THE STATISTICAL CHARACTER OF EVOLUTIONARY THEORY*

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This paper takes a critical look at the idea that evolutionary theory is a statistical theory. It argues that despite the strong instrumental motivation for statistical theories, they are not necessary to explain deterministic systems. Biological evolution is fundamentally a result of deterministic processes. Hence, a statistical theory is not necessary for describing the evolutionary forces of genetic drift and natural selection, nor is it needed for describing the fitness of organisms. There is a computational advantage to the statistical theory of population genetics, but population genetics succeeds only by eliminating causes from its account of evolutionary change.

1. Introduction. The received view of evolutionary theory is that it is a statistical theory. The consistency with which philosophers of biology subscribe to this position is remarkable: The view is not merely received, it is entrenched. But as A. D. Woozley observed, speaking of Locke's use of the word "idea", "if a philosopher uses a term which it never occurs to him will cause trouble, the term almost certainly will cause trouble" (1964, xxxii). Such is the status of the claim that evolutionary theory is a statistical theory. It is time for a closer look.

To some people, the phrase "evolutionary theory" means the general theory of evolution, the theory that uses evolutionary "forces" such as natural selection, to explain patterns of phylogenetic relationships. To others it denotes one of the components of this general theory, the theory of natural selection, or the theories of genetic drift, mutation, migration, linkage, or meiotic drive. To still others it refers to what might be called a set of "fitness theories", what are on R. Brandon's (1990) view instantiations of the definition of "relative adaptedness"—hypotheses about the "adaptive significance" or survival value of phenotypic traits.

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Each of these theories has, at one time or another, been the intended subject of the claim that evolutionary theory is a statistical theory. E. Mayr, for example, regards both genetic drift and natural selection as statistical phenomena (see esp. Mayr 1963, 203ff.). D. Hull agrees about drift: The founder effect introduces "an uneliminable random element into evolutionary development" (1974, 62–63) and entails that evolutionary theory is necessarily statistical at the level of populations and species. J. Beatty (1984) might agree about both since he views both selection and drift as sampling processes, the former "discriminate" with respect to fitness, the latter "indiscriminate".

Others have asserted the statistical character of evolutionary theory because of what they see as the statistical character of fitness theories. Beatty and others (e.g., Mills and Beatty 1979; Brandon and Beatty 1984; Brandon 1978, 1990; Burian 1984) have argued that the best characterization of the relationship between phenotypic traits and fitness level is in terms of probabilistic propensities. On this interpretation of fitness, an individual's fitness is its *propensity* to survive and reproduce in a particular environment.

Finally, many subscribe to the received view because they feel that the general theory of evolution is a theory about the evolutionary dynamics of populations and little, if at all, concerned with the fate of individual organisms. C. S. Peirce (1877) compared Darwin's application of the statistical method in biology to Maxwell's use of statistics in describing the behavior of gases. The mathematical geneticist R. A. Fisher observed that "[a] population mating at random immediately establishes the condition of statistical equilibrium between the latent and the apparent form of variance. The particulate theory of inheritance resembles the kinetic theory of gases with its perfectly elastic collisions" (1958, 11). This view is held by contemporary philosophers of biology as well. For A. Rosenberg, evolutionary theory is a statistical theory because it can only tell us about how populations can be expected to evolve "in the long run" (1985, 216; Rosenberg 1988 abandons this argument and reaches a conclusion similar to mine). Similarly, K. Sterelny and P. Kitcher state that evolutionary theory is a statistical theory because, like the theory of statistical mechanics, it aims to "make clear the central tendencies in the history of evolving populations" (1988, 345). Some philosophers have adopted the received view because of a predilection for the statistical theory of population genetics as the core or even canonical formulation of the neo-Darwinian theory of evolution. Thus, M. Ruse (1973, 1988) argues for the advantages of construing evolutionary theory syntactically with Mendel's laws and the Castle-Hardy-Weinberg principle of equilibrium as axioms. E. Sober (1980, 1984) maintains that the population genetics formulation

of evolutionary theory captures important biological generalizations that would be missed by focusing on the behavior of individual organisms.

This paper considers the consequences for *explanation* of accepting the statistical characterization of evolutionary theory. As currently practiced, evolutionary biology uses statistical properties to describe the fundamental evolutionary forces (selection, mutation, migration, and drift), as well as the fundamental concept of fitness. I argue that the statistical approach in biology is instrumentally motivated but *theoretically unnecessary*, given the underlying deterministic character of evolutionary processes. After elaborating this claim I briefly consider two potential objections. The first comes from Sober's (1984) theme that a statistical perspective, and a population genetics perspective in particular, *is* necessary in evolutionary biology. I examine and attempt to refute Sober's reasons for this view. The second objection is implicit in the work of I. Hacking (1990). Hacking argues that the autonomy of statistical theories invests them with an explanatory power that is *not* reducible to their deterministic origins. Here I raise a simple question about parsimony as a justification for the introduction of statistical theories.

The economy of statistical theories underwrites the view that the theory of population genetics is the sum and substance of evolutionary theory. However, as I argue in the last section of this paper, the theory of population genetics is not, and cannot be, a *causal* theory in the sense that it could offer explanations of evolutionary events in terms of the causes of those events. An insistence on a population genetics formulation will seriously compromise the explanatory aims of evolutionary theory insofar as they are understood in terms of the identification and description of the causes of evolutionary change. I conclude that both from the point of view of practice and from the point of view of theory, a more sophisticated understanding of the statistical character of evolutionary theory is needed.

2. Random, Indeterministic or Statistical? Descriptions of evolution sometimes appeal to the concept of randomness, a concept whose great utility owes something to systematic ambiguity. *Physical randomness* is a property of *processes* for which agreement on all physically relevant properties in some initial state *fails* to meet with agreement on those properties in subsequent states (see Earman 1986 for details). *Mathematical randomness*, on the other hand, is a characteristic of *outcomes* or *sequences*, those that cannot be generated by a recursively defined place selection function (Church 1940; von Mises 1964). Physical randomness is thus identical with *indeterminism*; mathematical randomness is a matter of *equiprobability* or *indifference* (see Hacking 1965). As numerous authors have shown, no simple, systematic relationship exists between physical

randomness and mathematical randomness (Reichenbach 1949; Coffa 1972; Hellman 1978; Humphreys 1978). Mathematically random outcomes can be produced by either physically random (indeterministic) or physically nonrandom (deterministic) processes. Hacking's account, for example, leaves open the question whether the "chance set-up", the situation or device that creates the possibility of one or more trials or outcomes, involves deterministic processes, indeterministic processes, or both. Some confusion on this score has no doubt been caused by our inability to distinguish physical randomness from physical nonrandomness on the basis of the (mathematical) randomness of outcomes. R. Giere tags Bayesians with this confusion:

> The most prominent personalists, e.g., Savage and de Finetti, insist that there is only one legitimate concept of probability, that which identifies probability with subjective uncertainty. Once this identification is made, however, one lacks the conceptual apparatus to distinguish uncertainty due to lack of information from uncertainty which no physically possible increase in present knowledge could eliminate. But this is just the distinction between physical determinism and physical indeterminism. (1973, 474–475)

Like the notions of "randomness" and "indeterminism", the concepts of "statistical property" and "statistical theory" are usually explicated in probabilistic terms. Common usage of the latter terms is illustrated by discussions of what are sometimes called "stochastic" theories. Because I am inquiring into differences *between* theories of the form $Pr(B|A) = r$, in particular, between theories that explain mathematically random outcomes using either physically random or physically nonrandom processes, this simple interpretation of "statistical theory" will not suffice. Moreover, I discuss the theory of population genetics, which is a statistical theory with a much richer structure than that given by the above probabilistic formula. We will therefore require an account with a finer grain.

Both indeterministic and deterministic processes may be described by statistical theories. Let us say that a theory is a *statistical theory* if it employs terms referring to *statistical properties*. Among the most common statistical properties are the average, or arithmetic mean, the frequency, the mode, the median, the standard deviation, and the variance. In the kinetic theory of gases, for instance, temperature is defined as the *average* of the kinetic energy of the individual gas molecules. Note that statistics is basically sophisticated counting (even for continuous variables, where the "counting" is a matter of integration). Statistical properties are therefore *numerical* properties. Statistical properties are also *abstract* properties. The average income of a family of four is not a prop-

erty that is possessed by any family in the way that a particular income is possessed by the Jones family. To ask which family possesses the average income is to make a category mistake of the classic sort. Statistical properties are abstract in part because they are properties of *populations*. A single individual cannot have a statistical property, though of course an individual can have a property whose numerical value coincides with that of the statistical property. The Jones family can have an annual income equal to the average income of a family of four. Statistical properties are not necessarily properties of series or sequences; however, a sequence of events, being a population (i.e., a collection or aggregate), can have statistical properties. Statistical properties are mathematical functions of properties possessed by *each and every* member of the population. The average income of a family of four is equal to the arithmetic sum of the incomes of *all* families of four divided by the total number of families. Because their values depend on properties of each and every individual, statistical properties are often, but misleadingly, regarded as *summaries* of the properties individuals in a population possess. A single sentence summarizes an entire paragraph if it says the same thing in an abbreviated fashion. Properties possessed by populations, however, are not the same kind of property as properties possessed by individuals; hence, statistical properties will not be summaries in this sense.

Statistical properties exist whenever individuals are treated as members of an ensemble. Statistical properties can be used either as descriptors called population parameters, or as estimators called sample statistics. Sample statistics estimate the frequency of a property in a long sequence of outcomes, or the proportion of individuals with the property in a fixed, perhaps large, population. Although some Bayesians have protested it, most statisticians, for example, Fisher (1947), have thought that random sampling is necessary for sample statistics to be reliable estimators of population parameters (see also Suppes 1983). The process of genetic drift, even if it involves some sort of sampling process, does not depend on randomness in this sense. The drift of gene frequencies that results from the reproduction of a small portion of the breeding population *creates* the properties the next generation will possess. It does not estimate them. In any case, even if it could be argued that thinking about drift as a sampling process was useful for predicting evolutionary change, it can be shown that the sampling involved in drift is not random sampling. I argue this in section 3.

Statistical properties are useful. Where the number of particles, molecules, or genes is large, and their interactions complex, there is a pressing need for some more convenient way to describe their motion and effects than that of calculating the kinematics or the evolutionary trajectories for all of them taken individually. Add to this the fact that exact

solutions to problems of motion involving more than two bodies are known for only a few restricted cases, or the fact that most population genetics equations are soluble for at most two loci, and the great relief scientists must have felt upon the introduction of statistical methods for the analysis of the behavior of large ensembles is obvious.

I do not wish to diminish our appreciation for the usefulness of statistical properties, but important distinctions are to be drawn among those cases that admit of the statistical approach. While it may often be helpful to consider the macroscopic or statistical properties of an ensemble rather than the microscopic properties of its individual members, it is nevertheless true that in many cases the individual members of the ensemble behave in a deterministic fashion. The molecules in an ideal gas and particles in Brownian motion are two examples. Their behavior is governed by deterministic laws. In these cases, as is well known, a Laplacian supercalculator in possession of a complete state description and knowledge of the relevant laws could predict and explain the behavior of the ensemble in terms of the physically *non*random behavior of its individual members. For the supercalculator therefore, and in principle for ordinary human calculators, statistical properties are *theoretically unnecessary*; that is, they are unnecessary for the explanation of the behavior of the ensemble.

These cases exist in stark contrast to the possibilities for prediction and explanation of the behavior of *indeterministic* systems, whose laws are irreducibly probabilistic. Feinberg et al. (1992) give a clear explication of this fact worth quoting in full:

> [A]ny transition from one quantum mechanical state to another, whether it takes place spontaneously, as in a decay, or as the result of some outside perturbation, can be predicted only statistically, except in very special circumstances, as when there is a very large outside perturbation. The reason for this is the law that governs how any system governed by quantum mechanics evolves in time. According to that law, if we express the initial state as a superposition of states with definite total energy, then each term in the superposition will change over time through being multiplied by a time-dependent complex number of magnitude one. This later superposition can be reexpressed in terms of whatever states correspond to the measuring process, such as an undecayed particle and its decay products. The coefficients in this expansion in terms of the states to be measured, which depend on time, then represent the probability of transitions. Usually, these coefficients are different from zero and one, which means that the probability of a specific transition is not zero or one, but some intermediate value. As a result, the transition does not take place with

> certainty at a specific time, but may take place at any time for which the expansion coefficient does not vanish. (P. 628)

The authors also make clear that the probabilistic character of indeterministic systems is ineliminable. Under the standard formulation of quantum mechanics, for example, it is to be accepted as a "brute fact" about the world. Under alternative formulations, either there are no reasons within the theory for choosing one of a number of different descriptions of the world, each of which make different predictions about the future, or there is no way to identify what additional measurements could be made that would allow precise predictions of future events.

Let us address the force of the in-principle possibility of microlevel explanations of deterministic systems in the face of the overwhelming impracticality of the Laplacian approach. What possible advantage could there be in studying the behavior of individuals when statistical methods exist that are simpler, more elegant, less tedious, and vastly more efficient? There may be no advantage to physicists in looking at individual trajectories, but there may well be an advantage to biologists. It is unfashionable, to say the least, to insist that biology resemble the physical sciences point for point. One important difference is in the character of their domains. Taken as a *kind*, the molecules of a gas, atoms, elementary physical particles, and so on can be considered to be identical in respect of the properties used to explain their behavior, for example, mass, velocity, and energy level. Indeed, quantum statistics arose because elementary physical particles are assumed to be not merely identical, as they are in classical statistical mechanics, but *indistinguishable*. But biological diversity is necessary for evolutionary change. Thus the sort of identity assumptions that physicists like to make about physical particles would contravene the fundamental precept of evolutionary theory. Because physical phenomena are uniform and the physical domain, as a consequence, is homogeneous, statistical methods are not inappropriate. However, one may at least raise the question whether the same approach will succeed in biology, given the variability of the phenomena biologists study and the resulting heterogeneity of the biological domain. This is the question I raise. (See Horan [forthcoming] for a discussion on individuation and complexity in the biological domain and their consequences for methodology.)

It might be argued that statistical properties are necessary for statistical explanations, so that they are necessary even in deterministic cases. This argument requires that statistical explanations should themselves be shown to be necessary. Long before Gibbs, Carnot, and others thought about temperature as a statistical property, the kinetic properties of matter had created that macroscopic property to which the thermodynamic concept

of "temperature" refers. So the idea that statistical theories are necessary for the *existence* of fundamental physical magnitudes like temperature will not show that statistical explanations are necessary. It might more plausibly be argued that macroscopic ensemble properties like temperature *are* essentially statistical properties, and it took until the nineteenth century to create a conceptual framework that could describe them. Even so, the arguments for the statistical character of evolutionary theory are different. These arguments suppose either that the forces of evolutionary change such as selection and genetic drift are *indeterministic*, or that there are statistical properties such as the average fitness or the total phenotypic variance of a population that help us understand the course and cause of evolutionary change. I argue below that the supposition of the first argument is false, and that the supposition of the second diminishes, rather than enhances, our understanding of evolutionary change.

3. The Statistical Character of Evolutionary Theory. I now consider in detail three arguments that are often given in support of the received view: (1) the argument based on the statistical character of evolutionary forces, especially genetic drift; (2) the argument about the statistical character of fitness theories; and (3) the argument that claims population genetics to be the best formulation of evolutionary theory. I use the distinctions drawn in the last section to argue that unclarity about randomness, indeterminacy and statistical properties has led to misunderstandings about the statistical character of evolutionary theory. If this theory is a deterministic theory, then the statistical properties it employs are, in principle, defeasible.[1] Arguments for its statistical formulation will then depend on the utility of statistical properties in algorithms for computing gene frequency change. However, this brings with it epistemological costs that outweigh the benefits of convenience. Construed as a statistical theory, evolutionary theory cannot be a theory of causes.

3.1. Genetic Drift. Genetic drift is typically described as the "chance fluctuations in allele frequency . . . particularly in small populations as a result of random sampling among gametes" (Hartl 1980, 142). Such

[1]Mutation is a likely exception to the claim that evolutionary forces are completely deterministic. Some mutations are known to result from genuinely indeterministic processes. Lewin (1985, 46–47) explains the mutation of a G-C base pair into an A-T base pair in the presence of the mutagen bromouracil in terms of an enol-keto tautomeric shift—a quantum physical phenomenon. There has been some discussion in the literature about whether the theory describing genetic processes that create the basis on which evolutionary forces act should be regarded as *part* of the theory of evolution or *ancillary* to it (see, for instance, K. Neander's 1988 criticism of Sober 1984). Standard population genetics textbooks treat mutation as a *source* of variation for, for example, natural selection. The randomness of the *process* of mutation might therefore be regarded as irrelevant. For discussion on a related point in archaeology, see M. Salmon (1982).

descriptions suggest that a "chance fluctuation" of gene frequencies is the consequence of a genuinely random sampling process whose effects require a statistical explanation. I argue that the appearance of random sampling is created by the sampling character of drift but that the sampling is not genuinely random. Therefore, the effect of drift on evolution gives no reason to suspect the deterministic character of evolution or to doubt the eliminability of statistical properties from evolutionary theory.

Random samples can be obtained by means of completely deterministic processes, but, of course, not all deterministic sampling processes will create random samples. Such is the case with genetic drift. What is required to obtain a random sample is, to follow Hacking's (1965) discussion, a *chance setup* that produces a sequence of outcomes or results from consecutive independent trials such that, for each trial, the probability of any one outcome is equal to the probability of any other. The resulting set of outcomes is a random sample. Beatty (1984) contrasted drift, which he called "indiscriminate sampling", with the "discriminate sampling" resulting from natural selection. Insofar as both drift and natural selection are regarded as sampling processes, their results are equally "chance-like". Thus regarded, the difference between them must lie in the randomness of the sampling process. Because the key to random sampling is the equiprobability of outcomes under something like Hacking's chance setup, we might say that the indiscriminate sampling of genetic drift is random, while the discriminate sampling of selection is not. Can this distinction, however, be drawn in fact?

Genetic drift causes an undirected shift in gene frequencies that respects neither the fitness of organisms nor the representativeness of alleles in the gene pool. As far as we know, the macroscopic environment faced by individual organisms is replete with deterministic processes, so all possible worlds that agree with this one in all respects relevant to the origin, course, and extent of a natural disaster, such as a famine or flood that creates a founder population, will also agree on the subsequent sample of breeding individuals. Fires, floods, famine and disease sample from a large population by eliminating individuals from the reproducing group. The same fire, the same flood, in the same conditions would create the same sample. However, it would not be a *random* sample.

There is contingency about drift, but not the equal likelihood of outcomes that is the hallmark of random sampling. Consider a brush fire that eliminates 90 percent of a breeding population. The individuals that survive may be regarded as a *sample* from the original population. *Ex hypothesi*, they will have become part of the sample in a manner that has nothing to do with the superior fitness of their phenotypes. But the sampling process will not be random either. Animals on the periphery of the population may escape the blaze simply because they happened to be far

enough away to outrun it, even though had they been near the center they would have been caught, and even though their equally speedy cousins perished. It might be argued that individuals on the periphery were somehow better adapted than individuals near the origin of the fire, but surely not that they were better adapted to survive *this particular* fire, which like all natural catastrophes, would have struck indiscriminately. The individuals who are progenitors thus carry a sample of genes from the gene pool of the larger population, but not a random sample of genes. The genes of centrally located animals had no chance of being in the sample.

The "chance-like" or indeterministic appearance of drift is created by the indiscriminateness of its sampling from the breeding populations: Organisms are chosen in a way that is completely independent of how fit they are. If the sampling process is also deterministic, then the statistical perspective introduced by the concept of "sampling" is not necessary. It is convenient and, given limitations in our knowledge of the vagaries of the environment, useful; however, it obscures the essentially deterministic character of evolutionary change. Similar remarks apply, mutatis mutandis, to "gamete sampling". The notion that gamete sampling is indefeasibly random is countermanded by what is believed to be the deterministic character of the meiotic process.

3.2. Fitness and Natural Selection. Nature's *contretemps* make the relationship between numerical measures of fitness and actual reproductive success probabilistic. Lightning strikes can reduce an organism's reproductive success to zero in a way that has nothing to do with its abilities to defend territory or flee from predators. If the only evolutionary force acting on a population were natural selection, fitness and reproductive success would be perfectly correlated. In this case there would be no need for a propensity account of fitness, which makes sense only relative to a realistic concept of the environment that includes possibilities for the elimination of organisms without regard to fitness differences (see Waters 1986). But if fitness theories describe a supervenient relationship, such that any two organisms with identical phenotypic traits subject to the same selection pressures must have the same level of fitness, then the relationship between phenotypic traits and fitness is deterministic (Rosenberg 1985). The propensity account of fitness thus does not show that fitness is a function of indeterministic forces that make fitness theories essentially statistical theories. It shows that reproductive success is not solely a function of fitness.

Let us now consider what it might mean to say, as Sterelny and Kitcher do, that fitness coefficients can be used to "represent the expected survivorship and reproductive success of organisms" (1988, 345). What does "representation" amount to? A clue is provided by Sober, who writes that

"genotypic fitnesses are survival probabilities, which is to say that they represent the average chance an organism of a given type has of surviving from egg to adult" (1984, 43). One plausible reading of Sterelny and Kitcher's claim is that fitness coefficients are quantities in population genetics models that stand for the expected survival and reproductive success of particular genotypes.

The relationship between the fitness coefficient of a genotype and the fitness of an individual organism possessing that genotype is far from clear. Let us consider this matter in the context of an example. Van den Berghe and Gross (1989) studied reproductive success in female Pacific salmon and found that females with large bodies are stronger, swim farther, and spawn earlier than females with small bodies. It makes sense (and is evidently true) to say that within a given population, large female salmon are more fit than small salmon, meaning that they have greater expected reproductive success. It is also true that the fitness coefficient of the genotype for large body size will be greater than that of a genotype for small body size, the fitness coefficient of a genotype being simply the average of the numbers of offspring produced by each individual of that type. But the fitness coefficient of the genotype for large body size does not *represent* the fitness values of the individuals from which it was computed in the sense that it "summarizes" individual fitness values. Fitness coefficients are statistical properties, and do not attach to individuals.

Moreover, if the fitness levels of organisms are the result of a deterministic process of natural selection in the sense that organisms with identical body size living in identical environments would have exactly the same level of reproductive success, then, although the relationship between an organism's fitness and its reproductive success might *usually* be expressed probabilistically in view of our ignorance about the environment, it *need* not be characterized in statistical terms. Here is an interesting juxtaposition. The fact that we understand the fitness of individual organisms in terms of their probabilistic propensity for survival and reproductive success is an acknowledgement of the variation in individual fitness values that is then *erased* by the attempt to use fitness coefficients as summaries of the fitness values of individual organisms. Even in a population in which every organism has the same genotype at loci responsible for body size, individual body size will vary because of environmental variation in available nutrition, individual variation in growth rate, exercise, and so on. If organisms with identical genotypes had exactly the same fitness levels, the fitness coefficient would describe—and represent or summarize—the fitness values of individual organisms simply because the value of the fitness coefficient would coincide exactly with the value of the fitnesses of individuals. Individual fitnesses would

not vary; hence, statements about fitness coefficients would give us information about the fitnesses of individual organisms. This understanding of "representation" thus presupposes not only that individuals in such a population have the same genotype, but also that they inhabit an environment that is identical with respect to factors determining body size. Recall that the propensity account of fitness was introduced because fitness levels for organisms with the same genotype will vary as a result of environmental accidents. The implicit contradiction between the propensity interpretation of fitness and the use of fitness coefficients to represent the fitness of individual organisms becomes apparent. If it were not for environmental accidents, catastrophes, and so on, there would be a perfect correlation between an organism's fitness level and the number of offspring it produced. Fitness coefficients *would* represent individual fitness values, but then the propensity account would not be needed. Alternatively, if the propensity account of fitness is to be nontrivial, it will be because there is variation in fitness levels among organisms with identical genotypes caused by environmental accidents. However, under these conditions fitness coefficients will not represent organisms' fitness levels.

So, fitness coefficients "represent" the fitnesses of individual organisms neither in the sense that they summarize the fitness values of individuals nor in the sense that they provide us with means to infer the fitness of individual organisms. As a last resort we might appeal to the theory of population genetics to supply a term that can be related to the fitness coefficient, namely, "average population fitness", which is computed by multiplying the frequency of an allele by its fitness coefficient and then adding together the products for every allele at the locus. It must not be thought that this use of the fitness coefficient means that a *population* of individuals with a genotype for large body size itself has a fitness level, which borders on "good of the species" reasoning. On the other hand, to suppose that the geno*type* for large body size has a fitness level in the sense that individual organisms have fitness levels is to resort to the worst kind of typological reasoning, precisely the thing to have been avoided in the first place by what Mayr (1975) called Darwin's "population thinking".[2]

3.3. Population Genetics. Population genetics is a discipline concerned with how genetic variation in populations originates and evolves. It employs as state variables descriptions of the genetic structure of pop-

[2]Note that Mayr's concern was to explain the shift in systematics away from thinking of species as types described in terms of essences toward thinking of them as composed of unique individuals who can be described as a group only in statistical terms. An emphasis on population thinking will not show that the theory of evolution is a statistical theory, since for the population thinker, individual properties are real and statistical properties are mere abstractions. For more discussion on this point, see Sober (1980).

ulations and as transformation laws rules governing the (re)distribution of genotypes within and across generations that results from mating, migration, selection, mutation, drift and Mendelian inheritance (Lewontin 1974).

S. Wright pointed out that population genetics systematizes the various causes of evolutionary change by focusing on their *effects* on gene frequency change:

> We need a means of considering all factors at once in a quantitative fashion. For this we need a common measure for such diverse factors as mutation, crossbreeding, natural selection and isolation. At first sight these seem to be incommensurables but if we fix attention on their effects on populations, rather than on their own natures, the situation is simplified. Such a measure may be found in the effects on gene frequency in each series of alleles. (1942, 224; see also Wright 1931)

A theory of evolution that focuses on the changing distribution of gene frequencies in populations, if this is all it does, cannot by itself explain these changes since a mere description of effects is itself in need of explanation. It might be thought that the transformation laws of population genetics do provide an explanation in the sense that they tell us how gene frequencies will change from one generation to the next as a result of the action of various evolutionary forces like natural selection and genetic drift. Let us note, however, that the transformation laws of population genetics are equations that relate sets of effects to one another. They do not relate causes to effects. Consider the following equation, which gives the magnitude of change $\triangle q$ in the frequency of an allele a in one generation as a result of gamete selection:

$$\triangle q = q_1 - q_0 = q_0(1 - s)/(1 - sq_0) - q_0 = -sq_0(1 - q_0)/(1 - sq_0).$$

Here q_1 is the frequency of allele a after selection; q_0 is the frequency of allele a before selection; and s, the selection coefficient against a, is $1 - W_a$, where W_a, in turn, is the proportion of a alleles in one generation that are reproduced in the next. (Thus if for a particular population p_1 there are 100 copies of the a allele at time t_0 and 99 copies at t_1, $W_a = 0.99$ and $s_1 = 0.01$. The selection coefficient $s_a = \Sigma_{s_i}/n$, $i = 1, 2, \ldots, n$ populations in which the a allele occurs.) The evolutionary force at work in this case, natural selection on gametes, is represented in this equation by the selection coefficient s, which is itself defined in terms of its effects, namely, the proportion of a alleles that survive to the next generation. What *causes* these effects—for instance, the differential viability of gametes or gametic incompatibility—has dropped out of the picture.

It might be objected that although the theory of population genetics

focuses on the effects of evolutionary forces on population gene frequencies, it is nevertheless a causal theory. What was Wright (1921) doing when he developed the method of path analysis if not spelling out the consequences of Mendelian inheritance for breeding populations, and using statistical methods for estimating the magnitude of those causes (Griesemer 1991)? Wright's use of path analysis depends crucially on the assumption that the causal relations between variables are *already known*. Path analysis gives us an efficient handle on their effects. Wright's biographer, W. Provine, goes to some length to clarify this point:

> Wright's experience analyzing MacDowell's bone data in the period of 1914–18 convinced him completely that the array of correlation coefficients or partial correlation coefficients (and he would later add any set program of statistical analysis) *could not yield by itself the pattern of causal relations among variables*. What was required was an initial scheme of causal relations of the variables deduced from all available evidence from every source (including of course correlation coefficients, space-time relations, etc.), followed by the attempt to quantify the causal relations already deduced. (1986, 131; emphasis added)

(Wright 1921, in the introduction, dissuades us from interpreting his method of path analysis as a means for deducing causal relations.)

Thus the objection is without force. Wright's introduction of path analysis as a method of quantifying the causes of gene frequency change does not show that population genetics supplies causal explanations of that change. Indeed, as Provine points out, the causal structure must already be in place in order for path analysis to have any application. So although a causal story does underlie the theory of population genetics, population genetics does not tell this causal story. It can describe and systematize the diverse forces of evolution only by *disregarding* evolutionary forces as causes in order to take advantage of their effects on gene frequencies as the common measure of evolutionary change. This move undoubtedly simplifies the determination of the genetic composition of evolving populations, but a price is paid for this computational achievement. I suggest that in shifting theoretical attention from causes to effects, the explanatory project of evolutionary theory is supplanted by the instrumentalist's objective of predictive success.

But is there an alternative? Are statistical properties really defeasible in the case of deterministic processes? Hacking's (1990) social history of the emergence of statistical laws argues for the concomitant erosion of determinism and of the concept of causality. He offers a rich story of the counting and measurement of human traits, especially of the deviant, the diseased and the dying. The heroes of this story are Quetelet, "the great-

est regularity salesman of the nineteenth century" (ibid., 105), and Galton, "who led us to the autonomy of statistical laws" (ibid., 177). Quetelet gets credit for the idea that statistical properties are not merely abstract mathematical contrivances, but real, that is, objectively existing, properties of populations. Hacking describes this "crucial step in the taming of chance" (ibid., 108) as a transition from the reality of the physical properties of individuals to the postulated reality of statistical properties, such as the mean height, "a number that objectively describes the population" (ibid., 109). Galton took this idea one step further. He saw that measurements of a population of individuals conformed to statistical laws generation after generation, and that therefore the *laws* could be regarded as independent from the individuals and their properties. "In one stroke [Galton] was (a) explaining and (b) leaving out the 'host of petty independent causes' story. He was regarding the Normal distribution of many traits as an autonomous statistical law" (ibid., 186).

The possibility of explanation by reference to the objective properties of populations constitutes what Hacking calls the *autonomy* of statistical laws. Such explanations do not, need not, and on Hacking's view, should not appeal to an underlying causal metaphysics. "The host of petty independent causes story" (ibid.)—the idea that the dispersion of single measurements of the height of different individuals should be explained by the same *causal* theory of errors that explains the dispersion of repeated measurements of height for the same individual—is one that Hacking finds *incoherent*, a vestige of a decaying deterministic worldview.

A similar conviction about the autonomy of statistical laws is evident in Sober's insistence on the importance of population thinking in evolutionary theory (see esp. Sober 1980). Sober (1984) argues that the importance of the statistical approach to evolution is that it *broadens* the explanatory scope of evolutionary theory. Population thinking enables us to subsume different populations under one general theoretical framework, whereas the Laplacian approach, which concentrates on the interaction of environments with single organisms, will miss biologically important generalizations. Although he agrees that the Laplacian supercalculator may not need statistical properties to give a microlevel explanation of the evolution of individual populations, Sober does not regard this as sufficient reason for abandoning the statistical approach. This would be "a precipitous application of Occam's razor" (1984, 129). The Laplacian explanation of the evolution of populations taken individually cannot achieve what the statistical approach does—a single theoretical framework that treats many different populations in a unified fashion. The statistical concepts of evolutionary theory, he writes, "have an autonomous explanatory power, whether determinism is true or not" (ibid.).

I have argued that deterministic systems allow, but do not require, ex-

planation in terms of statistical properties. Sober has introduced a reason for *preferring* the statistical approach to that of the Laplacian. I make one simple observation on the argument about autonomy offered by Sober and Hacking. Both admit that Laplacian, microlevel explanations of the behavior of single populations would differ from one another. But why would they differ? Presumably because their "petty individual causal stories" would differ, because populations vary, as do all biological phenomena—in their phenotypic and genotypic composition, in their environmental circumstances, and in their response to various evolutionary forces. If the microlevel causes differ, then the causal explanations should differ. The statistical approach Sober and Hacking defend requires that we neglect these differences in order to explain more with less—an argument from parsimony. It may be the statistical approach, therefore, and not the Laplacian, that precipitously applies Occam's razor (see Sober 1990 for a more sensitive treatment of Occam's razor).

Sober's defense of the population genetics approach to evolutionary theory parallels Dennett's (1987) argument for the intentional stance in psychology, which makes an epistemic virtue out of instrumentalist necessity. Dennett reasons that what justifies the ascription of intentional states (mental representations) to organisms are the *patterns* they reveal in its behavior. We would miss these patterns by concentrating on physical or even physiological descriptions. On his view we are justified in attributing intentional states like beliefs, desires, and so on to any system so long as we are able to successfully predict its behavior under the assumption that the system possesses and acts upon its intentional states. The intentional states are then understood to be those patterns of behavior we predicted; or, as Dennett puts it, we are entitled to interpret those patterns of behavior as being regulated by internal states that thereby qualify as representations, that is, as intentional states. *Which* intentional states such systems possess is fixed by a semantic interpretation that depends on a more or less complex, more or less complete, embedding of the system in the world. Thus his instrumentalist criterion of belief constrains the internal constitution of belief, and we are given back a robust, that is, realistic, concept of belief in the end.

This argument calls our attention to "objective, real pattern[s] in the world" (ibid., 34) that would be missed by the Laplacian strategy of focusing on causal mechanisms producing the behavior of individual systems. Like Dennett, Sober insists that the strength of the predictive strategy lies in its ability to detect "biologically significant generalizations" (Sober 1984, 130) that would go unnoticed by an emphasis on the fate of individual organisms subject to various evolutionary pressures. These generalizations are important, presumably, because either (i) they are important in their own right, or (ii) they provide us with clues to the pro-

cesses that underlie them, that is, because they give us back the causal story in the end. To advance (i) as a reason for adopting a population genetics formulation of evolutionary theory is clearly circular since the generalizations are none other than the equations of population genetics itself. But to advocate (ii) is to ignore what Lewontin (1985) has argued are insurmountable limitations to the population genetics approach to evolution.

A number of factors impede the program of computing the future genetic state of a population from information about its present state and the evolutionary forces acting on it. It is difficult to measure actual mutation and migration rates, developmental schedules and genotypic norms of reaction. It is difficult to identify the genotype for a particular character. Development, survival, reproduction, and behavior are contingent on a variable environment. Necessary information about past environments is impossible to acquire. The evolutionary pathways for all but the simplest traits are very complex. These problems force what Lewontin calls an *inversion* of the theoretical structure of population genetics. Instead of starting with information about initial states and predicting subsequent states, population geneticists must use information about subsequent states to make inferences about initial states. This is typical of certain kinds of statistical inference, but in population genetics the strategy is unsuccessful. In some cases, for example, Comstock's estimate of the average degree of dominance over all loci, the inverse inference depends on the use of an average quantity that estimates the parameter of interest only under certain simplifying assumptions, such as the absence of epistasis and linkage, so that different experiments yield different values of the parameter. In other cases the inverse inference is simply invalid: Whereas it is possible to predict gene frequency changes when fitness values of organisms are known, it is not possible to infer fitness values from genotypic frequencies. Finally, because causes and effects are often related as mathematical reciprocals, inverse inferences to the values of evolutionary parameters are subject to perturbations that may be small in one direction but very large in the other. Errors in estimates of effective population size N and actual population size n become enormous when these parameters are used as reciprocals as they must be in inferring effective population size from the frequency of allelism of lethal genes, "The examples given are not exceptions, but the rule. . . . [I]t will almost never be possible to invert the relation that predicts genetic structures from parameters of causal processes, in order to estimate the intensities of these processes" (ibid., 9).

Population genetics does not tell the causal story of evolution; if Lewontin is right, it *cannot* tell it. It surpasses all other current theories at discerning patterns of evolutionary change, but the systematization it achieves

diminishes rather than enhances the explanatory power of evolutionary theory. Although the current wisdom on the subject is that evolutionary theory is a statistical theory because it can be expressed in the language of population genetics, if our goal is an explanatory theory, we must consider the possibility that population genetics is not our best theory of evolution.

We must consider one last objection. In an interesting defense of theoretical population ecology G. Cooper (1990) has suggested that "equilibrium explanations" (p. 170) and explanations involving "radically supervenient properties" (ibid.) such as fitness "organize causes simply by their effects" (ibid.). On his view their explanatory power lies in "showing that, given the satisfaction of certain constraining assumptions, the system will be in that state regardless of the particular causal details at work" (ibid.; see also Woodward 1979). For Cooper the inadequacy of a causal model of explanation to accommodate explanations in population genetics and population ecology demonstrates the need for a new model of explanation.

I prefer a more conservative reading of this failure of fit. Not everything that scientists call an explanation *is* explanatory. It is perhaps a reflection of the scientism of the age that philosophers of science give away their sovereignty on the question of what counts as an explanation—a question which, it might be ventured, they have thought about more than anyone else. Scientific theories perform many tasks, but to think that all of them are explanatory tasks is a mistake. Had Copernicus computing power equal to what we have today, the challenge of adding a few more epicycles to an already cumbersome astronomical theory would have easily been met, and willingly if it increased the predictive power of the theory, "But this would not have satisfied Copernicus—not only, one likes to think, because of his mystical attachment to circles, but because *such an answer sacrifices understanding to prediction and control*" (Caws 1963, 163; emphasis added). In some cases this sacrifice must be made—I am in great sympathy with Cooper over the present state of theoretical ecology, which, as he points out, is in many instances *neither* predictive *nor* explanatory. Predictive success may in such situations appear the most readily attainable goal, and given our current ecological crisis, the most pressing. But dangers arise in adopting an instrumentalist criterion of theoretical success, namely, (i) that this goal obscures the importance of causes as a ground for scientific explanation and (ii) that it encourages a *complacency* about scientific theories manifested in a peremptory satisfaction with prediction and control.

4. Conclusion. Evolutionary theory—as a general theory of evolution, a theory about one or another evolutionary force, or as a fitness theory—

is deterministic. In this respect it is like classical statistical mechanics: Its statistical character is defeasible by sufficient information. Unlike that theory, however, there is at present no analogue to the Newtonian picture to reassure us that, despite our reliance on its statistical character, evolutionary theory nevertheless gives us a genuine understanding of the causes of evolution. The principles of population genetics enable us to predict gene frequency change, but if I am correct about its suppression of causes, those principles cannot explain it. We may want to reject a causal model of explanation on the grounds that it does not fit the sort of explanation given by population geneticists. I am suggesting, instead, that we retain the causal model and admit that since population genetics does not fit it, it cannot explain.

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