How Do Natural Selection and Random Drift Interact?

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One controversy about the existence of so called evolutionary forces such as natural selection and random genetic drift concerns the sense in which such "forces" can be said to interact. In this paper I explain how natural selection and random drift can interact. In particular, I show how population-level probabilities can be derived from individual-level probabilities, and explain the sense in which natural selection and drift are embodied in these population-level probabilities. I argue that whatever causal character the individual-level probabilities have is then shared by the population-level probabilities, and that natural selection and random drift then have that same causal character. Moreover, natural selection and drift can then be viewed as two aspects of probability distributions over frequencies in populations of organisms. My characterization of population-level probabilities is largely neutral about what interpretation of probability is required, allowing my approach to support various positions on biological probabilities, including those which give biological probabilities one or another sort of causal character.

1. Introduction. Biologists (e.g., Gillespie 1998; Roughgarden 1979) often say that evolution is the result of the interaction of what they call evolutionary "forces" such as natural selection, mutation, random drift, migration, and mating preferences. Sober (1984) argued for a realist interpretation of such talk, claiming that it describes factors which causally influence evolution. Recently, some authors have attacked this realist interpretation of "force" talk (Walsh 2000; Matthen and Ariew 2002, 2005; Walsh et al. 2002). Aspects of Sober's conception have been defended

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(Stephens 2005; Reisman and Forber 2005; Shapiro and Sober 2007).[1] However, advocates of a realist interpretation have not presented a clear, unified conception of what it is for forces of evolution to interact. In this paper I will tackle what seems to be the hardest case for realists: giving an account of how natural selection and random drift interact.

In particular, I will argue that both selection and drift are aspects of a probability distribution over future frequencies of genotypes or phenotypes in a population. Selection is the aspect of the distribution controlled by differences in fitness, while drift is the aspect of such a distribution controlled by population size (apart any from effects of population size on fitness). This distribution is derived from probabilities concerning individual organisms. I will argue that if probabilistic causal factors govern the fate of individual organisms, then the distribution over future frequencies in a population will also be causal. Since there are existing views which make the probabilities which govern fates of individual organisms causal, my account allows the possibility of a clearer understanding of the sense in which random drift and natural selection are causal.

"Force" may be somewhat misleading since it suggests that evolutionary forces are closely analogous to Newtonian forces. "Causal factor" would work as well as "force" for my purposes, but the latter term is common in evolutionary biology. As will become clear, I do not think that evolutionary forces combine by vector addition.[2] Sober (1984) made much of the analogy between interaction of evolutionary forces and vector addition of physical forces, though he did not require that evolutionary forces combine by vector addition. Matthen and Ariew (2002) criticize the idea that evolutionary forces combine by vector addition, and Stephens (2005) discusses ways in which evolutionary forces are and are not like physical forces. Despite my inclinations toward realism about evolutionary forces, my characterization of interactions between so-called forces of evolution will be reminiscent of some anti-realists' (Matthen and Ariew 2002, 2005) claims that factors involved in evolution combine via rules of probability.

The view presented here is consistent with arguments in Reisman and Forber 2005 and Shapiro and Sober 2007 that drift and selection are distinct causal factors because they can be independently manipulated by changing, respectively, population size and fitness differences. My view thus satisfies Woodward's (2003) approach to identification of causes via manipulability. However, the present view goes farther than Reisman and Forber's and Shapiro and Sober's in that it makes it clearer *how* the

1. Other sorts of responses to the anti-realists can be found in Bouchard and Rosenberg 2004; Rosenberg and Bouchard 2005; and Brandon and Ramsey 2007.

2. Nor, perhaps, must physical forces (Corben and Stehle 1994, 29).

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manipulation of population size affects evolution. More particularly, I show how, if individual-level probabilities are causal, then population-level probabilities are as well, even though these population-level probabilities are partly controlled by population size and partly controlled by fitness differences.[3] This provides a way of responding to Plutynski's (2005) argument that mere manipulability is not enough to show that drift is a cause of evolution.

Like Millstein (2006) I will argue that selection is a population-level causal factor. What I add to Millstein's claim is an account of how natural selection is nevertheless constituted by individual-level probabilities. My view is that natural selection as a force is constituted by fitness differences among individual organisms in a population, where fitnesses are constituted by certain probabilities.

2. Fitness and Probability. My account of the interaction of natural selection and drift grows out of what is known as the propensity interpretation of fitness (PIF), although it does not depend on all aspects of the PIF. A brief introduction to the PIF will be helpful.

Natural selection involves, *prima facie*, differences in organisms' fitnesses.[4] The propensity interpretation of fitness is a relatively popular account of what fitness is. In its simplest form the PIF says that one organism or one kind of organism is fitter than another if its expected number of offspring—its average number of offspring—is greater than the other's (Brandon 1978, 1990: Mills and Beatty 1979; Burian 1983; Richardson and Burian 1992). Here the "average" is not an average of numbers of offspring produced by actual organisms. Rather, we look at an organism's probability of producing various numbers of offspring in a given environment. We then compute the average by weighting these numbers of offspring by their probabilities and summing the resulting numbers. Further, according to the PIF, these probabilities of producing various numbers of offspring are to be objective probabilistic dispositions known as propensities (this use of the term was originated by Popper [1959]).

The PIF is controversial, and even many of its sympathizers think that

3. As I use the terms, an individual-level or population-level probability is a probability of an outcome concerning an individual organism or population, respectively. An individual-level or population-level causal factor is a causal factor that can affect the state of an individual or population, respectively.

4. That frequencies change is often taken to be a necessary condition for natural selection to occur. That is a legitimate use of "natural selection," but natural selection in this sense cannot explain evolution, since evolution is then a part of what natural selection consists in.

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it must be revised. First, there are reasons to think that the relationship between fitness and propensities to produce offspring cannot be captured with a simple average, although this issue will not concern us in this paper (see Beatty and Finsen 1989; Brandon 1990; Sober 2001; Krimbas 2004; Ariew and Lewontin 2004). Second, there are doubts about whether propensities can ground fitness even if they exist (e.g., Graves et al. 1999; Abrams 2007). There are also debates about what propensities are and whether any such things exist (Eagle 2004). However, since I simply want to use propensity to illustrate the idea of a causal probability, I need not address any of these debates here. For my purposes, all that matters about propensities is that they are supposed to be (indeterministic) dispositions of some sort, so *mutatis mutandis*, propensities inherit the properties of whatever account of dispositions you favor. It may be that biological properties are not propensities, but that biological probabilities nevertheless are causal in some sense. There are explorations of other kinds of probabilities that might support a probabilistic account of fitness (Millikan 2000; Weber 2001; Rosenberg 2001; Millstein 2003; Bouchard and Rosenberg 2004; Sober 2000, 2007; Abrams 2005).

Evolution consists in changes of frequencies of biological types in populations. But what happens to a population is ultimately the result of what happens to individuals in the populations. Debates between realists and anti-realists about forces of evolution sometimes involve debates about the way in which individuals play a role in evolution, and about whether evolutionary theory itself involves claims about individuals. Anti-realists (Walsh 2000; Matthen and Ariew 2002, 2005; Walsh et al. 2002) argue that evolutionary theory concerns properties of populations, and that the only causal factors responsible for evolution concern very particular facts about particular individual organisms. These are physiological and chemical and physical facts, and facts about particular configurations of organisms and abiotic elements. Though such facts are often biological, but they lie outside the province of *evolutionary theory* proper. Thus evolutionary theory would not concern causes of evolution. The anti-realists oppose views like the propensity interpretation of fitness, according to which natural selection is defined in terms of fitness differences, in turn defined in terms of propensities. Although these propensities arise at the level of individuals, the PIF would make fitness causal (in the sense that dispositions are causal). Since fitness is a central concept of evolutionary theory, and natural selection can be defined partly in terms of fitness differences, the PIF would place one of the causes of evolution within evolutionary theory.

In this paper I cannot address all aspects of the debates between the realists and the anti-realists. In particular, the paper will not directly address the question of whether individual-level causal factors are in the

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province of evolutionary theory. Instead, I will show how, if there are individual-level probabilities which are in some sense causal, natural selection and drift are causal in the same sense, and natural selection and drift interact. If the PIF is correct, this framework will provide an elaboration of the PIF. However, I want to leave room for the possibility that some other kind of probability should play the role that propensity plays in the PIF.

3. Derivation of Population-Level Probabilities. From probabilities concerning individuals in a population, we can mathematically derive probabilities concerning frequencies of types in populations. There are a number of common mathematical techniques from the theory of Markov processes for modeling this relationship. (See, e.g., Bharucha-Reid [1960] 1996; Grimmett and Stirzacker 1992. Roughgarden [1979] and Grimmett and Welsh [1986] provide introductory material.) I will illustrate the idea with relatively simple mathematical models below. More complex techniques could be used to deal with the details of more realistic examples.

I am not claiming, however, that existing mathematical techniques allow tractable analyses detailed enough to capture the subtleties of the probabilities in a real biological population. I do claim that if there are objective probabilities of the right kinds concerning the fates of each organism in a population (perhaps conditional on the presence, location, and properties of other members of the population, environmental circumstances, etc.), then these probabilities combine mathematically to produce a probability distribution over frequencies in the population. That is so whether we are able to calculate those probabilities or not. The important point, then, given the derivability of population-level probabilities from individual-level probabilities, is this: If lower-level probabilities are objective and causal, then the population-level probabilities derived from them will have those same properties, because the derived probabilities will be of the same kind. Thus causal probabilities concerning individuals will determine causal probabilities concerning populations. I will illustrate this idea next.

Suppose we have a seasonally-reproducing population of fixed size N containing haploid organisms with alternative genotypes A and B. Let i represent the number of A's in the parent generation, and j the number of A's in the next generation. If there are no fitness differences between A and B, we can assume that each organism has an equal probability of contributing to the next generation. A common way of explaining how to calculate the probability P_{ij} of a transition from a population of i A's to one with j A's is to say that we take N samples from the parent generation and consider the possibility that j of the samples turn out to be A's. The probability p of an A contributing to the next generation in

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a single sample is then the relative frequency of A's in the population, and similarly for the probability q of a B contributing to the next generation:

$$p = \frac{i}{N}, \quad q = \frac{(N - i)}{N}. \tag{1}$$

Here P_{ij} is the probability of the set of sequences of N samples in which A is chosen exactly j times—samples from the offspring of a population with i A's. The probability of a particular sequence of samples which includes exactly j individuals with the A genotype is the probability p of getting an A in j instances, multiplied by the probability q of getting a B in $N - j$ instances. Thus the probability of such a sequence is $p^j q^{N-j}$. There are usually several possible sequences with the same numbers of A's and B's but with the A's and B's in different orders. So to get the probability of a new population including exactly j instances of the A type, we will have to multiply $p^j q^{N-j}$ by the number of such sequences; this number can be shown to be

$$\binom{N}{j} = \frac{N!}{j!(N - j)!}.$$

Putting this all together, the probability P_{ij} of a population going from i A's to j A's in the next generation is[5]

$$P_{ij} = \binom{N}{j} p^j q^{N-j} = \binom{N}{j} \left(\frac{i}{N}\right)^j \left(\frac{N - i}{N}\right)^{N-j}. \tag{2}$$

Thus, beginning from an assumption that each organism has an equal probability of contributing to the next generation, and that there are i organisms with the A type in a population of size N, we have derived probabilities for various possible frequencies of A in the following generation. That is, for a given number of A's, i, in the parent generation, we have a probability distribution over all possible frequencies of A's in the next generation. Further, we can iterate application of the same reasoning to derive probability distributions for frequencies of A in subsequent generations as well.

Note, however, that the pedagogically useful characterization of the process of producing the next generation as "random sampling" tends to

5. Similar equations are given by Roughgarden (1979, 65), and can be found in discussions of the binomial distribution in probability textbooks such as Grimmett and Welsh 1986, chapter 2. Gillespie (1998, section 2.7) explains the relationship between a similar model and some other simple models of drift.

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hide the fact that the entire calculation is based on assumptions about probabilities. The probability calculus does not create probabilities, but merely allows calculation of probabilities from previously assumed probabilities. In this case, the assumption is that each organism has an equal probability of contributing to the next generation.[6] It is these probabilities which might be objective and causal (as they would be if they were propensities). If so, then the transition probabilities P_{ij} from one population state to another would have the same causal character, as would probabilities of frequencies in subsequent generations. Thus, for example, if the individual level probabilities are propensities, then P_{ij} is the propensity for a population of i A's to produce a population with j A's in the next generation.

Equation (2) does not represent fitness differences—we can use it to model drift but not natural selection. Let's see how the equation (2) can be modified to take into account fitness differences. Suppose that the A and B types differ in fitness, with fitnesses w_A and w_B, respectively (these could be viabilities or expected numbers of offspring). Then the probability of producing an A is not simply equal to the ratio between the number of A's and the size of the population. Instead, we must weight the chance of each type contributing to the next generation by its relative fitnesses. The probability p of one organism in the next generation coming from an A parent, or the probability q of coming from a B parent is then

$$p = \frac{w_A i}{w_T}, \quad q = \frac{w_B(N - i)}{w_T}, \tag{3}$$

where

$$w_T = w_A i + w_B(N - i) \tag{4}$$

is the total fitness in the population. These definitions of p and q in effect capture the proportion of the total fitness in the population which is due to A and B, respectively.[7] (One way to view equations [3] is as describing the proportions of all gametes which are produced by A and B.) In this

6. The assumption that sampling is indiscriminate with respect to a set of heritable traits does not imply that probabilities of choosing one type rather than another are *equal*. Further assumptions would be needed to derive a claim about probabilities.

7. The more common technique of dividing by the mean fitness is appropriate when we use the relative frequency of a type in a population rather than explicitly using the number of instances of a type as we do here.

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case the probability of a population with j A's being produced from a population with i A's is

$$P_{ij} = \binom{N}{j} p^j q^{N-j} = \binom{N}{j} \left(\frac{w_A i}{w_T}\right)^j \left(\frac{w_B(N-i)}{w_T}\right)^{N-j}. \quad (5)$$

This again gives us transition probabilities which concern changes of frequencies in populations. However, here the transition probabilities are derived partly from fitnesses of individual organisms. It remains true, however, that if the fitnesses are defined in terms of objective, causal probabilities, then so are the population-level transition probabilities. Note that in fact equation (2) is just a limiting case of equation (5) when fitnesses w_A and w_B are equal. The probabilities p and q in (3) then reduce to (1).

The equations above assumed that the fitnesses of different organisms are probabilistically independent of each other, but that is often unrealistic. More complicated mathematics are needed to deal with frequency dependent and density dependent fitness, diploidy, cross-generational effects due to nongenetic parental influence or niche construction, nonrandom mating, and other factors such as migration. In some contexts population size might have to be replaced by a measure of effective population size. Nevertheless, it seems reasonable to hold that if the appropriate individual-level probabilities exist, then population-level probabilities derive from them and inherit properties such as, for example, being dispositional.

Suppose that all of the probabilities in equations (2) or (5) are propensities concerning the fates of individual organisms. These probabilities are then indeterministic dispositions concerning those fates. I will not try to resolve controversies about what "disposition" means here. What is important here is that if we begin with propensities, then the population-level probabilities P_{ij} are simply the original, population-level propensities, combined and massaged into population-level propensities. The probabilities P_{ij} are simply the result of applying the probability calculus to a collection of propensities concerning fates of individuals. But the probability calculus does not perform metaphysical transformations; it simply gives us probabilities of the same ontological character as the ones with which we began. Thus, if the individual-level probabilities are propensities, the probabilities P_{ij} are as well. Hence the probability distribution over future frequencies constituted by the set of such population-level probabilities would have exactly the same dispositional, causal character as the individual-level propensities.

4. Drift and Selection. If random drift is anything, it is not one thing. There seem to be variety of processes, effects, forces, anomalies, aspects of models, and so on, all called "drift" by one biologist or another in different

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contexts (Beatty 1992). The term applies to many effects on populations or organisms which are said to be due to "chance," and to factors which are thought to help to produce such effects. However, many core senses of random drift make it something which varies inversely with population size.

I want to suggest that for many uses of "drift," the kind of relationship that often obtains between individual-level causal factors and drift is exemplified by the individual-to-population relationships described above. Indeed, equation (2) is nearly identical to an equation that Roughgarden (1979, 65) uses to characterize drift. Equations like (2) are particularly useful illustrations of how drift can arise because in such equations the probabilities P_{ij} for transitions between populations with various frequencies are sensitive to only one parameter: the population size N. When N is small, the probability that frequencies in the population will go to fixation after a small number of generations is high (so drift is considered a strong force); when N is large, the same probabilities are small (so drift is considered a weak force; see Roughgarden 1979, chapter 5).

However, equation (5) exhibits the same sensitivity to population size. The difference is that in this case the probabilities of future frequencies are also affected by the individual fitnesses w_A and w_B. Now, as I explained above, if the individual-level probabilities are objective and causal, then so are the population-level probabilities derived from them. This means that in the simple kind of case characterized by (5), we have population-level probabilities that are determined by two kinds of factors: population size and relative fitness. However, fitness differences constitute the force of natural selection. Thus, if the probabilities in equation (5) are causal, we have two evolutionary forces simultaneously affecting evolution.

The sense in which natural selection and drift causally interact is this: Both natural selection and drift are in part embodied in the same set of probability distributions over future frequencies in a population. However, selection is the aspect of the distribution controlled by differences in fitness, while drift is the aspect of such a distribution controlled by population size (apart from any effects of population size on fitness). Of course there are not cleanly separable "aspects" to the probability distributions over future frequencies. (Cartwright (1983) gives an analogous example in which different causal factors combine nonlinearly to affect gradients of chemicals in a liquid.) Nevertheless, there are systematic differences in the ways in which changing population size and changing fitness differences affect future frequencies. For example, if w_A is greater than w_B, the distributions over future population states are distorted so as to favor higher frequencies of A and ultimate fixation of A. When N is smaller, the distributions are distorted so as to favor relatively rapid fixation of either A or B without regard to their difference in fitness. Figures 1a and 1b contain four graphs giving the probability (y-axis, same scale) of fre-

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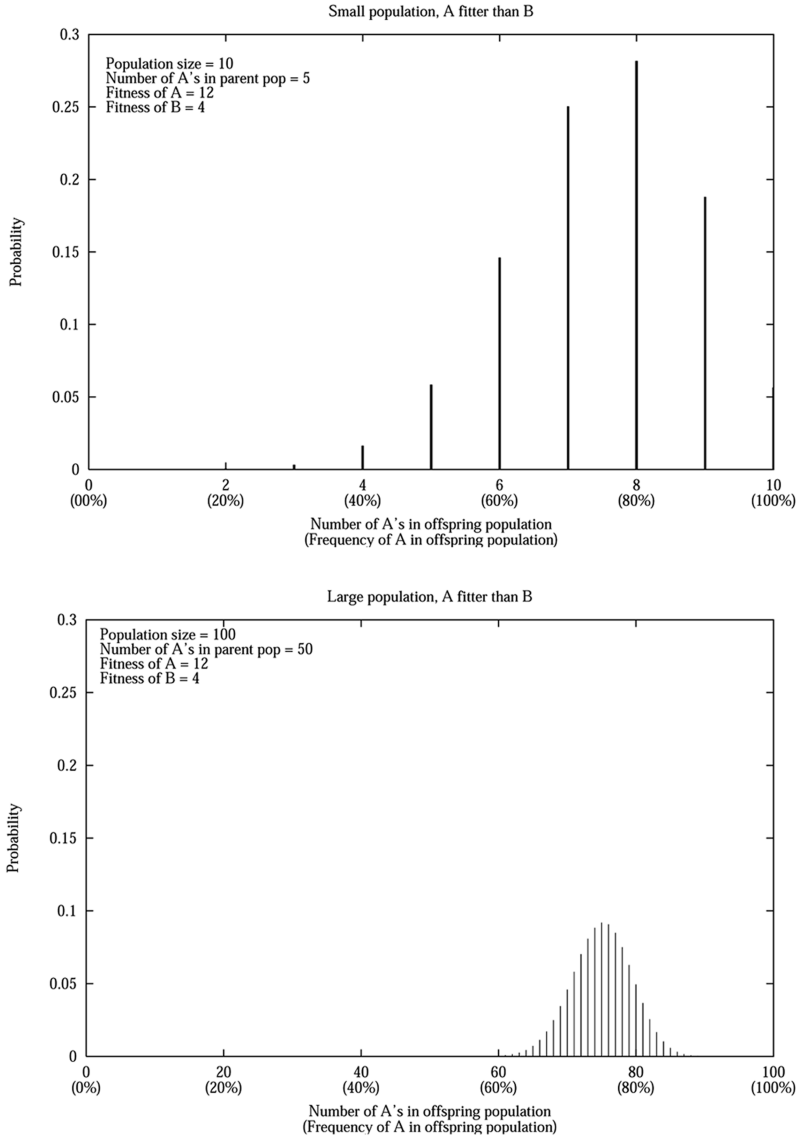


Figure 1a.—Variation in both fitness and population size.

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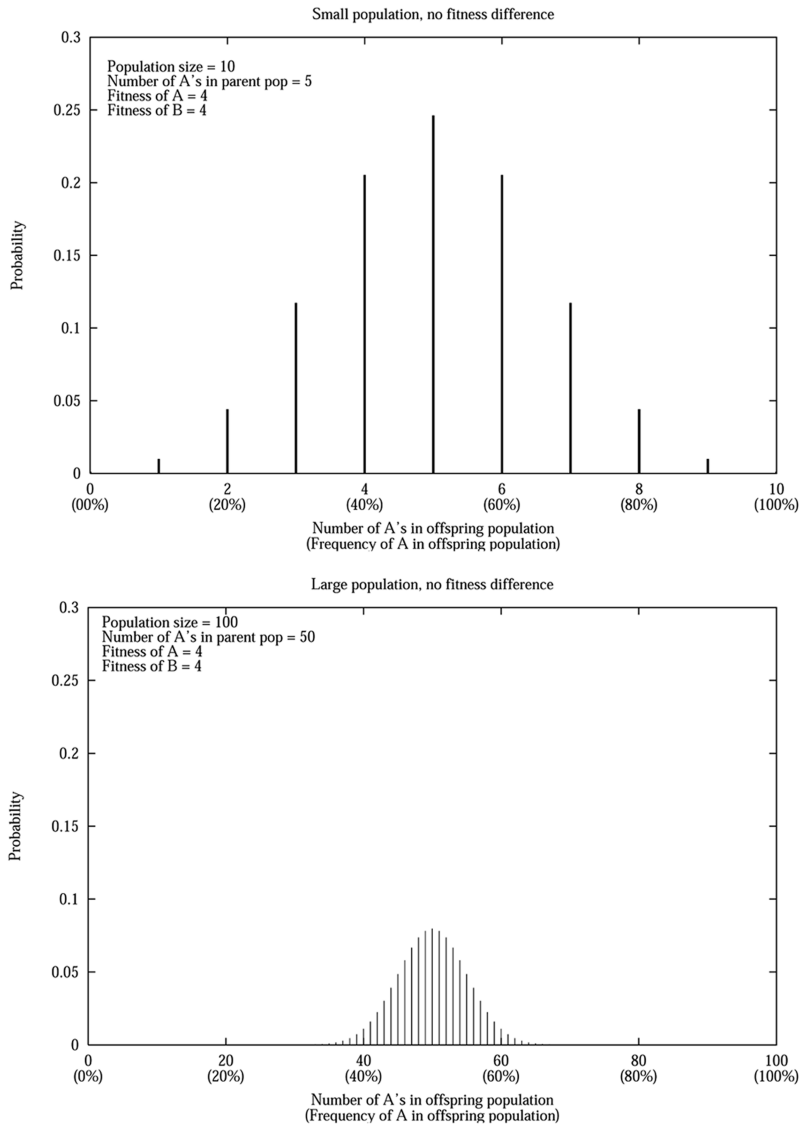


Figure 1b.—Variation in both fitness and population size.

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quencies (x-axis) in the next generation. Notice that in the smaller population (top row, $N = 10$), frequencies are more spread out than in a larger population (bottom, $N = 100$), and that when fitnesses are different (right column, $w_A = 3 \times w_B$), the curve is shifted relative to cases in which fitnesses are equal (left column).

5. Conclusion. I have argued that population-level probabilities can be derived from individual-level probabilities, and that the former are causal in whatever sense the latter are. Thus, for example, if individual-level probabilities are propensities and propensities are dispositions, then population-level probabilities are causal in whatever sense dispositions are causal. I also argued that natural selection and one sort of random drift can correspond to different aspects of such a population-level probability distribution. Natural selection is the aspect of the distribution which is under the control of fitness differences, and drift is the aspect of the distribution which is under the control of population size (or perhaps effective population size). If the probabilities involved are causal, then changes in the probability distribution are changes in causal probabilities of future frequencies in the population. Exactly how we view the causal roles of selection and drift in producing future frequencies will depend on what sort of causal character the probabilities have. For example, if they are dispositional, then the relation of selection and drift to actual frequencies will be the relation of dispositions to their manifestations. We therefore have a framework in which natural selection and drift can count as causal factors, "forces" if you will. Moreover, it is now clear just what their causal character will turn on.

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