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What Fitness Can't Be

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Abstract Recently advocates of the propensity interpretation of fitness have turned critics. To accommodate examples from the population genetics literature they conclude that fitness is better defined broadly as a family of propensities rather than the propensity to contribute descendants to some future generation. We argue that the propensity theorists have misunderstood the deeper ramifications of the examples they cite. These examples demonstrate why there are factors outside of propensities that determine fitness. We go on to argue for the more general thesis that no account of fitness can satisfy the desiderata that have motivated the propensity account.

1 Introduction

According to advocates of the propensity interpretation of evolutionary fitness, fitness has 'two faces' (Sober 1993)—it is both a descriptor of an organism's propensity to reproduce in their local environment and a mathematical predictor of a type's expected reproductive success. Recently, propensity interpretation advocates have noticed that certain evolutionary dynamics count against their mathematical account of fitness as expected number of offspring (Beatty and Finsen 1998; Beatty 1998; Sober 2000). Yet, they maintain that the ecological description of fitness as an individual's propensity is unaffected by these evolutionary dynamics.

We argue that the advocates of the propensity account of fitness fail to notice that these evolutionary dynamics are, in fact, counter examples to the propensity interpretation of fitness. In the first section, we identify three desiderata for an

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account of fitness that propensity theorists accept. In the second section, we review standard arguments in favor of the propensity interpretation of fitness; in particular, we focus on arguments that seek to motivate the propensity interpretation by alleging that it meets the three desiderata. In the third section, we give an informal review of a phenomenon that was first noted by John Gillespie (1973, 1974, 1977) in which the spread of a trait depends upon statistical facts that are independent of any individual's 'fitness', where 'fitness' is understood according to the propensity interpretation. In section four, we diagnose this case, identifying why it provides a powerful argument against the propensity interpretation. Finally, we argue for a more general thesis of wider importance—that once we see why the propensity interpretation of fitness fails, it is clear that no account can meet the three desiderata.

2 Three Desiderata For an Account of Fitness

It should be uncontroversial that the fitness concept must be reconstructed so that it can play its traditional role in evolutionary explanation. To a first approximation, we employ the concept of fitness when we want to explain why a trait spreads through a population when it does. In other words, when we want to know why a trait is better represented in a population than another trait, the typical explanatory strategy is to identify what features make organisms with the better-represented trait fitter than those with alternative traits. Let us put the point as follows:

(A) A fitness concept must be able to explain why one trait is expected to be better represented in a population under the influence of natural selection.

To take a simple example, we might ask why zebras tend to be fast rather than slow—that is, why the trait of being fast is better represented in a zebra population than the trait of being slow. A fitness concept must be capable of being pressed into service for the explanation that answers this question. That is, we must be able to say that fast zebras are 'fitter' than slow ones, and that natural selection has thereby favored the spread of the fitter trait.

This is clearly a contrastive explanation. To explain why one trait has spread in a population is to contrast it with other, alternative, traits. And obviously, if the fitness concept is to play its role in such contrastive explanations, then it must be possible to compare traits by ordering them according to their fitness. The fitness concept, therefore, must be capable of providing a metric that allows us to say, for any two alternative traits, that one is fitter than the other, or that they are of equal fitness. This is our second adequacy constraint on the fitness concept:

(B) A fitness concept must enable us to compare the degree to which natural selection will favor the spread of one trait over another, alternative trait.

So far, we have spoken of the fitness of traits and the spread of traits throughout a population. But explanations of the spread of traits in the population should be, according to the propensity theorists, grounded in simple, metaphysically unproblematic events in the lives and deaths of individuals in the population. By adhering to this principle, we at least hold out hope that our fitness concept will play a role in

causal explanations of the statistical properties of a population. Let us put this point as follows:

(C) The fitness of a trait must be a function of the properties of individual members of the population within their local environmental conditions.

The ever-popular propensity interpretation of fitness aims at fulfilling the three desiderata. Accordingly, propensity refers to 'the particular traits of the organisms in question: specific aspects of its physiology, anatomy, behavior, and so on, all of which contribute to its viability and fertility and ultimately to its overall ability to leave offspring in its particular environment' (Beatty 1998).

3 The Propensity Interpretation

What is slightly more controversial is that the propensity interpretation of fitness meets these criteria. However, we do say that it is only slightly more controversial—for the propensity interpretation has as much of a claim as any to be considered the received view.

An appeal of the propensity interpretation is that it may be understood in close analogy with 'garden variety' dispositions, as Elliott Sober has pointed out (Sober 1993). We say, for example, that salt has the dispositional property of dissolving when placed in water. When we attribute such a dispositional property to salt, we do not allege any metaphysically distinct property to it in addition to its ordinary physical properties. Rather, we just mean that it has a physical structure such that it will dissolve when placed in water.

However, a slightly more complex example is required in order to motivate the propensity interpretation of fitness. For when we say that salt has the dispositional property to dissolve when placed in water, we mean that it always will (under normal conditions). But an object might also have a statistical dispositional property. For example, a fair coin will have the dispositional property of landing heads about one-half of the time. But just as in the case of the salt's solubility, when we say that the coin has a statistical or probabilistic disposition, we are not alleging anything metaphysically distinct from its physical properties. For the coin's disposition to land on heads one-half of the time supervenes on its physical structure, including its being shaped symmetrically, and its having its weight evenly distributed.

The propensity interpretation of fitness says that the fitness of an organism is its expected number of offspring. Like a coin's expected proportion of tosses landing on heads, the propensity to have a certain number of offspring is understood as a dispositional property that supervenes on the organism's physical characteristics. So it is not metaphysically mysterious.

Of course, the lives of individual organisms will be filled with unforeseeable events that will appear random to any observer, and these random events will often cause the individual to have a larger or smaller number of offspring than its mathematical expectation. Thus, in order to talk meaningfully about the expected number of offspring, and use this talk in explanations, we need to assume that the

noise of these random events will be relatively unimportant. Of course, the best way to argue that this is the case is to invoke the law of large numbers. As a population increases in size, the importance of such noisy events diminishes correspondingly.

So far, we have spoken of the fitness of individuals in a population, and not of traits, and one might worry that this talk cannot be carried over into an explanation of the spread of traits. But the advocate of the propensity interpretation has a ready response to this worry. The fitness of a trait is simply the expected number of offspring of an individual in the population, given that the individual has the trait. So to return to our simple example, the fitness of the trait of being a fast zebra is simply the expected number of offspring of an individual zebra, given that the individual is fast. Thus, Sober remarks that the choice of whether to talk about the fitness of an organism or the fitness of a trait is merely a 'harmless stylistic convenience' (Sober 2000).

At this point, we can clearly see how the propensity interpretation is supposed to meet the three criteria. Fitness—understood as propensity to have a particular number of offspring—is to satisfy condition (A) by showing that the expected number of offspring of an individual with a certain trait increases or decreases because of the presence of that trait. Because the propensity interpretation equates the fitness of an organism with a particular number that can be greater or lower than another, every trait's fitness can be compared with others, satisfying condition (B). Lastly, because the fitness of a trait is simply the fitness of individuals with that trait, we are also guaranteed that condition (C) is satisfied.

4 Variance as Influencing the Spread of Traits

A number of biologists have noted that statistical features of a population may be capable of influencing the spread of traits throughout that population. A particularly clear, and yet surprising, example was first discussed in detail by John Gillespie (1973, 1974, 1977). Here we focus on Gillespie's discussion, not only because it is so clear, but also because similar examples have appeared in discussions by advocates of the propensity interpretation. Thus, it is important to diagnose the relevance of these cases. Of course, we shall argue that the importance of these cases has not been adequately appreciated by other authors Ariew and Lewontin (2004) Michael Strevens (unpublished manuscript) are notable exceptions.

A number of population geneticists have demonstrated why fitness should not be defined as expected value of offspring, especially when there is stochastic variation in viability or fertility. Gillespie, for instance, demonstrates cases in which the type with lower variance, not the one with the higher expected number of offspring, will increase in representation. To illustrate, consider Sober's example (Sober 2000). Suppose Type *X* produces two offspring every generation (generations are discrete, reproduction is asexual). Type *Y* will either produce one or three offspring with probability one-half. The mathematical expectation for the number of offspring is the arithmetic average of the number of offspring each is expected to produce. Obviously, for both types, this number is two. But this does not imply that Type *X* and Type *Y* will be equally represented in future generations. For suppose we start

with two members of each type. In the next generation there will be four Type *X* individuals and either two or six Type *Y* individuals. While the expected number of offspring for each type is equal, the expected frequency of each type differs in the next generation. To see this, suppose that a population starts with one individual of each type. There is a probability of one-half that there will be three individuals in the population in the next generation, and a probability of one-half that there will be five individuals in the population. Thus, the expected frequencies of the two types are as follows:

$$\text{Freq}(X) = \frac{1}{2}\left(\frac{2}{3}\right) + \frac{1}{2}\left(\frac{2}{5}\right) = \frac{8}{15}$$

$$\text{Freq}(Y) = \frac{1}{2}\left(\frac{1}{3}\right) + \frac{1}{2}\left(\frac{3}{5}\right) = \frac{7}{15}$$

On the face of it, we might expect a propensity theorist to be concerned with such phenomena. After all, the example is stipulated so that the Type *X* and Type *Y* individuals have exactly the same expected number of offspring. So there is no fact about their fitness (understood as a propensity to have a particular number of offspring) that could explain why Type *X* individuals will do better than Type *Y* individuals. In other words, the Gillespie case demonstrates a tension between conditions A and C, since the properties of the individual members in their local environmental conditions do not explain why Type *X* is expected to be better represented in future generations (according to condition C). In this case what does the explaining is differential variance, a factor outside of the propensity of individuals to survive and reproduce in their local environmental condition.

5 Diagnosis of the Case

Interestingly, examples such as Gillespie's have not particularly worried propensity theorists. In this section, we shall survey responses that propensity theorists have offered. We shall argue that these responses miss the force of the cases.

5.1 Fitness is a 'Family of Propensities'

Beatty and Finsen (1998) proposes to modify the account by significantly broadening the propensities that figure into an organism's fitness. Rather than construing fitness as a propensity to produce a particular number of offspring, they propose that fitness should be defined as a 'family' of propensities, including reproductive scheduling abilities, summarized by a variety of statistical parameters (see also Beatty 1998; Rosenberg 2006; Bouchard and Rosenberg 2004; Brandon 1990). Of course, different statistical parameters will be appropriate under different circumstances. Nevertheless, at bottom, fitness remains a propensity. Thus, Beatty and Finsen hold out the possibility of upholding all three desiderata.

However, there are at least two reasons why Beatty and Finsen's response will not work. First, we must note that Beatty and Finsen acknowledge that different propensities will affect a population differently under different circumstances. So

for example, suppose we consider a population in which the individuals vary as to their reproductive schedules. As Gillespie shows, this fact will tend to affect the growth of the population to a greater degree if the initial size of the population is small—for in such a case, the variation as to their reproductive schedules will entail a higher statistical variance across the population. But if the population is very large, then it is less important that the individuals have different reproductive schedules.

For our purposes here, this fact is important because it lowers the explanatory value of the fitness concept. If we follow Beatty and Finsen's advice in construing fitness as a family of propensities that affect population growth, then we will have to include different propensities under the concept of fitness in different circumstances. But what the fitness concept is supposed to provide is a set of propensities that we can look to in helping us to understand the spread of traits across various populations (thus satisfying condition A via condition C). But if different propensities are to be included under the heading of 'fitness' under different circumstances, then in order to deploy the fitness concept in the first place, we must already understand which factors are contributing to the spread of traits before we can even determine which fitness concept is applicable. And if so, then the fitness concept will obviously be of no use in helping us to understand and explain the explananda it is supposed to. We thereby aver that the 'family of propensity' response does not succeed.

The second reason why it fails points to a tension between conditions A and C: the propensity interpretation is committed to explaining the spread of a trait by recourse to the causally efficacious properties of the individuals in the population. However, the relevant features of the population that causes the type with lower variance to have a higher frequency are not causally efficacious features of the individuals at all.

The crucial fact that brings out the failure of Beatty and Finsen's response is that the population size itself—and not any particular causal interaction—is what explains why the type with lower variance is expected to have the higher frequency. This is because the importance of variance rapidly diminishes as the population size increases. The easiest way to see this is by considering the limiting case in which the population size is effectively infinite. In this case, about half of the Type *Y* individuals will have one offspring, while the other half have three. Thus, the expected number of Type *Y* individuals in the next generation will be the same as the expected number of Type *X* individuals. But if there is only a single Type *X* individual and a single Type *Y* individual, then there cannot be the same number of Type *X* and Type *Y* individuals in the next generation—the expected frequency of Type *X* is higher in this case.

It is important to note that nothing in the previous argument depends upon the presence of any causal interactions among members of the population at all. So suppose that a population size is increased by adding members who do not causally interact with the existing members of the population at all. In such a case, the importance of variance is decreased, but (by hypothesis) there are no new causal interactions that account for this fact (Sober 2000). Thus, if the propensity interpretation is motivated by the desire to provide causal explanations (satisfying

condition C), then we cannot simply include such a large family of propensities under the concept of fitness.

5.2 The 'Why Worry?' Response

Perhaps one might resist the conclusion that this sort of case poses a serious worry for the propensity interpretation. Consider a simple and unproblematic case in which the long-term behavior of a system diverges from what one might expect from the individual propensities. Suppose, for example, that we flip a fair coin ten times. Although the coin has a propensity to land on heads one-half of the time, we would not be surprised to find that the coin's behavior diverges from that prediction over a series of ten tosses. Obviously, the coin is subject to a large number of influences that may affect its behavior in unpredictable ways. As Sober puts the point, 'just as a coin's probability may fail to coincide exactly with the actual frequency of heads in a run of tosses, so an organism's fitness need not coincide exactly with the actual number of offspring it produces' (Sober 2000).

The point is that we should not be too concerned with deviations from the individual propensities because external factors may cause the system's actual causal trajectory to differ from any mathematical prediction. However, this response is a *non sequitur*, for the following reason. When a coin deviates from the mathematical prediction, it is because unpredictable causal influences (such as air currents or tremors in the coin-flipping mechanism) make up a background of noise that influences the coin in unpredictable ways. From the standpoint of an observer who is unable to quantify all of those influences, the deviations from the coin's propensity will appear random and unpredictable.

The type of scenario discussed by Gillespie is the inverse of Sober's coin-flipping example. In it, the deviations are causal and predictable (and indeed, can be brought out in an extremely simple model). It is an important point that no background noise needs to be posited, and the variance in reproductive success of Type *Y* individuals need not be explained by random noise at all. For example, suppose that systematically, in every even-numbered generation, all Type *Y* individuals have one offspring, while in all odd-numbered generations, all Type *Y*'s have three offspring. Over the course of many generations, Type *X*'s will have a higher frequency than Type *Y*'s, even though there is no noise whatsoever in this model. Thus, the coin-flipping example does not bear upon the argument.

Sober also rejects the propensity account of fitness, but for a distinct reason (Sober 2000). He argues that in the context of within generation variance of offspring number, fitness is defined as a holistic property that includes both the propensities of organisms in their local environment that affect offspring numbers and a property of the entire population which has no effect on the organism's reproductive behavior. However, it is unsatisfactory to include properties of the entire population for the same reason that Beatty and Finsen's response fails. For different properties of the population will matter to greater or lesser degrees in different circumstances. Thus, we would need to already understand which factors are contributing to the spread of traits throughout a population before we could deploy the fitness concept. So a fitness concept—revised in the way that Sober

suggests—cannot meet its explanatory burden. In this way, Sober's holistic conception of fitness fails to satisfy the explanatory condition A.

5.3 Are These Cases Common Enough to be Important?

One might allege that the Gillespie case—while a technical possibility—is unusual enough that it poses no important difficulty for the propensity interpretation. After all, any explanatory tool—including the fitness concept—will be expected to have only a limited range of application; so the fact that it is possible to concoct examples in which the fitness concept (understood as a propensity) breaks down is neither surprising nor disturbing.

However, it would be a mistake to downplay the importance of the example, for it is neither unusual, contrived, nor a technicality. Indeed, other examples that equally demonstrate a tension between conditions A and C are easy to come by. Ariew and Lewontin (2004) for instance, argue that for species with overlapping generations, the minimum dynamic model for predicting trait frequency changes requires information about whether the population size is increasing or decreasing. Models by Charlesworth and Giesel (1972) and Demetrius (1992) show that if the population as a whole is increasing in size the precocious genotype will increase in frequency relative to the strategy of delaying reproduction; while if the population size as a whole is decreasing, the delayer will increase relative to the precocious. And Maynard Smith's Haystack Model for the evolution of altruism requires that subpopulations in the metapopulation be small; and this is precisely because small populations have a higher variance than larger ones (Maynard Smith 1976).

6 Beyond the Propensity Account

A stronger conclusion is emerging concerning whether it is possible to offer an account of fitness that satisfies conditions A–C. But in particular, condition C is problematic, because facts such as population size—which is not a causal property of an individual at all—may influence whether a trait's frequency will increase or decrease. Our critique of the propensity account is thus reinforced while the scope of the critique is extended to any attempt to satisfy all three desiderata. For a large fraction of organisms, the explanation for why a trait will increase or decrease depends in part on factors that are extrinsic to the causal properties of individual. We are forced to conclude that an account of fitness jointly satisfying all three desiderata is not possible.

Yet, it is possible to stake out a position according to which we ought to abandon the propensity interpretation of fitness, without abandoning all attempts to provide a causal and individualistic account of fitness. Such a moderate position is advocated by Alexander Rosenberg. While accepting the conclusion that the propensity account is untenable (Rosenberg 2006, p. 176). Rosenberg resists the stronger conclusion that a general account of fitness is non-causal. He argues that there is something in common that 'explains and unifies' every evolutionary dynamic that features one type becoming better represented than other types in the population.

That commonality simply is that in every case, the properties of individuals in relation to their local environments contributes to the success of its lineage. Rosenberg calls the suite of properties that are relevant to the success of the lineage, 'ecological fitness'.

However, Rosenberg's 'ecological fitness' is neither necessary nor sufficient for an adequate account of fitness that satisfies conditions A–C. In our discussion of Beatty and Finsen, we have enough to demonstrate why 'ecological fitness' is not sufficient. That is, even in cases where a type's ecological fitness plays some role, as in the case of within-generation variance, it is important to note that what explains trait spread is the whole model, which includes factors outside of individual properties. Hence, pointing out properties of organisms in relation to their local environmental condition is *part* of the explanation is a *non sequitur* because the issue at hand is what sufficiently explains differential spread of trait types.

As for necessity, we note that in the Gillespie case Type *X* and *Y* have identical ecological fitness. The point of the example is to show that the long term expected relative frequency of the two types is not influenced by their ecological fitness.

7 The Impossibility of Jointly Satisfying Conditions A and B

Suppose in light of the considerations above, including the lessons learned about Gillespie cases, we abandon condition C as an adequate account of fitness and assert that only conditions A and B really matter. That is, accept merely that an adequate account of fitness both: (A) explains why one type is expected to have a higher frequency than the others; and (B) explains by invoking a comparison between types. Such an account of fitness need not uphold the Darwinian ideal that fitnesses reflect natural properties of individuals at all.

According to Ariew and Lewontin (2004), with the rediscovery of the Mendelian principles of inheritance and the development of knowledge of the chromosomal basis for the statistical properties of inheritance patterns, it became apparent that the Darwinian conception of evolution was incomplete, especially in its reliance on a simple resemblance of parent to offspring. As a result evolutionists including Fisher, Haldane and Wright developed genetical theories of evolution by natural selection, theories that make essentially no reference to the properties of individuals and their relation to their environmental conditions. That is, theories where Darwin's concept of fitness plays no role. Accordingly, changes in representation of types is explained by citing differential reproductive rates, or a scalar value.

Contrary to the set of conditions we have outlined at the outset, modern evolutionary genetics does not rely on condition C at all. In fact, the motivation for the genetic theory of evolution is the failure of the Darwinian scheme to explain and predict the direction of change of a population solely from the natural properties of organisms and their environments. The failure is the result of the extra considerations required to account for differential type frequencies which are the consequence of mechanisms of inheritance and details of reproductive schedules. That is to say, the forerunners of modern population genetics had already realized that an adequate account of fitness that includes condition C is not feasible. They

abandoned any such attempts and replaced a 'Darwinian' conception of fitness with a ordinal scalar that satisfies conditions A and B.

Ariew and Lewontin (2004, p. 348) call this ordered scalar a 'reproductive rate'. Yet, despite lowering the bar, as it were, for an adequate account of fitness in modern evolutionary genetics, they argue that no single unified account of fitness that satisfies conditions A and B can be found: 'any attempt to introduce a unitary analogous concept of 'reproductive fitness' into dynamical models as a scalar ordinal, which will explain or predict quantitative changes in the frequency of types, must fail'. Rosenberg seems to concur. He writes, 'there is no single conception of fitness that applies to all populations dynamics where one type outreproduces another' (Rosenberg 2006).

The reason for the failure is that nature is too variegated. Different biological situations call for different algorithms to explain changes in trait frequencies. To see this, consider a very simple concept of fitness. Viability, or the probability of a survival of a genotype from egg to adult is an adequate account of fitness for cases in which there are no differences in fertility among genotypes and that the genotypic viability differences are independent of the relative frequencies of the genotypes. Such an account of fitness has been fruitful for evolutionary theory and has been incorporated by pioneers such as Wright, Haldane, and Fisher. Yet, it fails to adequately explain relative genotypic differences in the vast number of cases where genotypic viability differences are dependent of relative frequencies of the genotypes. In the so-called 'frequency-dependence' cases there is no single value that can be invoked to account for differential frequencies for genotypes in all populations (e.g. the famous case of butterfly mimicry is often cited as an example of frequency-dependent selection).

Suppose, apropos the frequency-dependence case, one defines fitness as a *set* of functions of genotypic frequencies (rather than a single value) in order to provide as general a concept of fitness as possible to satisfy conditions A and B. As it turns out, our new set of fitness functions is insufficient for a different set of conditions, for instance when the assumption of no variation in fertility is violated. Such a model needs to take into account not only genotypic frequencies but also information about reproductive schedules. It is easy to come up with new, biologically real factors that will affect the frequency of trait types. For example, Wright's treatment of the effect of population structure on gene frequency provides a continuum of different models in which additional information is required in order to predict the frequency of gene types (Wright 1943, 1945, 1969).

Because it is always possible to discover new factors that would need to be taken into account to salvage the fitness concept, we conclude that no account of fitness respecting conditions A and B can be found.

8 Darwin Versus Fisher

The scope of the argument of this essay concerns whether it is possible to offer an account of fitness that satisfies the three desiderata A–C. We have entitled the account the "propensity account of fitness" because desiderata A–C most closely

resembles the accounts provided by Beatty (1998), Beatty and Finsen (1998), Sober (1993, 2000). We have applied the work of John Gillespie (1973, 1974, 1977) to show that it is not possible to provide an account of fitness that satisfies desiderata A–C. We further considered attempts to save the "propensity account" by identifying fitness as a "family of propensities" including those summarized by statistical measures of the population Beatty and Finsen (1998), Brandon (1990), Bouchard and Rosenberg (2004), Rosenberg (2006). We showed that these attempts are not feasible either.

Whether it is possible to provide an account of fitness that references "propensities" yet rejects any one of the desiderata A–C is not discussed in this essay. However, it is worth making some general considerations about potential attempts.

First, we view our results as consistent with the more general claim that an account that identifies all probabilities as propensities is inadequate (Salmon 1984; Humphreys 1985). The reason why a propensity account of probabilities is inadequate is that some probability statements describe relations that are not mere cause and effect. Sober's example is the probability that an individual's parents were heterozygotes given that the individual itself is a heterozygote. That probability does not refer to a cause and effect relation. Offspring genotypes do not cause the genotypes of parents. Our example is Gillespie's whereby the probability statement ranges over variance and is affected by population size, not the causal properties of individuals.

Second, suppose we try to save the propensity account of fitness from our criticisms by identifying fitness not as individual propensities but population level properties to reflect the results of the Gillespie cases (Brandon 1990; Millstein 2006, pp. 627–653—though Millstein never mentions the word "propensity"). Under this proposal we would identify fitnesses as statistical properties of the population including variance and population size. Such an account would be a victory for the propensity theorist only by name since it amounts to rejecting desiderata C. It is akin to responding to the argument from evil by redefining "God" to allow for the existence of evil.

Finally, some readers might note that the results of this essay are consistent with views espoused by Walsh et al. (2002) and Matthen and Ariew (2002, 2005). The scope of these other papers is much broader than the present essay. The former concern more general questions about *natural selection*, including arguments in favor of viewing the relation between natural selection and drift from a probabilistic point of view rather than as a sum of forces. The present essay is of much narrower scope, concerning *fitness* which is a *component* of natural selection. Much to the chagrin of one of the authors of the present essay it is possible to accept the specific conclusion of this essay concerning the propensity account of fitness without accepting the more general conclusions espoused in the other papers mentioned.

9 Conclusion

Bouchard and Rosenberg claims that the attempt to expunge ecological fitness from the theory of natural selection 'makes the theory unrecognizable' (Bouchard and

Rosenberg 2004; Rosenberg 2006). Accordingly, ecological fitness is indispensable to the theory of natural selection. If by 'the theory of natural selection' Bouchard and Rosenberg is referring to Darwin's version, then they are right. Yet, Darwin's assumption that ecological fitness explains changes in trait frequency in all cases is false. That Darwin was mistaken in his theory of changes in trait frequency should not come as a surprise. The limitations of ecological fitness explanations have been known since the first quarter of the 20th century, when it became apparent that Darwin's assumption of simple resemblance of parents to offspring is mistaken. The result was the development (by Fisher, Haldane, and Wright, among others) of a genetical and statistical theory of natural selection in which Darwin's fitness was replaced by a reproductive rate. Fisher's version of his genetical theory of natural selection (from which he grounds his 'fundamental theorem of natural selection') relies on a definition of fitness that he calls the 'Malthusian parameter'. This amounts to the per capita rate of increase of a type.

Since the increase of a type might be due to factors that have nothing to do with a type's ecological fitness and all to do with differential reproductive rates, Fisher's 'Malthusian parameter' is not the same as Darwin's fitness. If we take Rosenberg's commitment to a Darwinian concept of fitness its natural conclusion, then we would be forced to rule out Fisher's version of natural selection as 'unrecognizable'. But it is important to note that the fault lies not in Fisher but in Darwin, for it was Darwin who mistakenly thought that evolution of traits are due to differential ecological fitness.

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