polymorphism (Gillespie 1991, 229). The reversal of Wright's putative mistake shows the crucial role of analytic results for Schemske and Bierzychudek's explanation of the *Linanthus* polymorphism: temporally variable selection would have been regarded as even a *candidate* explanation for a persistent polymorphism without the correction.



9. Applied Population Genetics and Adaptationist Reasoning

I want to now unite the two investigations in this paper concerning how adaptationist reasoning and applied population genetics work, with an eye to exhibiting the considerable overlap between the two. Both ultimately attribute population dynamics to a causal scenario that explains those dynamics. Both make appeals to plausibility considerations. And both explain the dynamics of their target populations as a *sort* of dynamics that is liable to result from the causal scenario attributed to the population. Lastly, both exploit determinate versions of general principles of inference that allow one to make inferences about the sort of long-term dynamics undergone by their populations.

In particular, adaptationist reasoning parallels what John Endler has referred to as the functional approach to selection theory:

> It is an excellent trend that more and more people are working on the function and ecology of adaptive traits because the functional approach allows one to predict variation in fitness and evolutionary change rather than simply proving its existence. A particular advantage of knowing the function of a trait in detail is that is allows specific predictions about fitness and the direction of evolution. (Endler 2000, 253-254)

The functional approach is not the only approach to detecting selection in natural populations; signature patterns of variation in the genome can indicate a history of selection at a genetic locus. But for the cases of applied population genetics surveyed here, causal reasoning about populations is critical. The researchers' causal interpretations underwrite their deployments of specific generic equations from which analytic results about the sort of dynamics that can be expected from a population can be, or have been, derived. Without a causal understanding of their populations, researchers would not be in a position even to choose among the great variety of generic equations available for abstract manipulations.

The dramatic difference between adaptationist reasoning and the quantitative cases of applied population genetics reasoning considered here is the effort made by researchers applying population genetics to secure their claims concerning the *reality* of the causal scenarios that they propose are driving their populations' dynamics. Insofar as the researchers deploy quantitative techniques, these are almost always used to justify the causal scenario that the researchers propose is at work in the population. I have accordingly tried to isolate the source of the problem with adaptationist reasoning as its reliance on nothing more than plausibility to underwrite its ascriptions of causal scenarios to populations. Except for this flaw, adaptationist reasoning is not fundamentally different from how population genetics can be applied to natural populations.

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