A critical review of the statisticalist debate

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Abstract

The statisticalist interpretation of evolutionary theory construes the modern mathematical genetics as a purely phenomenological theory that explains evolutionary changes by statistical, but not causal, features of populations. The view has provoked heated discussions over the past decade, prompting numerous philosophical analyses from various perspectives but at the same time making it difficult to draw a clear picture of the controversy. In view of evaluating these analyses and attaining a correct understanding of evolutionary theory, this article reviews the debate by breaking it down to three aspects, respectively focusing on the assumptions, applications, and explanations of evolutionary theory. Under each rubric the claims made by statisticalists and their opponents are assessed with a view to arriving at a definite conclusion. In so doing the article will also ask why the debate got so prolonged and intricate, trying to identify a part the reason in an assumption that has been shared, often implicitly, by both sides of the controversy.

1   Introduction

Understanding the nature of evolutionary theory is one of the central goals of the philosophy of biology. According to the traditional account, evolutionary theory explains changes of a population based on various causal factors including environmental conditions and reproductive mechanisms. From the beginning of this century, however, this view has come under criticism by a group of philosophers, called *statisticalists*, who claim evolutionary changes to be purely statistical phenomena accounted for by statistical, but not causal, features of populations (e.g. Matthen and Ariew, 2002; Walsh et al., 2002). It follows population genetics, the mathematical core of modern

evolutionary theory, is not concerned with causes of evolution, but studies evolution as abstract relationships among various statistics estimated by census. The statisticalists' claim has provoked a number of critical responses by opponent *causalists* who espouse the traditional, causal account of evolutionary theory. The purpose of the present article is to introduce and critically review some major issues discussed in this debate, with a view to arriving at a definite conclusion in each contention.

During the decade of (sometime heated) exchanges, the number of relevant articles has grown and the debate ramified to cover various topics including, to name a few, the interpretation of fitness, the conceptual distinction between selection and drift, the possibility of the population-level causation, and metaphorical representations of evolutionary theory. This review does not aim to cover all these contributions or topics. In particular, I do not address the question as to whether evolutionary theory should be understood as a theory of *force* (Sober, 1984), the major factors of evolution such as selection and drift being akin to Newtonian forces. Although a criticism to this metaphorical understanding was among the early statisticalist arguments to support their non-causal view of evolutionary theory, as some authors have pointed out (Stephens, 2004; Lewens, 2004) these two issues are logically independent and can be examined separately. This review concentrates on the problem of the causal nature of evolutionary theory, and address the above listed issues only to the extent they are related to this main focus.

But what does it mean for a scientific theory to be causal? This is already a non-trivial question, and the lack of an explicit consensus on this regard has posed an obstacle to understand or assess the claims made by either side. Prior to the review, therefore, we should be clear on in what sense evolu-

tionary theory, or any scientific theory in general, is claimed to be causal or non-causal by the participants of the debate. First, evolutionary theory has been said to be causal (or non-causal) for it involves explicit (no) causal *assumptions*. According to statisticalists, the core principles of evolutionary theory, or at least of its "formalized" version after the Modern Synthesis, are mathematical theorems that hold regardless of causal details of populations. In response, causalists maintain the derivation of these principles requires some form or another of causal assumptions. Because the nature of a theory, scientific or mathematical, is largely determined by its premises, analyses and interpretations of the assumptions of the evolutionary principles have formed the first contention in the statisticalist-causalist dispute. The second criterion of a "causal theory" related to but nevertheless distinct from the first is whether its *empirical application* requires any causal facts or information about target phenomena or objects. What do we need to know about a population in order to predict its evolutionary trajectory? To be sure, we need some key statistics such as fitness of organisms, but do we also need to know about its causal basis? If so, it will provide a strong evidence for the causal interpretation of evolutionary theory, and vice versa. Finally, a theory may be considered causal if its *explanations* are causal, that is, if they invoke causal relationships or concepts in an essential way. In the causalist picture, the goal of evolutionary theory is to explain population changes by pointing to one or more of their causes, such as fitness variation. This is opposed by statisticalists who deny fitness to be a cause of evolution, and construe explanations in population genetics as subsumptions of a target population under some mathematical theorem.

Obviously these three criteria or standards are not logically independent from each other: if a theory contains some causal assumptions they must be

justified in its empirical application, and for a causal explanation to be valid its (presumably causal) *explanans* surely needs to be true. Clarifying the logical connections among different contentions in the debate is a part of the goal of this review. Nonetheless they represent distinct strategies to argue for either the causal or non-causal interpretation of evolutionary theory, and for this reason the following review is structured according to these three criteria. The statisticalist-causalist dispute over the theoretical assumptions of evolutionary theory is examined in Section 2, followed by the issues on empirical applications (Section 3) and the epistemic status of evolutionary explanations (Section 4).

By setting this agenda I by no means pretend this review to be neutral "view from nowhere." To the contrary, much of the following analyses will be based and developed upon the causal graph approach to evolutionary models I defended elsewhere (Otsuka, in press). The choice of this particular framework reflects my conviction that the problem and controversy at hand are best elucidated when viewed as one concerning the theoretical assumptions and constructions of evolutionary models. This, however, is not necessarily the way the question has been framed by the participants of the debate: rather it has most often been construed as a meta-scientific question over the correct *interpretation* of evolutionary theory or concepts. Such a methodological framing of the issue by itself reveals something about the debate, and in particular is not unrelated to the reason it has been so prolonged. This point will be explicated to some extent in the concluding section to draw a meta-philosophical diagnosis of the debate.

2 Round 1: The theoretical basis of evolutionary theory

2.1 Evolution = mathematical necessity?

Modern evolutionary biology, like many other mature sciences today, is highly mathematized. The Modern Synthesis of the Darwinian theory of evolution and the Mendelian genetics was achieved by, and the subsequent developments of evolutionary theory in the 20th century have centered around, population genetics that studies population changes with mathematical formulae. According to statisticalists, however, population genetics is not only mathematical — it is *a mathematics*. That is, not only does it deal with complex math formulae (after all physics also employs sophisticated mathematics), but rather its principal equations describing evolutionary changes are all mathematical theorems, whose derivation requires nothing more than assumptions or axioms of, say, probability theory. Thus Matthen and Ariew (2009, p.211) assert: "When there are heritable differences in traits leading to differential reproduction rates, the probability of the fitter types increasing in frequency is greater than that of the less-fit types increasing. *This is simply a mathematical truth*"(my emphasis). From this observation they conclude "Mathematical population genetics is, in large measure, an application of probability/frequency theory."

But what are those theorems that are said to govern evolutionary changes? One example featured by Matthen and Ariew (2002) is *Li's theorem* which gives the change in the overall growth rate in terms of the variance of relative growth rates, or $\Delta \bar{W} = \text{Var}(W)/\bar{W}$, where the growth rate W of each type (e.g. allele) in the population represents to what extent it increases or decreases its share in the next generation. Another example is the *Price*

equation $\Delta \bar{Z} = \text{Cov}(Z, W)/\bar{W}$, which expresses the change in the phenotypic mean $\bar{Z}$ in terms of the covariance of phenotype Z and fitness (i.e. the number of offspring) W divided by the mean fitness $\bar{W}$. It is well known that the Price equation is an algebraic truth that holds just in virtue of the axioms of probability theory and the definitions of the mean, covariance, and the variables used therein.[1] The same is true of Li's theorem, which is a special case of the Price equation obtained by substituting phenotype Z in the Price equation for fitness or growth rate W and noting the covariance of a variable with itself is its variance.

If the general principles of evolution are a priori truths, it follows causal assumptions play no substantive role in predicting evolutionary changes — it is just a matter of mathematics. On this ground statisticalists conclude "selection is mathematical in nature, and independent of the particular causal laws that produce growth"(Matthen and Ariew, 2002, p. 74). This is not to deny that each selective episode consists of a host of causal interactions that culminate in individual births and deaths. The claim is rather that mathematical population genetics abstracts away all these causal substrates and studies selection as a purely mathematical relationship that can be described with a priori theorems like Li's theorem. Hence it is concerned exclusively with numerical changes of population frequencies, but not with their causes: "Li's theorem tells us nothing about causes of growth: it is a general truth about growth regardless of how it is caused"(Matthen and Ariew, 2002, p. 74). The "general truth" of evolutionary changes is rather described with statistics such as variance or covariance. This reasoning underlies the core doctrine of the statisticalism that evolutionary theory is not a causal but purely statistical theory — "it explains the changes in the statistical struc-

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[1]See e.g. Okasha (2006) for a derivation of the Price equation.

ture of a population by appeal to statistical phenomena"(Walsh et al., 2002, p. 471).

2.2 Causalist responses

Critics of the statisticalism have challenged this purely mathematical characterization of population genetics in two ways. The first line of response is to assert that statisticalists are looking for a wrong place to read off a causal implication of the theory. Millstein et al. (2009) criticize statisticalists for concluding selection to be non-causal just because it is expressed by some mathematical formula. That something can be represented with an a priori equation does not prove its non-causal nature, for it is not an equation itself but its *interpretation* that gives a causal content. Take as an example a binomial equation $(p + q)^2 = p^2 + 2pq + q^2 = 1$. This same equation can be thought as representing either genotype frequencies at Hardy-Weinberg equilibrium, or the area of a unit square divided into four rectangular subparts (one square with the size p^2, another with q^2, and two rectangles with pq). But the equation itself is silent as to which of these representations is *true*: the representational content of the equation, and thus whether it represents a causal or physical process at all, is determined by its pragmatic context or the intention of the user who applies this equation to a particular object or phenomenon. If so, that evolutionary equations turn out to be mathematical necessities would have no implication for the (non) causal nature of evolutionary theory — it is rather how they are used that counts. On this ground Millstein and colleagues argue that equations of population genetics, or more specifically the binomial representation of drift, have full causal meaning for they have been used by geneticists, most notably Fisher and Wright, to represent a particular class of causal processes which they

call "indiscriminate sampling process."

It is certainly right that the presence or absence of a mathematical expression does not determine the causal or non-causal nature of a given relationship, but why and how can an interpretation accomplish the required job? An interpretation maps a theory — a set of linguistic entities such as mathematical equations like a binomial equation — to a particular domain like a set of squares or sexually reproducing populations. The premise of Millstein et al. (2009) is that this connection "infuses" a theory with the empirical or causal contents of the target domain. This reflects a pragmatic stance toward scientific theories (e.g. van Fraassen, 1980), where a theory is just a tool and by itself does not have any empirical or causal implication. As a consequence, the contention on the nature of evolutionary *theory* is reduced to a metaphysical inquiry of evolutionary *phenomena*. Metaphysical, in the sense such an analysis tries to identify the nature of evolutionary phenomena prior to or independent from a particular scientific theory that deals with these phenomena. It is in this context that Millstein distinguishes *discriminate and indiscriminate sampling processes*, as two types of causal processes that are affected or not by phenotypic differences between organisms (Millstein, 2002, 2005, 2006). This is an ontological distinction made without resorting to any conceptual apparatus of evolutionary theory. Rather evolutionary concepts — selection and drift — are introduced post hoc as representing these two processes, and by this fact, Millstein et al. (2009) argue, they acquire definite and distinct causal meanings.

I will postpone an examination of this distinction between two processes till Section 4.3; here I want to note some issues regarding the *argumentative strategy* taken by Millstein and colleagues. As seen above, their basic strategy is to reduce the theoretical contention to the metaphysical distinc-

tion between two (allegedly causal) processes. One may question, however, whether such a metaphysical investigation alone can help identifying the nature of evolutionary theory, or even phenomena. Let's suppose, for the sake of argument, there really are two distinct processes in nature. To argue they constitute the referents of selection and drift, however, it must be further shown how these processes generate these evolutionary behaviors as quantitatively characterized in population genetics. On this regard Matthen (2010) questions: supposedly indiscriminate sampling is acting in an infinite as well as finite population, but then why does drift manifest only in the latter? Or in general, why does its action depend on the population size at all? To answer these questions one needs to "embed" the alleged processes within population genetics, identifying their place and role in the mathematical equations of evolutionary changes. Until this is complete one cannot conclude these processes to be the real world referents of selection and drift as conceptualized in evolutionary theory.

Moreover, their approach will not convince those statisticalists who think the issue in question is ultimately an epistemological question about the nature of the theory. Ariew and Ernst (2009) and Ariew et al. (2015), for example, distinguishes the modern genetical theory from Darwin's original theory of natural selection, limiting the target of their non-causal claim only to the former while admitting the Darwinian theory to be fully causal. They do not deny, therefore, evolutionary phenomena (a class of phenomena represented by theories of evolution) consist of causal processes: what they deny is that population genetics deals with these causal relationships. Such a position is immune to Millstein et al.'s criticism, for Ariew and his colleagues can fully admit the causal basis of evolutionary phenomena while denying the causal nature of population genetics as a theory, which they claim to

study evolutionary phenomena only after abstracting away all these causal contents.

The above discussions suggest that to fully resolve the dispute one cannot avoid analyzing the theoretical or mathematical structure of evolutionary theory. In this regard Millstein et al. (2009) may concede too much by accepting or at least not questioning the statisticalists' premise that evolutionary principles used in population genetics are of purely mathematical nature. The second line of response challenges this premise. According to Rosenberg and Bouchard (2005), it is a mistake to think that the foundation of evolutionary theory is provided by mathematical formulae such as Li's theorem or Fisher's fundamental theorem of natural selection (FTNS). More fundamental than these equations is the following *principle of natural selection*:

> PNS (x) (y) (E) [If x and y are competing populations and x is fitter than y in E at generation n, then probably, (x's size is larger than y in E at some generation n' later than n)].

Rosenberg and Bouchard then make two claims: (1) the PNS is a causal principle, for what it compares is the *ecological fitness*, the causal capacity of individual organisms to survive and reproduce; (2) the abstract formulae of evolution such as Li's theorem or Fisher's FTNS are all derived from this PNS. Taken together, they conclude that mathematical equations of population genetics, despite their abstract and purely statistical appearance, are in fact based on a causal principle.

I believe this approach to be on the right track, but remains incomplete for two reasons. First, to substantiate this claim Rosenberg and Bouchard must show that the FTNS or Li's theorem is actually derivable from their PNS, and despite their verbal assertion that the derivation is "fairly di-

rect and intuitive" it is far from obvious how a quantitative equation like the FTNS follows from a merely comparative principle like the PNS (see Matthen and Ariew, 2005, we will return to this open question later in Sec. 3.1). Second, even if we grant the derivation is possible it is not clear in what sense the PNS is said to be causal. What exactly is the ecological fitness, and in what sense is it causal? Although we are told that the causal nature of the PNS stems from the concept of ecological fitness, "they do not tell us what this is," as Matthen and Ariew (2005, p. 359) complain.

The task for causalists, therefore, is to *actually* derive evolutionary equations from *explicitly causal* assumptions. Since most evolutionary equations are written in probabilistic forms, this requires one to connect two conceptually different realms, probability and causality. Although this problem has been a source of bewilderment both for philosophers and statisticians over centuries, considerable progress has been made in the past few decades by the *causal graph theory*, which studies formal relationships between a causal structure, expressed by a directed graph, and a probability distribution generated from that structure (Pearl, 2000; Spirtes et al., 2000). Using this theoretical framework and Sewall Wright's *trek rule*, Otsuka (in press, see Box) identified causal models of evolving populations and derived the standard predictive equations of population and quantitative genetics from these causal assumptions. What this means is that these predictive equations including the two-locus population genetics model, the breeder's equation, and the FTNS are all *theorems*, not of probability theory but of the causal graph theory which explicitly models causal relationships. Population genetics, therefore, is not *a mathematics* nor are its equations a priori truths; they are empirical propositions that hold only in virtue of certain causal assumptions.

Box. The causal reconstruction of population genetics

The causal graph theory (Pearl, 2000; Spirtes et al., 2000) studies the formal connection between a probability distribution and a causal structure expressed by a directed graph and structural equations. To derive the standard equations in population/quantitative genetics, Otsuka (in press) defines a causal model for an evolving population based on the following assumptions: (1) a parent's alleles $(X_1, \ldots, X_n)$ affect its phenotype Z, which then contributes to the fitness W defined by the number of its offspring; (2) the parental genes are passed down to offspring, which then affect offspring's phenotype Z'; (3) environmental effects (E_W, E_Z, E'_Z) are independent; (4) all causal relationships are linear. The causal model thus defined (see Fig. 1 below) enables one to rewrite the breeder's equation $\Delta \bar{Z} = Sh^2$, which gives the between-generation response to selection, as a function of the causal parameters and the genetic variance such that:

$$\Delta \bar{Z} = \frac{1}{\bar{W}} \beta \sum_{i,j} \alpha_i \alpha_j \, \mathrm{Cov}(X_i, X_j)$$

where α and β are causal parameters of the structural equations (Fig. 1).

The model can also be used to evaluate intervention effects on evolutionary responses, i.e. $P(\Delta \bar{Z} | do(Y = y))$ where $do(Y = y)$ denotes an intervention that sets the value of Y to y (Pearl, 2000, this is more fully discussed in Sec. 4.3). Although the causal model in Fig. 1 is the simplest possible case, Otsuka (2015) shows that this basic model can be extended to deal with more complex mechanisms such as epigenetic

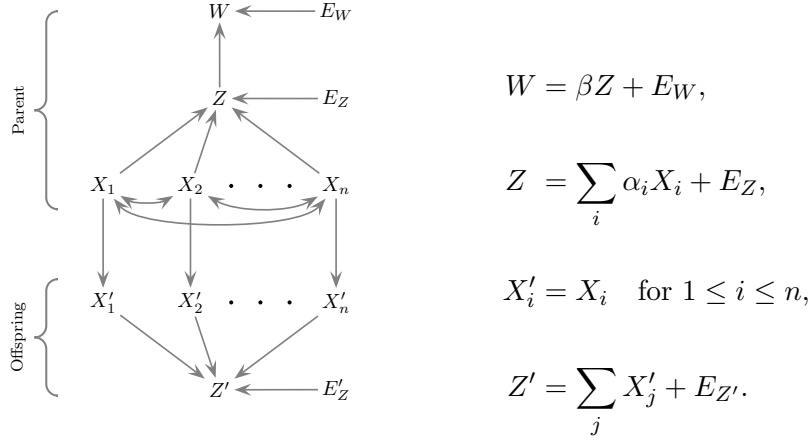inheritance, maternal effects, and niche construction.

$$W = \beta Z + E_W,$$

$$Z = \sum_i \alpha_i X_i + E_Z,$$

$$X_i' = X_i \quad \text{for } 1 \leq i \leq n,$$

$$Z' = \sum_j X_j' + E_{Z'}.$$

Figure 1: A causal model underlying the breeder's equation. For the sake of simplicity the model here does not consider sexual reproduction or mutation. Double-edged arrows in the graph represent statistical dependence, or *linkage disequilibrium*, among parental genes. The structural equations on the right quantitatively specify each causal relationship in the graph.

From a very general perspective, the goal of mathematical genetics can be understood as identifying a function that returns the population change based on some information about the current population structure, such that

evolutionary change $= f($ information about the current population $)$.

The statisticalism asserts the derivation of f in general does not require any more than the theory of probability or statistics. This, however, is not correct. The result given by Otsuka (in press) makes it clear that to obtain such a function we need causal assumptions and the theory that explicitly handles them. In this sense, the causal relationships underlying evolutionary phenomena are far from abstracted away but provide the very basis for the quantitative principles of evolutionary theory.

2.3 Evolutionary principles: explanatory or descriptive?

But, one may wonder, what about the Price equation and Li's theorem? Aren't they mathematical truths? Yes they are, and as such their derivation does not require any causal assumption, as we have seen above. The problem, however, is that they are *not explanatory* at all: they may give a correct *description* of evolutionary changes, but not a *prediction* or *explanation*. This becomes obvious if one takes a moment look at, say, the Price equation $\Delta \bar{Z} = \mathrm{Cov}(Z, W)/\bar{W}$. Suppose you are to "predict" the change in the phenotypic mean, $\Delta \bar{Z}$, by calculating the right hand side. To do so you need to know the fitness W, i.e. the number of offspring, of each individual. But if you know this, and assume perfect heritability, you also know the phenotypic distribution of the offspring generation, and thus the change between two generations. Thus an application of the Price equation for the purpose of "prediction" would presuppose the very information you want to predict with it. In other words, it does not give a function f of the form above, for the right hand side of the Price equation involves information about the *next* generation. No causal assumption in, no prediction out — this is the reason why the Price equation or Li's theorem, being free from any causal assumption but hence devoid of predictive power, is seldom if ever used in empirical studies of adaptive evolution.[2] This is by no means to deny their theoretical importance: no doubt these mathematical identities contribute to our understanding of evolutionary theory. But by themselves they do not provide a prediction or explanation of evolutionary changes, as Price was well aware when he noted that his reformulation is "intended mainly for use in deriving general relations and constructing theories, and

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[2]An empirical application of the Price equation can be found in Morrissey et al. (2012), but it is for the purpose of a post-hoc check of predicted adaptive responses, and not for predicting evolutionary response or detecting a selective pressure.

to clarify understanding of selection phenomena, *rather than for numerical calculation*"(Price, 1972, p. 485, my emphasis).[3]

The false impression that the entire population genetics is based on mathematical theorems stems from this confusion between predictions or explanations on the one hand and mere descriptions on the other. Unfortunately, this is not the first time that the philosophical literature was perplexed by a similar confusion. What I have in mind here is the notorious "charge of tautology," which claims evolutionary theory fails to be an empirical science for its core principle, "survival of the fittest," is a tautology. Although much ink has been spilled on this "problem," only a moment reflection is needed to dismiss it — for the slogan in question has nothing to do with the explanatory structure of the Darwinian theory. At best, "survival of the fittest" is an acceptable (though vainly pedantic) *description* of selection, but does not capture at all the kind of explanations Darwin provided. For one, Darwin's explanadum in his *Origin* was *evolution* but not survival, and "evolution (or spread) of the fittest" is not a tautology at all. Hence I second Birch (2014) that the tautology problem is a pseudo-problem. The same diagnosis applies to the statisticalist conundrum: "how can population genetics be causal, if it is based on a priori principles like Li's theorem or Price's equation?" The short answer is, "no, it is not based on a priori truths." The purely algebraic identities play no significant role in explaining evolution: it is rather causal assumptions about a population under study that enable geneticists to derive predictive equations of its evolutionary trajectories.

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[3]I thank Jim Griesemer for pointing me to this passage of Price's paper.

3 Round 2: Empirical applications of evolutionary theory

Although the causal reconstruction of the predictive equations discussed above highlights the importance of causal assumptions in population genetics, in a strict sense it does *not disprove* the statisticalist claim. For it is still logically possible, even very unlikely, that these or other causal assumptions prove to be dispensable in the derivation. The statisticalism could be vindicated if any of these predictive equations is shown to follow from purely mathematical axioms. Instead of engaging in such a direct proof, however, statisticalists have resorted to indirect arguments that focus on *empirical applications* of the equations: that is, evolutionary theory is argued to be non-causal for its application to an actual population does not require any information about causal features of the population, but only statistical data. Two arguments have been put forward in this line:

1. Causal analyses of the survival or reproductive capacity of organisms are at best comparative by nature and cannot yield the quantitative measure of fitness as used in population genetics (Matthen and Ariew, 2002, 2009; Pigliucci and Kaplan, 2006).

2. The causal features of a population are irrelevant in predicting its evolutionary change. Applications of evolutionary equations require only statistical information (Matthen and Ariew, 2002; Ariew and Ernst, 2009; Ariew et al., 2015).

If a theory makes some causal assumptions they must be confirmed before its application. Then by contraposition a successful defense of either of the these claims would establish the non-causal nature of evolutionary theory.

Among all concepts in evolutionary biology, philosophers — statistical-
ists and causalists alike — have paid almost exclusive attention to *fitness*,
primary because of its assumed centrality in evolutionary explanations. In
this context, the above two claims respectively assert the (1) impossibil-
ity and (2) dispensability of a causal analysis of fitness in applications of
evolutionary theory. We will examine these claims in turn.

3.1   The formal and vernacular notions of fitness

As noted in the introduction the statisticalism emerged as an antithesis
to the traditional meta-scientific account of evolutionary theory. Much of
this account is due to Elliott Sober's seminal book, *Nature of Selection*
(1984). One of the major topics in this book and the one that followed
(Sober, 1993) was the aforementioned charge of tautology, and to defend
the empirical nature of evolutionary theory Sober distinguished two kinds
of laws operative in evolutionary theory. The ones are the "consequence
laws" that are represented by mathematical formulae of population genetics
and calculate population changes with some quantitative parameters; while
the others are the "source laws" that estimate these parameters based on
behavioral or morphological features of organisms. Sober's solution to the
problem was that even if the consequence laws contain some a priori truths,
it "does not hurt" the theory for its empiricalness is guaranteed by the source
laws which are based on causal, and thus empirical, analyses of organismal
performance. Sober thus thought the mathematical laws of evolution acquire
an empirical and causal nature in the course of application via the source
laws. If this is correct, the alleged a prioriness of evolutionary principles
would not entail non-empirical or non-causal nature of evolutionary theory
as a whole. Statisticalists, therefore, needed to dismiss such a possibility in

order to put forward their purely statistical interpretation of evolutionary theory.

For this purpose Matthen and Ariew (2002) distinguished two concepts of fitness, vernacular and formal. The *vernacular* fitness is roughly what we have in mind when we say that one organism is advantageous than another in a Darwinian race of survival and reproduction. For any pair of organisms in a given environment, we can ask which is more adaptive or "fitter" based on their physical properties, say speed, body size, etc. According to Matthen and Ariew, this vernacular understanding of fitness is at best a comparative notion — e.g. one is faster, bigger, or stronger than another. In contrast, formal models of population genetics require a more fine-grained measure of *formal fitness*, defined as "the *expected* rate of increase ... of a gene, a trait, or an organism's representation in future generations"(p.56, their emphasis). The per capita rate of increase is not just comparative, but comes in degree and is represented by a rational number. Now the problem they see in the Soberian solution is that there is a fundamental gap between a merely comparative order on the one hand and a quantitative measurement on the other: one can never arrive at the latter by comparison, but only by a direct census, they claim.

By the same token Matthen and Ariew (2005) reject Bouchard and Rosenberg (2004)'s attempt to ground their PNS mentioned above on pairwise comparisons of organisms' capacity to solve a specific design problem posed by the environment. Such a capacity, or what Bouchard and Rosenberg call "ecological fitness," is nothing but the vernacular fitness in Matthen and Ariew's parlance, and for this reason they find it impossible to sustain quantitative formulae of evolution.

But are these two — comparative-vernacular and quantitative-predictive

— notions of fitness really inconsistent to each other? The contrary is suggested by measurement theory, a branch of applied mathematics that identifies operational criteria for assigning quantitative measures to a set of objects (e.g. Krantz et al., 1971). According to this theory, one of the most fundamental requirements for objects to be measured with the ratio scale (which the "formal fitness" is) is that they allow pairwise comparison. This is intuitive if one recalls familiar measures, such as the kilogram system, are ultimately reduced to repeated pairwise comparisons by using, say, a balance. That the vernacular or ecological fitness is a comparative notion, therefore, is far from inconsistent but rather a necessary condition for there to be a quantitative measurement of organisms' survival and/or reproductive performance.

In fact, under certain conditions repeated comparisons of reproductive success prove to be sufficient to give rise to the fitness measure as used in population genetics. Wagner (2010) devised such a pairwise competition test, where in each hypothetical experiment a pair of genotypes compete to each other. The "winner" of the competition is the one that increases its share against the other. Repeating the experiment with different pairs of genotypes generates an order over the set of genotypes, upon which Wagner constructs a ratio scale measure of fitness and derives Wright's selection equation. This result substantiates Bouchard and Rosenberg (2004)'s idea to reduce the predictive measure of fitness to pairwise comparisons of reproductive or survival success, *pace* the statisticalist assertion that any such reduction is impossible.

3.2 The causal basis of fitness

Statisticalists, however, may be quick to respond as follows. Granted that Wagner's method allows us to construct a formal measure of fitness out of pairwise comparisons. What this method compares, however, are relative growth rates, not physical properties, of genotypes. And since the growth rate of a genotype is estimated by census (i.e. by counting the number of its offspring), it is still far cry from analyzing the formal fitness in terms of its causal basis.

The point is well-taken. Whether fitness is and should be based on causal properties of organisms is an old problem in the philosophy of biology, often debated under the heading of the *propensity interpretation* of fitness (Brandon, 1978; Brandon and Ramsey, 2007; Mills and Beatty, 1979; Sober, 1984, 2001, 2013; Rosenberg, 1985; Ariew and Ernst, 2009; Pence and Ramsey, 2013). This labeling, however, may blur rather than reveal what is at issue, for historically the "propensity interpretation" has been used by different authors to denote different theses, to name a few (i) that fitness denotes a *propensity or capacity* of an organism to survival and reproduce, rather than its actual performance; (ii) that fitness should be defined by the *statistical expectation*, rather than a sample moment; (iii) that fitness is *caused by organismal phenotype*; and (iv) that for any fitness function there is a *scalar value* that summarizes the direction of the adaptive response. Here we focus only on the third "interpretation" according to which fitness, as used in evolutionary theory, is a causal consequence of physical or behavioral properties of organisms. Statisticalists have challenged this thesis at two fronts: first, they deny an organism's fitness to be determined from its properties; second, it is argued that the fitness-phenotype relation needs not be causal as long as there is a statistical association between them.

The first line of skepticism resorts to the context-dependency of fitness. It is well known that in frequency-dependent selection the fitness of an individual organism depends on population-level parameters such as the population size or genetic/phenotypic frequencies (Ariew and Lewontin, 2004; Ariew and Ernst, 2009). Gillespie (1974), for example, has shown that when a population consists of two genotypes that produce offspring at different variances, the evolutionary trajectory is affected by the population size. Ariew and Ernst (2009) take this theoretical observation to contradict the propensity interpretation of fitness in the sense defined above, for it shows a case where fitness cannot be uniquely determined from properties of an individual organism.

An obvious flaw in this argument is that proponents of the propensity interpretation do not need to assert the fitness of an organism to be determined *solely* from its own properties. All they need to defend is that an individual property is *a* — not *the* — cause of fitness. To make an analogy, the premium of my car insurance is determined by, along with my own driving record, "population parameters" that summarize various conditions of hosts of drivers whom I haven't even met. Even still my driving record and habit affect my premium, and do so causally — it could have been cheaper should I have gotten less tickets, or used my car less frequently, and so on. Likewise, the fact that fitness depends on population parameters in some cases does not preclude an organismal property (either genetic or phenotypic) from being *a* cause of fitness.[4]

The second criticism of the propensity interpretation concerns the nature of the fitness-phenotype relationship. For even if fitness is a function of an organismal character, the functional relationship may not be causal, but

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[4]For this reason some statisticalist, e.g. Walsh (2007, p. 288), dismisses this line of approach.

just associational. Statisticalists in fact argue that it *needs not be* causal, claiming the essential condition for adaptive evolution to be nothing other than differences in expected trait fitness (Walsh et al., 2002). This condition — more formally $E(W|z_i) \neq E(W|z_j)$ for different types $z_i \neq z_j$ — is of purely statistical nature and does not require type Z to be a cause of fitness W. For this reason statisticalists claim that modern evolutionary genetics does not concern causes, but only statistical properties of a population which can be estimated just by census (Matthen and Ariew, 2002; Ariew and Ernst, 2009; Ariew et al., 2015).

This, however, is belied by actual practices of evolutionary ecology, one of the central concerns of which is to identify whether and how a phenotypic character under study *causally contributes* to the survival or reproductive success of organisms. Millstein (2006, 2008) analyzes some case studies where field biologists try to establish causal relationships between fitness on the one hand and phenotypic or genetic characters on the other, in order to confirm their adaptation hypotheses that the traits in questions were formed by selection and not by drift. Another classical example is Andersson (1982)'s field study of sexual selection in which he confirmed tail length of widowbirds affect their mating chance (a surrogate measure of fitness) by experimentally manipulating the phenotype. Why did these biologists insist on causality? The answer is because a mere phenotype-fitness correlation is not enough to induce adaptive response: the relation must be causal (Glymour, 2011; Otsuka, in press). The essential condition for a particular trait to change its frequency in the subsequent generation in response to selection is not just a correlation or differences of expected trait fitness, but that the trait *causes* fitness. Two conditions, statistical and causal, come apart when there is a confounder, e.g. an environmental factor that affects both fitness

and the phenotype. In such cases no evolutionary response occurs even if fitness correlates with the phenotype (that is, even if there are differences in expected trait fitness). To avoid making wrong predictions, therefore, biologists must ascertain that the observed correlation is fully accounted by the causal effect of the trait on fitness (Rausher, 1992; Morrissey et al., 2010).

3.3 The apparent autonomy

In a recent defense of the statisticalism Ariew et al. (2015, pp. 647–8) claimed:

> in each case [of explanation in population genetics] the explanans is 'statistically autonomous', involving two general steps: assumptions that allow for the use of a statistical model and then deduction from that model ... this deductive procedure is sufficient for explanation and no further appeal to causes is necessary.

That is, all we need to know to use population genetics models are "the statistical properties of the population — for example, its mean and variance (p. 651)" but not the causal properties. This view is supposedly motivated by the fact that population genetics models are usually expressed in terms of statistical functions. But since these equations obtain only under certain causal assumptions (Sec. 2), a violation of these causal assumptions may result in a wrong prediction, *even if one gets all the relevant statistics right.*

Any model is only as good as its assumption. Hence a successful application of an evolutionary model depends on the veracity of its causal assumption from which the predictive equation is derived. An ideal application of evolutionary models will take the following steps: (1) identify the model to be used based on the causal features of the population under study (e.g. the system of reproduction, inheritance, the number of traits

contributing to fitness); (2) verify that the population satisfies the causal assumptions specified by the model (e.g. random mating, fitness-phenotype relationships); (3) estimate the parameters via statistical methods such as regression and/or analysis of variance. The statisticalist claim that explanations in population genetics are "statistically autonomous" — that they require only statistical information — stems from an exclusive focus on the last step. The alleged "autonomy" is illusory in two senses. First, these statistics are in fact estimates of the causal parameters (e.g. parameters in the structural equations). Second, the justification that such statistical functions correctly predict evolutionary changes can come only from the verification of the underlying causal assumptions (steps 2 and 3). Hence far from being unnecessary, "an appeal to causes" is crucial in empirical applications of mathematical models, and for this reason biologists take pain to identify the causal structure of a population in evaluating selection hypotheses or predicting future evolutionary trajectories.

4   Round 3: Evolutionary explanations and interventions

Thus far we have discussed the statisticalist controversy from two perspectives, one regarding the theoretical structure and the other empirical applications of evolutionary theory. The debate has yet another face, which concerns the nature of evolutionary *explanations* — does the theory provide causal explanations of population changes?

To answer this question we must first ask when an explanation in general is considered causal. The traditional account sees a scientific explanation as a relationship between two sets of propositions, *explanans* and *explanan-*

dum. An explanation is called causal if the former identifies a cause of the phenomenon described by the latter (Sober, 1984, ch. 5). Statisticalists have thus argued that the *explanans* of evolutionary changes refers only to statistical, but not causal, features of the population. This, as we have seen, was the gist of Ariew et al. (2015)'s claim that evolutionary explanations are "statistically autonomous."

In response, causalists have tried to show that evolutionary explanations indeed identify causes of evolutionary changes. For this aim most resort to the *interventionist account of causation* (Woodward, 2003), according to which some variable X is a cause of another Y if there is a hypothetical intervention on X that changes the probability distribution of Y.[5] Thus the causalist strategy is to point to a manipulation of selection, fitness, or drift that affects population frequencies. Sober and Shapiro argue that manipulating fitness or the variance thereof makes differences in evolutionary response, and thus that explanations of adaptive changes citing fitness count as causal explanations (Shapiro and Sober, 2007; Sober, 2013). Reisman and Forber submit a similar argument with respect to drift, arguing an intervention on the population size affects the strength of drift (Reisman and Forber, 2005; Forber and Reisman, 2007).

These claims did not go unchallenged. Statisticalists criticized such putative interventions do not satisfy some criterion or another of the interventionist account, and thus fail to establish the causalist conclusion. These challenges are examined in detail below, followed by a general diagnosis of the debate.

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[5]The precise definition given by Woodward (2003) is more complicated than this due primary to a possible violation of faithfulness, but these details can be ignored here.

4.1 Walsh's description independence thesis

In the Sober-Shapiro approach, the key contention is whether fitness can be a cause of adaptive evolution.[6] This has been put into question by a series of papers by Walsh (2007, 2010, 2014), who claims fitness fails to satisfy the necessary criterion of being a cause.

His argument is hinted by a well-known statistical puzzle called Simpson's paradox. Suppose two variables X and Y, and some partition of a population. Our intuition tells us if X and Y are positively correlated within every subpopulation, they must be so too in the overall population. This expectation is belied — the sign of correlation can flip between sub- and whole-population. In fact such an association reversal is not paradoxical and has long been recognized by statisticians as well as philosophers. Walsh, however, maintains that the phenomenon is peculiar only to merely statistical associations. That is, he claims in cases where X causes Y Simpson's reversal cannot happen: if, for example, X positively contributes to Y in each subpopulation, it must do so too in the whole population. Walsh (2007) calls this "description independence" of causal relationships, and seeks its justification in Judea Pearl (2000)'s *Sure Thing Principle* (Walsh, 2010).[7]

The second step of Walsh's argument is to show that under a certain circumstance fitness does not satisfy this context independence. The case in question is again Gillespie (1974)'s model discussed earlier (Sec. 3.2): if two types, say A and B, reproduce at different *variances*, their long-term growth rates depend on not only the individual performance of each type but also the population size. The moral Walsh draws from this is that if one

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[6]Note that this differs from the question as to the causal basis or propensity interpretation of fitness as discussed above (Sec 3.2), which asks whether phenotype can be a cause of fitness.

[7]A criterion essentially identical to Walsh's was already proposed by Cartwright (1979) and criticized by Dupré (1984).

describes the competition in small subpopulations A might be fitter than B, while in the whole population the opposite may hold. Fitness, hence concludes Walsh, is not description independent and thus cannot be a cause of evolution.

There are some confusions in Walsh's argument, most notably that the growth rate of a genotype in the Gillespie model is different from its fitness. But this aside, there are fundamental errors in the both premises of Walsh's alleged *reductio ad absurdum*, namely that (1) causal relations must be description independent; and that (2) Gillespie's model generates Simpson's reversal. With respect to (1), Northcott (2010) points out Walsh's description independence applies only to additive causes — if a cause acts in a non-linear fashion its contribution to the effect variable depends on the background contexts, not only in amount but also in sign. Similarly, Otsuka et al. (2011) demonstrate Walsh's justification based on Judea Pearl (2000)'s Sure Thing Principle simply misunderstands Pearl's theory of causality, and is unsound. Taken together these critics reveal the description independence is far from a necessary condition for causal relationships, and thus cannot be used to disqualify the causal power of fitness.

Otsuka et al. (2011) also note (2) the alleged "fitness reversal" in Gillespie's model obtains only under an invalid assumption that one can set the population size in an arbitrary way, as if whether an organism belongs to the larger or smaller population is a matter description. Such a supposition, however, not only is inconsistent with the construction of the Gillespie model (which is derived for a predetermined population size), but also contradicts biologists' general wisdom that the (effective) population size is an objective feature of the population under study. The last point cannot be emphasized enough, for the correct estimation of the size of an evolving population is one

of the most important and challenging problems in the modern population genetics since Fisher and Wright (e.g. Caballero, 1994; Coyne et al., 1997; Wade and Goodnight, 1998; Lynch, 2007). Were it purely a "matter of description," these efforts for estimation and debates over *the true* population size would lose their entire meaning.

Some causalists have resisted this realist take on population size, pointing to that scientists reserve the right to choose (the size of) a population to be studied (Abrams, 2013; Ramsey, 2013). It is true, or even truism, that scientists can and *must* decide on which population they are going to investigate, and their decision surely reflect varieties of epistemic or pragmatic factors such as research interests, available resource, considerations on statistical power, etc. Abrams cites cases of selection study on human populations that pooled some subpopulations for a greater statistical power. Yet another research group may well prefer a smaller population due to sparse data or limited resources. Such decisions must be made, but that's not the end of the story. They must further be *justified* vis-a-vis their research goal, and such justifications can come only from the nature. Pooling populations is allowed only when they are homogeneous (no mixture distribution), and inferences from a small population always risk overgeneralization. Hence although it is scientists who demarcate the population to be investigated, whether their decision turns out to be correct is not up to them.

Before moving, let us note that the statisticalist supposition that a population can be demarcated in arbitrary ways is a logical consequence of their doctrine that evolutionary equations are purely mathematical truths. Indeed, nothing prohibits one from applying the Price equation to a gerrymandered population. Suppose a "population" consisting of all American citizens whose first name start with "T," all kangaroos living in Queensland,

and my three goldfish. Count their descendants at some later time and the Price equation gives the exact change of any arbitrary chosen phenotypic mean, say height (length). This is precisely because the Price equation, as an a priori mathematical theorem, applies to whatever set of objects as long as they satisfy certain measurement conditions. This is not true with *predictive* evolutionary equations such as the breeder's equation, whose derivation requires certain causal assumptions (Sec. 2). To apply these equations, a population must be homogeneous with respect to the causal structure and consistent with the assumptions of the models. A causal structure is the unit of evolutionary theory which both affords and delimits the generalizability of evolutionary equations, and for this reason an evolving population cannot be demarcated willy-nilly.

4.2 Fitness-evolution relationship: causal or identical?

Recall under the interventionist account X counts as a cause of Y if there is an intervention on X that changes $P(Y)$. Based on this idea the causalists have suggested interventions on fitness (Shapiro and Sober, 2007; Sober, 2013) or on the population size (Reisman and Forber, 2005; Forber and Reisman, 2007) affect adaptive evolution and drift, respectively. But to establish the causal relation the hypothetical interventions must satisfy an additional condition: namely, the intervened variable X and the supposed effect Y cannot be logically related. Manipulating a man's marital status would certainly change whether he is a bachelor or not, but it is not because they are causally related, but rather logically the same. According to Matthen and Ariew (2009), the same applies to the interventions proposed by the causalists. Although manipulating, say, the fitness variance may affect evolutionary changes, it is just because they are the logically same thing — a

variation in fitness *is* evolution.

The argument they develop to support this claim is not straightforward, but may be summarized into two points. The first is the now-familiar statisticalist doctrine that evolutionary equations relating fitness variation to evolution are mathematical truths — "natural selection is mathematically necessary."(Matthen and Ariew, 2009, p. 211) As we have already seen, however, they are *not* mathematical truths, and thus this line of reasoning may be dismissed. The second point concerns their peculiar definition of selection: "natural selection is evolution due to heritable variation in fitness."(Matthen and Ariew, 2009, p. 204)[8] Defined in this way, of course selection logically implies adaptive evolution, but concluding the causal inertness of selection on this ground is just moving the goalposts. In fact, this "definition" of natural selection is a far cry from its common usage, and contradicted by the opening sentence of Fisher (1930): "Natural selection is not evolution."

4.3 Causes of evolution

Although the charge made by Matthen and Ariew (2009) may be dismissed as ungrounded, it does not automatically vindicate the causalism. To prove some variables to be causes of evolution, it must be shown that an intervention on those variables is well defined and effectively affects the evolutionary response. How can this be achieved? In the causal graph theory an intervention is represented as a manipulation of a causal model, and using this manipulated model the effect of the intervention can be evaluated in a straightforward manner (Pearl, 2000; Spirtes et al., 2000). Hence the causal model underlying the breeder's equation (Fig. 1) may be used to

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[8]Matthen and Ariew attribute this definition to Sober (1984, pp. 21-22), but I couldn't locate it in the pages they point to.

examine if a variable of interest, such as fitness, causes population changes. Otsuka (in press) identifies two types of intervention affecting linear evolutionary changes. First, manipulating selective pressure β affects the rate and direction of evolutionary responses by regulating the contribution of the phenotype to fitness. Second, so-called "soft interventions" (interventions that leave other causal inputs intact) on fitness influence the rate of evolutionary changes. Suppose, for example, the skin thickness of some lizards contributes to fitness by functioning as thermoregulation. Then raising (or decreasing) the environmental temperature will lead to a negative (positive) response in the mean skin thickness, with the rate of evolutionary change being proportional to the absolute value of the temperature change. On the other hand, culling a certain number of offspring of each individual regardless of its skin will not affect the direction of response, but will accelerate adaptive evolution of the skin thickness. Hence *pace* statisticalists these interventions clearly indicate causes of adaptive evolution.

Although Otsuka (in press) focuses exclusively on selection, a cause of drift can be shown in a similar manner. In a linear selection the strength of drift is measured by the variance of the average phenotypic change, $\mathrm{Var}(\Delta \bar{Z})$, where the upper bar denotes the sample mean in this context. For the sake of simplicity let us focus only on drift due to selection, assuming a perfect heritability (i.e. $\mathrm{Var}(E_Z) = \mathrm{Var}(E'_Z) = 0$ in Fig. 1). Then taking the variance of the sample covariance in the Price equation $\Delta \bar{Z} = \mathrm{Cov}(W, Z)/\bar{W}$, it can be shown

$$\mathrm{Var}(\Delta \bar{Z}) = \frac{1}{N \bar{W}^2} \mathrm{Var}(Z) \, \mathrm{Var}(W) \tag{1}$$

(Rice, 2004, pp. 183–185). This equation identifies three factors contributing

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to drift: population size N, phenotypic variance $\mathrm{Var}(Z)$, and fitness variance $\mathrm{Var}(W)$. Using the structural equation for fitness in Fig. 1, the last factor is unpacked as

$$\mathrm{Var}(W) = \beta^2 \, \mathrm{Var}(Z) + \mathrm{Var}(E_W). \tag{2}$$

Combined with Equation 1, this means one can regulate the strength of drift by manipulating independent error term E_W.

The independent error term summarizes all causes of fitness that are independent of and act additively with respect to the phenotype in question. Examples along the line of the above hypothetical lizards might include predators' attack and mating chance, provided these factors do not interfere with the thermoregulation of the skin.[9] These additive factors of fitness are what Millstein (2005, p. 171) calls *indiscriminate sampling process* "in which physical differences between organisms are causally irrelevant to differences in reproductive success," if we interpret the causal irrelevance as meaning that the process is not affected or regulated by "physical difference between organisms." Our conclusion here thus accords with the claim of Millstein et al. (2009, see also Sec. 2.2) that such indiscriminate processes underlie drift. On the other hand, the selective pressures that regulate the fitness contribution of the focal phenotype can be thought as a *discriminate sampling process* "in which physical differences between organisms are causally relevant to differences in reproductive success."

It has long been an issue whether selection and drift should be understood as mere "outcomes" or "processes" (e.g. Walsh et al., 2002; Stephens, 2004; Matthen, 2009, 2010; Millstein, 2002, 2005). Proponents of the mere-outcome view hold there to be no causal factors or forces specific to selection

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[9]But since interventions on these error terms count at the same time as soft-interventions on fitness, they may also affect the rate of adaptive response if they change the mean fitness. I thank Bruce Glymour for pointing to this.

or drift, the difference between these evolutionary phenomena emerging only as a result of statistical abstraction. But if selection and drift appear to be irreducible statistical facts, this is only because they exclusively focus on the equations which just describe evolutionary outcomes. It is by uncovering the causal basis of these equations that we find causes of evolutionary changes.

It should be noted that the causes of evolutionary changes as shown above all belong to *the level of individuals*, in the sense that these variables, including environmental factors, denote properties of individuals.[10] In contrast some authors have argued that selection and drift should be understood as population-level causes that "act" on an entire population (Reisman and Forber, 2005; Millstein, 2006; Abrams, 2007). To make her case Millstein points to the fact that selection is a comparative notion: the reproductive success of one organism almost always depends on its peers' success. That means the fitness of one organism is determined only with respect to all other individuals in the population, and Millstein thinks such a reference to the population makes selection a population-level process (Millstein, 2006, pp. 643–4). One problem about this argument is such comparative processes are ubiquitous. According to this criterion school admission, lottery, and stock market all count as population-level processes. Moreover, the reference to a population is not unique to fitness measure: indeed, the possibility of pairwise comparisons is a necessary requirement for any type of measurement, including banal properties like length or weight, as we have seen in the discussion of measurement theory (Sec. 3.1.) Hence if the reference to the population in the overall comparison is the issue these properties should also qualify as population properties — but then calling selection a population-level process would cease to elucidate much about its nature.

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[10]Environmental factors in the causal graph represent these aspects of environment that are "experienced" by each individual, and are properties of individuals in this sense.

Another argument for the population-level account focuses on the role of the population size in regulating the strength of drift (see Eqn. 1). Reisman and Forber (2005) cites the famous experiment of Dobzhansky and Pavlovsky (1957) who measured the strength of drift by controlling the sizes of multiple Drosophila populations. Since the variable manipulated in this experiment — population size — is a property of populations, Reisman and Forber conclude drift to be a population-level cause of evolution. A concern about this argument similar to the one raised by Matthen and Ariew (2009) is that it is not clear where in the causal graph population size N figures in, and unless this is specified one cannot determine whether or in what sense the population size *causes* evolution. Alternatively, one may interpret N to be not a population parameter but a kind of contextual variable, i.e. the variable that measures the size of the surrounding population an organism happens to be in, and thus makes it an "individual property." In either way, the possibility of a population-level cause of evolutionary changes cannot be discussed separately from the debate on levels of selection (Okasha, 2006), and will need further investigations.

4.4   In what sense are evolutionary explanations causal?

The above discussions based on the causal model suggest that *pace* statisticalists there are some variables that count as causes of adaptive as well as non-adaptive evolution. A few caveats, however, are in order regarding the explanatory role of fitness in evolutionary theory. As we have seen, there are some interventions on fitness that affect evolutionary changes, and on this ground fitness may well be said to be a cause of evolution. But it does not necessarily mean that such causal statements play an important epistemic role. True, the common lore holds that evolutionary theory explains

adaptive changes by variation in fitness. I don't claim this to be wrong, but incomplete and misleading as a characterization of evolutionary explanations. As mentioned above, what really matters in Darwinian evolution is not fitness per se, but its causal *relationship* to phenotype. When a biologist concludes a certain phenotype to be an adaptation, what she means is that there has been an environmental factor that regulated the fitness contribution of the phenotype, so that had the environment, and thus the fitness-phenotype relation, been different a different phenotypic distribution would have obtained (Wade and Kalisz, 1990; Glymour, 2011). Of course this explanation assumes a certain variability in fitness, but it is not the most explanatory part of the story.

Nor is it my view that explanations provided by evolutionary theory are causal *because* they point to fitness as a cause of evolutionary change. Such a reasoning tacitly presupposes that the epistemic status of a theory is revealed by analyzing its key *explanans*. This way of casting the theory into a few summary propositions, however, blurs the fact that most explanatory works in evolutionary science proceed by building models of the target systems (Lloyd, 1988). If so, the straightforward way to identify the nature of evolutionary explanations is to analyze the construction process and assumptions of these models, rather than trying to interpret their verbal recapitulations. I thus believe evolutionary theory does provide causal explanations of population changes, not because "it cites a cause of evolution," but because it relies on models that explicitly deal with the causal structure of evolving populations.

5   Conclusion

Statisticalists hold that evolutionary theory is not a causal but purely statistical theory. The present review critically examined this claim from three perspectives, each concerning the assumptions, applications, and explanations of evolutionary theory. From any perspective the statisticalist doctrine cannot be maintained. Contrary to the claim that evolutionary changes are "mathematical necessities," deriving predictive equations in population genetics requires more than probability theory, but certain causal models and assumptions (Sec. 2). To apply any of these equations to an actual population, therefore, one needs not only statistics but also information about the causal features of the population (Sec. 3). This also means that evolution is explained from the causal features of a population, with adaptive as well as non-adaptive evolution having corresponding causes in the sense of the interventionist account of causation (Sec. 4).

In *Critique of the Pure Reason* Kant emphasized the importance of formulating a question in the right way — trying to answer an ill-formed question represents, as he puts it, "the ridiculous sight ... of one person milking a billy-goat while the other holds a sieve underneath."(Kant, 1998, p. 197, A58) I think a similar moral applies to the debate under review. How should we put our question, if we want to know the causal nature of evolutionary theory? From the beginning the statisticalist controversy has been framed as a problem about *interpretations* — of fitness, selection, or drift. Matthen and Ariew (2002) alleged an inconsistency between two interpretations of fitness — vernacular and formal — while Walsh et al. (2002) aimed to "distinguish dynamical and statistical interpretations of evolutionary theory." To these challenges causalists have responded with counter interpretations, such as the propensity view of fitness or the process view of selection/drift.

But why does interpreting the concepts like these have anything to do with the causal or empirical nature of the theory? One implicit rationale, I suspect, is the aforementioned belief that evolutionary theory can be summarized into a few explanatory *explanans*, like "adaptive evolution results from variation in fitness" or "survival of the fittest." Given these slogans, it was hoped the correct interpretation of the concepts therein would uncover the epistemic nature of evolutionary explanations and theory.

One should not confuse, however, a summary with the theory. The popular principles or equations of evolution do not stand alone but are derivative of underlying models, and the concepts or parameters loose their meaning if detached from the theoretical context. To neglect this and ponder just about interpretations of linguistic expressions is like "holding a sieve underneath" a goat without asking its sex, whereas what one should really do is to examine the goat, i.e. the model, itself! Once we turn our attention to the construction process of the models used in population genetics, it instantly becomes evident that they are far from a priori but based on causal, and thus empirical, assumptions. At the same time, the theoretical as well as causal role of the concepts like fitness, selection, and drift is determined unequivocally within these causal models.

The question about the causal nature of evolutionary theory, therefore, is not about its interpretation, but about its models or theory itself. Or to borrow Patrick Suppes' famous slogan, the problem is properly addressed by a scientific, rather than meta-scientific, analysis. Any meta-scientific interpretation unaccompanied by a serious analysis of the theory itself fails to establish a secure conclusion but leads only to an endless disputation. This, I think, is the lesson we should draw from the debate that has lasted for over a decade.

References

Abrams, M. (2007). How Do Natural Selection and Random Drift Interact? *Philosophy of Science*, 74(5):666–679.

Abrams, M. (2013). Populations and pigeons : Prosaic pluralism about evolutionary causes. *Studies in history and philosophy of biological and biomedical sciences*, 44:294–301.

Andersson, M. (1982). Female choice selects for extreme tail length in a widowbird. *Nature*, 299:818–820.

Ariew, A. and Ernst, Z. (2009). What fitness can't be. *Erkenntnis*, 71:289–301.

Ariew, A. and Lewontin, R. (2004). The confusions of fitness. *The British Journal for the Philosophy of Science*, 55(2):347–363.

Ariew, a., Rice, C., and Rohwer, Y. (2015). Autonomous-Statistical Explanations and Natural Selection. *The British Journal for the Philosophy of Science*, 66:635–658.

Birch, J. (2014). Hamilton's rule and its discontents. *The British Journal for the Philosophy of Science*, 65:381–411.

Bouchard, F. and Rosenberg, A. (2004). Fitness, probability and the principles of natural selection. *The British Journal for the Philosophy of Science*, 55:693–712.

Brandon, R. N. (1978). Adaptation and evolutionary theory. *Studies in History and Philosophy of Science Part A*, 9(3):181–206.

Brandon, R. N. and Ramsey, G. (2007). What s Wrong with the Emergentist Statistical Interpretation of Natural Selection and Random Drift ? In

Hull, D. L. and Ruse, M., editors, *The Cambridge Companion to the Philosophy of Biology*, pages 66–84. Cambridge University Press, Cambridge.

Caballero, A. (1994). Developments in the prediction of effective population size. *Heredity*, 73:657–679.

Cartwright, N. (1979). Causal laws and effective strategies. *Nous*, 13(4):419–437.

Coyne, J. A., Barton, N. H., and Turelli, M. (1997). Perspective: a critique of Sewall Wright's shifting balance theory of evolution. *Evolution*, 51(3):643–671.

Dobzhansky, T. and Pavlovsky, O. (1957). An experimental study of interaction between genetic drift and natural selection. *Evolution*, 11:311–319.

Dupré, J. (1984). Probabilistic causality emancipated. *Midwest Studies in Philosophy*, 9(1):169–175.

Fisher, R. A. (1930). *The genetical theory of natural selection*. Oxford University Press, Oxford.

Forber, P. and Reisman, K. (2007). Can There Be Stochastic Evolutionary Causes? *Philosophy of Science*, 74(5):616–627.

Gillespie, J. H. (1974). Natural Selection for Within-Generation Variance in Offspring Number. *Genetics*, 76(3):601–606.

Glymour, B. (2011). Modeling Environments : Interactive Causation and Adaptations to Environmental Conditions. *Philosophy of Science*, 78:448–471.

Kant, I. (1998). *Critique of Pure Reason*. Cambridge, UK.

Krantz, D., Luce, R., Suppes, P., and Tversky, A. (1971). *Foundations of Measurement (Additive and Polynomial Representations), vol. 1.* Academic Press, San Diego.

Lewens, T. (2004). *Organisms and artifacts: design in nature and elsewhere.* MIT Press Cambridge, MA.

Lloyd, E. A. (1988). *The structure and confirmation of evolutionary theory.* Princeton University Press, Princeton.

Lynch, M. (2007). *The origins of genome architecture.* Sinauer Associates Inc., Sunderland, MA.

Matthen, M. (2009). Drift and Statistically Abstractive Explanation. *Philosophy of Science*, 76(4):464–487.

Matthen, M. (2010). What is Drift? A Response to Millstein, Skipper, and Dietrich. *Philosophy & Theory in Biology*, 2:e102.

Matthen, M. and Ariew, A. (2002). Two ways of thinking about fitness and natural selection. *The Journal of Philosophy*, 99(2):55–83.

Matthen, M. and Ariew, A. (2005). How to understand causal relations in natural selection: Reply to Rosenberg and Bouchard. *Biology & Philosophy*, 20:355–364.

Matthen, M. and Ariew, A. (2009). Selection and Causation. *Philosophy of science*, 76:201–224.

Mills, S. K. and Beatty, J. (1979). The propensity interpretation of fitness. *Philosophy of Science*, 46:263–286.

Millstein, R. (2002). Are Random Drift and Natural Selection Conceptually Distinct? *Biology & Philosophy*, 17(1):33–53.

Millstein, R. (2005). Selection vs. drift: A response to Brandon's reply. *Biology & Philosophy*, 20(1):171–175.

Millstein, R. (2006). Natural selection as a population-level causal process. *The British Journal for the Philosophy of Science*, 57(4):627–653.

Millstein, R. (2008). Distinguishing drift and selection empirically:The great snail debate of the 1950s. *Journal of the History of Biology*, 41(2):339–367.

Millstein, R. L., Skipper, R. A. J., and Dietrich, M. R. (2009). (Mis)interpreting Mathematical Models: Drift As A Physical Process. *Philosophy & Theory in Biology*, 1:e002.

Morrissey, M. B., Kruuk, L. E., and Wilson, A. (2010). The danger of applying the breeder's equation in observational studies of natural populations. *Journal of Evolutionary Biology*, 23(11):2277–2288.

Morrissey, M. B., Parker, D. J., Korsten, P., Pemberton, J. M., Kruuk, L. E., and Wilson, A. J. (2012). The Prediction of Adaptive Evolution: Empirical Aapplication of the Secondary Theorem of Selection and Comparison to the Breeder's Equation. *Evolution*, 66(8):2399–2410.

Northcott, R. (2010). Walsh on Causes and Evolution. *Philosophy of Science*, 77:457–467.

Okasha, S. (2006). *Evolution and the Levels of Selection.* Oxford University Press, Oxford.

Otsuka, J. (2015). Using Causal Models to Integrate Proximate and Ultimate Causation. *Biology & Philosophy*, 30(1):19–37.

Otsuka, J. (in press). Causal Foundations of Evolutionary Genetics. *The British Journal for the Philosophy of Science.*

Otsuka, J., Turner, T., Allen, C., and Lloyd, E. A. (2011). Why the Causal View of Fitness Survives. *Philosophy of Science*, 78(2):209–224.

Pearl, J. (2000). *Causality: Models, Reasoning, and Inference.* Cambridge University Press, New York, NY.

Pence, C. H. and Ramsey, G. (2013). A new foundation for the propensity interpretation of fitness. *The British Journal for the Philosophy of Science*, 64:851–881.

Pigliucci, M. and Kaplan, J. (2006). *Making Sense of Evolution: The Conceptual Foundations of Evolutionary Biology.* University of Chicago Press, Chicago.

Price, G. R. (1972). Extension of covariance selection mathematics. *Annals of Human Genetics*, 35:485–490.

Ramsey, G. (2013). Organisms, traits, and population subdivisions: Two arguments against the causal conception of fitness? *British Journal for the Philosophy of Science*, 64(3):589–608.

Rausher, M. D. (1992). The measurement of selection on quantitative traits: biases due to environmental covariances between traits and fitness. *Evolution*, 46(3):616–626.

Reisman, K. and Forber, P. (2005). Manipulation and the Causes of Evolution. *Philosophy of science*, 72:1113–1123.

Rice, S. H. (2004). *Evolutionary theory: mathematical and conceptual foundations.* Sinauer Associates, Sunderland, MA.

Rosenberg, A. (1985). *The structure of biological science.* Cambridge University Press, Cambridge, UK.

Rosenberg, A. and Bouchard, F. (2005). Matthen and Ariew's Obituary for Fitness: Reports of its Death have been Greatly Exaggerated. *Biology & Philosophy*, 20(2-3):343–353.

Shapiro, L. and Sober, E. (2007). Epiphenomenalismthe Do's and the Don'ts. In Wolters, G. and Machamer, P., editors, *Thinking about Causes: From Greek Philosophy to Modern Physics*, pages 235–264. University of Pittsburgh Press, Pittsburgh.

Sober, E. (1984). *The nature of selection: Evolutionary theory in philosophical focus*. University of Chicago Press, Chicago.

Sober, E. (1993). *Philosophy of biology*. Westview Press, Boulder.

Sober, E. (2001). The Two Faces of Fitness. In Singh, R. S., Krimbas, C. B., Paul, D. B., and Beatty, J., editors, *Thinking about Evolution*, pages 309–321. Cambridge University Press, Cambridge.

Sober, E. (2013). Trait fitness is not a propensity, but fitness variation is. *Studies in history and philosophy of biological and biomedical sciences*, 44(3):336–41.

Spirtes, P., Glymour, C., and Scheines, R. (2000). *Causation, Prediction, and Search*. The MIT Press, Cambridge, MA, 2nd edition.

Stephens, C. (2004). Selection, Drift, and the "Forces" of Evolution. *Philosophy of Science*, 71(4):550–570.

van Fraassen, B. C. (1980). *The scientific image*. Oxford University Press, Oxford.

Wade, M. J. and Goodnight, C. J. (1998). Perspective: the theories of Fisher

and Wright in the context of metapopulations: when nature does many small experiments. *Evolution*, 52(6):1537–1553.

Wade, M. J. and Kalisz, S. (1990). The Causes of Natural Selection. *Evolution*, 44(8):1947–1955.

Wagner, G. P. (2010). The measurement theory of fitness. *Evolution*, 64(5):1358–1376.

Walsh, D. (2014). Variance, Invariance and Statistical Explanation. *Erkenntnis*.

Walsh, D. M. (2007). The Pomp of Superfluous Causes: The Interpretation of Evolutionary Theory. *Philosophy of Science*, 74:281–303.

Walsh, D. M. (2010). Not a Sure Thing: Fitness, Probability, and Causation. *Philosophy of Science*, 77(2):147–171.

Walsh, D. M., Lewens, T., and Ariew, A. (2002). The Trials of Life: Natural Selection and Random Drift. *Philosophy of Science*, 69(3):452–473.

Woodward, J. B. (2003). *Making Things Happen: A Theory of Causal Explanation*. Oxford University Press, New York.