2015

# Autonomous Statistical Explanations and Natural Selection

André Ariew

Collin Rice
*Bryn Mawr College*, crice3@brynmawr.edu

Yasha Rohwer



Custom Citation

Ariew, André, Collin Rice, and Yasha Rohwer. 2015. "Autonomous Statistical Explanations and Natural Selection." The British Journal for the Philosophy of Science 66.3: 635–658.

Autonomous Statistical Explanations and Epiphenomenalism: A Reply to Shapiro and Sober

**Abstract.** In "Epiphenomenalism—the Do's and the Don'ts"(2007) Shapiro and Sober (henceforth S/S) claim that Walsh, Ariew, Lewens, and Matthen (henceforth WALM) give an a priori defense of natural selection and drift as epiphenomenal and that this defense is mistaken. Contra S/S, we first argue that WALM's explanatory doctrine does not require a defense of epiphenomenalism. We then defend WALM's explanatory doctrine by arguing that the explanations provided by the modern genetical theory of natural selection are autonomous statistical explanations analogous to Galton's explanation of reversion to mediocrity and an explanation of the diffusion of gases. We then argue that whereas Sober's theory of forces is an adequate description of Darwin's theory, WALM's explanatory doctrine is required to understand how the modern genetical theory of natural selection explains large-scale statistical regularities.

**1. Introduction.**

In "Epiphenomenalism—the Do's and the Don'ts"(2007) Shapiro and Sober (henceforth S/S) claim that Walsh, Ariew, Lewens, and Matthen (henceforth WALM) give an a priori defense of natural selection and drift as epiphenomenal and that this defense is mistaken.[1] They argue that if natural selection and drift are epiphenomenal, this fact should be defended on empirical grounds, modeled after Weismann's experiments with mice. To S/S, what qualifies WALM's views about natural selection and drift as epiphenomenal is WALM's general belief that "natural selection really does occur and it really does explain. It explains changes in trait frequencies, not by citing their causes, but by a statistically apposite kind of bookkeeping" (Shapiro and Sober, 2007, 251).

In this paper, we will first argue that S/S misunderstand WALMs' views about natural selection and drift (or at least they miss a central line of thinking).[2] As the quote suggests, WALMs' is an explanatory doctrine, not an a priori epiphenomenalist doctrine. For WALM, evolutionary ensemble change is explained by deriving a mathematical consequence of a model equation that refers to the statistical properties of a population. Contrary to S/S, this position

[1] Walsh, Lewens, Ariew (2003); Matthen and Ariew (2002).

[2] In this draft rather than providing textual evidence for our representation of WALMs' views, we focus on what we see as a central insight that S/S ignore.

does not require epiphenomenalism. Consequently, experiments of the kind performed by Weismann are inadequate for testing WALM's position.

After distinguishing WALM's explanatory doctrine from epiphenomenalism, we will defend WALM's view by arguing that the modern genetical theory of natural selection provides autonomous statistical explanations. We do this by showing that the explanations provided by the modern genetical theory (which is constituted by a collection of evolutionary models) are analogous to other autonomous statistical explanations—namely, Galton's use of the normal curve to explain the reversion to mediocrity (Hacking, 1990) and the use of Boyle's law to explain the diffusion of gases. Biological populations, we will argue, are instances of what James Woodward calls a "complex system" where there is a very large number of different possible causal processes that produce a macroscopic outcome (Woodward, 2011). Furthermore, we will argue that large-scale regularities in these complex systems can be explained by referring to the deductive consequences of statistical models (independent of considerations of causation). This is the kind of autonomous statistical explanation involved in WALM's *explanatory doctrine* (we will use this label throughout).

Clarifying WALM's explanatory doctrine in this way and identifying the context in which it is intended to apply provides important insights for the debate over how to interpret the explanations provided by the modern theory of natural selection. In order to settle the question about whether evolution is appropriately characterized as "a theory of forces," as Sober and others would have it, or a statistical model theory, as WALM would have it, we have to pay attention to what is being explained and how evolutionists propose to explain it. After distinguishing Darwin's theory of natural selection from the modern genetical theory, we argue that whereas Sober's theory of forces is an adequate description of Darwin's theory, WALM's explanatory doctrine is required to understand how the modern genetical theory of natural selection explains large-scale statistical regularities.

The following section outlines why S/S believe WALM's view is committed to epiphenomenalism and the empirical test of epiphenomenalism they advocate. Section 3 reviews reasons provided by Matthen and Ariew (2009) for why the empirical test of epiphenomenalism advocated by S/S fails to show that natural selection is a cause of

evolutionary change. Section 4 argues that WALM's explanatory doctrine can be defended independent of epiphenomenalism. In support of this defense, Sections 5 and 6 present two examples of what we call, following Ian Hacking (1990), *autonomous statistical explanations*. These examples help to clarify WALM's explanatory doctrine and the context in which it is intended to apply. Section 7 defends WALM's explanatory doctrine (independently of epiphenomenalism) by distinguishing the modern genetical theory of natural selection from Darwin's theory and arguing that the former provides autonomous statistical explanations. The final section responds to a possible objection.

**2. S/S's epiphenomenalism do's and don'ts.**

Epiphenomenalism is a view commonly associated with mental properties, but it can be applied beyond that case. Epiphenomenalism claims that higher-level properties are the effects of physical processes, but are themselves causally inert. For example, in biology, an epiphenomenalist might maintain that phenotypes are the effects of genes, but phenotypes are causally inert with respect to genetic processes.

S/S advocate an empirical, rather than an a priori defense of epiphenomenalism, modeled after August Weismann's experiments with mice. Weismann snipped off the tails of newborn mice to see if the snipping would have any effect on the tail size of their offspring. It did not. To S/S the results demonstrate why "parental phenotypes are epiphenomenal with respect to the process of genetic inheritance" (Shapiro and Sober, 2007, 237).

S/S argue that it is also important to notice what Weismann did *not* do to demonstrate epiphenomenalism. Namely, Weismann did not argue that parental phenotypes are casually inert, simpliciter. That is, he did not argue that they have no effects of any kind. Rather, he demonstrated that parental phenotypes are epiphenomenal *with respect to the process of genetic inheritance*.[3] In addition, Weismann did not treat causation as a synchronic relationship between the macro-state of an organism at a particular time t and its micro-state at t. Weismann's experiment did not involve manipulating the parental phenotype while holding

[3] S/S compare the results favorably with Wesley Salmon's explication of a rotating beam of light as a "pseudo-process" (Salmon, 1984). Salmon does not claim that a circle of light moving across the ceiling of the Astrodome has no effects whatsoever; rather, he argues that the shape or color of the circle of light at one time has no causal influence on the shape or color at any other moment in its trajectory.

fixed its micro-supervenient base. Instead, Weismann treated the potential causal relationship between parental phenotypes and offspring genotypes as appropriately diachronic.

According to S/S, epiphenomenalists often fail to take heed of the lessons learned from Weismann's example. First, rather than viewing causation as a diachronic relationship, as Weismann did, they view causation as a synchronic relationship: "The idea is that the micro-state at t determines the macro-state at t; however, the converse is not true, owing to the fact that macro-properties are multiply realizable at the micro-level" (Shapiro and Sober, 2007, 240). According to S/S, many epiphenomenalists use this multiple-realizability to argue that "Given that any instance of a [higher-order] property X has a micro-supervenience base MSB(X), it would appear that X has no causal powers in *addition* to those that MSB(X) already possess. The absence of these additional causal powers is then taken to show that the [higher-order property] X is causally inert" (Shapiro and Sober, 2007, 7). The problem, S/S claim, is that this argument is based on a fallacy which is "the thought that if X causes Y, then X must have an impact on Y additional to the impact on Y that MSB(X) has" (Shapiro and Sober, 2007, 241). According to S/S, this is the wrong way to test whether X (e.g. a parental phenotype) causally influences Y (e.g. the offspring phenotype) because it requires asking an imponderable counterfactual question, "would Y occur if a sufficient condition for X occurred but X did not?" (Shapiro and Sober, 2007, 240). This is an imponderable question because the sufficient condition for X (e.g. its microsupervenient base) could not occur if X did not as well. S/S label this mistaken argument from multi-realizabilty, the "master argument for epiphenomenalism" (Shapiro and Sober, 2007, 241).

According to S/S, a better argument for epiphenomenalism takes its cue from Weismann's mice experiment. First, it should show that one class of properties has no effect on a second class of properties, not that the first class has no effects at all. Second, epiphenomenalism should be settled empirically by considering diachronic relationships, not a priori by considering synchronic relationships of multiple-realizability. Weismann did not need to rely on metaphysical considerations about supervenience and the nature of causation; all he needed was to deploy the appropriate "holding fixed" argument: the common causes of both X

and Y are held fixed while manipulating X to see whether that corresponds in a change in Y (see Figure 1).

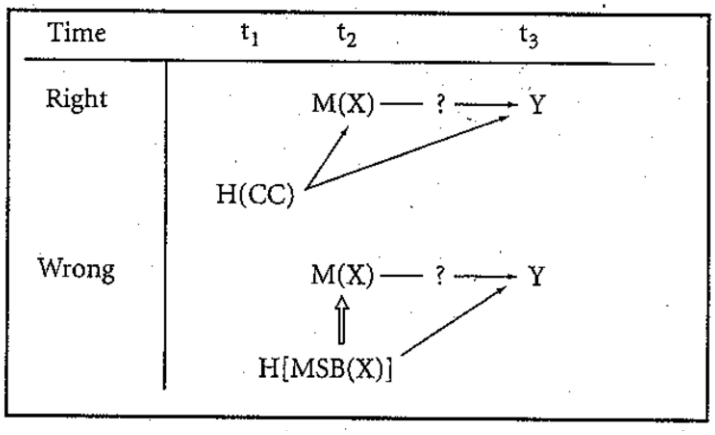


Figure 1: S/S's figure illustrating the right and wrong manipulations for determining whether X causes Y. M(X) means that X is manipulated by an intervention, H(Z) means that Z is held fixed, and MSB(X) is the microsupervenient base of X (Shapiro and Sober, 2007, 240)

S/S believe instances of the mistaken master argument for epiphenomenalism can be found in the philosophy of biology. Specifically, they target WALM's view of natural selection explanations via Denis Walsh's account of natural selection as a non-causal process. The title for Walsh's (2000) paper is "Chasing Shadows", an allusion to Salmon's work on causal processes and pseudo-processes. But, rather than successfully arguing that natural selection is a pseudo-process, Walsh supposedly invokes the erroneous "master argument for epiphenomenalism". S/S paraphrase Walsh in this way:"[T]here is no need to invoke a distinct force [of natural selection] operating over populations, when, at the level of individual organisms, there already are the many causes of individual births and deaths" (Shapiro and Sober, 2007, 250-1). S/S believe this amounts to invoking the mistaken master argument for epiphenomenalism since, "Walsh demands that selection contribute something to evolution beyond the contributions made by the causal processes that impinge on individual organisms" (Shapiro and Sober, 2007, 251). So it seems that S/S take Walsh to be correctly claiming that natural selection is multiply realizable, but incorrectly inferring that this entails that it is epiphenomenal. According to S/S, Walsh, and by proxy WALM, argue for epiphenomenalism incorrectly and therefore they do not present a good reason to deny that selection is a cause.

**3. S/S's manipulation tests cannot test whether natural selection is a cause.**

One could see why S/S might think that WALM's explanatory doctrine and epiphenomenalism go hand in hand. Consider the iconoclastic view, defended by Walsh (2007, 2010, manuscript) and Matthen and Ariew (2009), that (A) natural selection is not a cause of evolution. This view does share some features with epiphenomenalism; e.g. it requires that natural selection and drift are effects of physical processes, but are themselves causally inert. Some critics claim that natural selection is a cause of evolution on the grounds of manipulationist (or interventionist) criteria for causation (Reisman and Forber, 2005; Shapiro and Sober, 2007; Northcott, 2010). That is, they argue because you get effects on the population structure when you manipulate trait fitness distributions, natural selection is a cause of evolutionary change. Let us briefly review Matthen and Ariew's (2009) defense of (A) against the critics before presenting the main view to be defended in this paper.

According to Matthen and Ariew (2009) the kind of empirical manipulability tests advocated by S/S—i.e. Weismann's experiments with mice—fail to test the correct variables when considering whether or not natural selection is a cause. To show that natural selection is a cause, one must manipulate natural selection while holding fixed all other variables—including heritable variation in fitness. However, the arguments given in Matthen and Ariew (2009) aim to show that the manipulation tests envisioned by S/S are doomed to fail when they are appropriately applied to natural selection because natural selection does not meet the basic requirements of manipulability tests of causation.

Matthen and Ariew (2009) defend (A) by first granting that trait differences, such as variation in camouflage, often are causes of evolutionary change. This can be shown using various tests of causation; e.g. the probability raiser test and Woodward's (2003) manipulability test. These same tests, Matthen and Ariew argue, fail to establish causation when we consider the claim: natural selection causes evolutionary change because the relationship between variation-in-advantageous-traits and natural selection is purely mathematical. This entails that one cannot manipulate natural selection independent of variation-in-advantageous-traits. Yet, manipulability tests of causation require that causes be modular in the sense that they can be manipulated independently of other causes of the phenomenon (Woodward, 2003). The upshot

is that not all "holding fixed" tests are sufficient to establish causal relations. For example, in some cases, such holding fixed considerations only reveal analytic or mathematical relationships between variables.[4] Therefore, Matthen and Ariew conclude that natural selection is not a cause since it cannot be manipulated independent of variation-in-advantageous-traits.[5] This argument supports the ontological conclusion, (A): that natural selection does not cause evolutionary change. Moreover, contra S/S (2007) and Forber and Reisman (2005), Matthen and Ariew argue that any test that aims to manipulate the correct variable—i.e. natural selection—will fail to establish that natural selection is a cause over and above heritable variation in fitness. So while an empirical test of epiphenomenalism might be preferable, in the instance of natural selection, Matthen and Ariew (2009) aim to demonstrate (a priori) that no such "holding fixed" demonstration of epiphenominalism (or causation) is possible.

**4. WALM's explanatory doctrine.**

For the purposes of this paper, a brief survey of the debate concerning (A) suffices because our primary interest is an alternative view concerning natural selection explanations. Instead of defending (A), we are primarily interested in S/S's claim that WALM's statistical view of natural selection explanations *requires* a defense of epiphenomenalism at all. S/S seem to be ruling out (by necessity) that WALM could defend the explanatory doctrine without invoking epiphenomenalism or any other claim about whether X causes Y. We will argue that WALM's explanatory doctrine can be distinguished from metaphysical claims made by certain proponents of the WALM view. That is, WALM could argue that the explanations provided by the modern genetical theory of natural selection are what we will call *autonomous statistical explanations* without having to demonstrate that necessarily there is no population-level causal process, nor macro causal properties, that *could* be cited to explain changes in trait frequency. This position is independent of any commitment to epiphenomenalism and is the view we will defend here.

[4] For example, manipulate a man's marital status while holding fixed everything else and you will change whether he is a bachelor. But one should not conclude from this kind of holding fixed test that being unmarried causes one to be a bachelor. Instead, this test merely reveals an analytic relationship.

[5] They also argue that the same is true of the relationship between population size and drift. But that debate is tangential to our purposes here.

In order to clarify the view defended here and its relationship to epiphenomenalism, it is necessary to distinguish two claims that have been defended previously in the literature:

(A) *Natural selection* is not a *cause* of evolution (Matthen and Ariew, 2009; Walsh 2007, 2010).

(B) The modern genetical theory of natural selection *explains* by citing a statistical model, not by citing the causes of evolutionary change (Matthen and Ariew, 2002; Walsh, Lewens, Ariew, 2003).

(B) is a claim about what explains when one invokes the modern genetical theory of natural selection to account for some evolutionary phenomenon. This is WALM's *explanatory* doctrine. It claims that, "natural selection explanations appeal to a set of statistical properties of populations, viz. the mean (and variance) of fitnesses between trait types. Explanations of this sort *do not avert to forces*." (Walsh, Lewens, Ariew, 2003, 462). The nature of this explanation is, importantly, independent from any metaphysical claims about what causes what. Even if there is a population-level causal process that we want to call "natural selection", this does not entail that the statistical models used by the modern genetical theory provide explanations by citing that causal process. In other words, this kind of statistical explanation can proceed independent of any citation of causal processes. Consequently, (B) is independent of (A) in that (B) can be true even if (A) is false. Importantly, however, (B)'s being true is still *consistent* with (A)'s being true. Indeed, if there is no distinct causal process called "natural selection" that *could* be cited by evolutionary explanations, then that is a good reason for believing that the theory of natural selection does not explain by citing causes. In the end, neither (A) nor (B) will be appropriately tested by the kind of empirical test of epiphenomenalism envisioned by S/S. If one adopts (B), then this explanatory doctrine does not require any further ontological commitments of the kind involved in epiphenomenalism. Alternatively, if one adopts (A) then one is committed to a form of epiphenomenalism, but natural selection will, according to Matthen and Ariew (2009), always fail the manipulability tests of causation advocated by S/S.

In what follows we will defend (B), WALM's explanatory doctrine, by first showing how this kind of statistical explanation can be autonomous of considerations of causation. We will argue for this claim by considering two analogous cases of autonomous statistical explanations:

Galton's explanation for the reversion to mediocrity and the use of Boyle's law to explain the diffusion of gases. We will then argue that the modern genetical theory of natural selection also provides autonomous statistical explanations.

**5. Galton's "statistically autonomous" explanation.**

In order to illustrate the difference between autonomous statistical explanations and causal explanations we will first present Francis Galton's explanation of "reversion to mediocrity". One reason Galton's example is important to consider is that the methodology behind the modern genetical theory of natural selection has its roots in Galton's statistical methodology (Fisher, 1953).

Like Weismann's mice, Galton's case involves inheritance. Importantly, however, Galton's phenomenon to be explained is quite different from Weismann's. Weismann seeks to understand the determinants of the causal processes of inheritance. Galton, in contrast, is interested in explaining another feature of inheritance: the stability of variation for quantitative features of populations both within a single population (over time) and across multiple diverse populations.

For many human characteristics, offspring tend to resemble their parents. For example, exceptionally tall parents tend to have tall children. Yet, by and large, the lineages that begin with an exceptional ancestor do not end up with exceptional descendants. Rather, the opposite occurs, the descendants tend to "revert" to the population average or mean. This is a strange phenomenon; it is as if inheritance carries information about the whole population and not just information about the parents. As Galton put it, the next generation is "meaner" than the previous one. Put another way, although each generation will have a variety of, say, tall and short individuals, the distribution of these individuals in the population tend to be constant from generation to generation. Galton writes: "The processes of heredity are found to be so wonderfully balanced, and their equilibrium to be so stable, that they concur in maintaining a perfect statistical resemblance…" (Galton, 1877, 1).

As an example, Galton asks us to suppose there is a population of 100 giants and 100 medium men and the latter are "more fertile, breeding more truly to their like, being better

fitted to survive hardships, &c" (p. 2) [**Andre: is &c correct in that quote??**]. The result is that the medium men will leave a larger proportion of their progeny than that of the giants and, hence, we would expect "there would be fewer giants and more medium-sized men in the second generation than in the first". But, that's not what is observed in the census. Instead "the giants and medium-sized men will, in the second generation be found in the same proportions as before". That's the phenomena to be explained, called "reversion". Galton expresses it this way: "The question, then, is this: How is it, that although each individual does not as a rule leave his like behind him, yet successive generations resemble each other with great exactitude in all their general aims?" (p. 2). Despite selection favoring the medium sized men the proportion of all sizes remains the same from generation to generation. The phenomenon to be explained is the stability of population's distribution of variation despite character variation and selection over some of those variants in the population.

Galton borrowed statistical data from his contemporary, Aldolphe Quetelet, to illustrate this intergenerational stability. Galton's aim was to show that regardless of the population under study, the differences in the quantitative characters, in this instance height, conforms to a single statistical law, the "law of derivation" which Galton demonstrates to be graphically represented as a bell-shaped "curve of error" (Galton eventually renames it the "Normal curve").

According to Quetelet (following LaPlace), a curve of error is what you would expect to see if individuals in a very large population shared some common causal features—even if these features were not experienced by all individuals or were not experienced all in the same way. The mean (represented by the top of the curve) represents the results of underlying "constant" causes acting on each of the individuals in the population. The dispersion around the mean represents "error" or evidence of "accidental causes" that affect each individual in numerous ways, but the overall effect is to perturb development of the population character away from what would have happened had the constant causes acted alone. What this statistical technique shows is that Quetelet was interested in discovering the causal factors acting on individuals that determine the mean and variance for a feature within a population. As Elliott Sober puts it, the point of Quetelet's explanatory techniques was to "see through"

variation in the population in order to discover hidden invariances, possessed "by each individual organism" (**DATE?** 370).

In contrast, Galton viewed the significance of a population conforming to the curve of error very differently. Rather than trying to identify invariant tendencies of individual organisms, Galton was trying to identify the invariances in the statistical properties at the level of the entire population (**CITE ELLIOTT!!! CORRECTLY!**). The phenomenon of reversion is the entirety of the curve of error regenerating itself generation after generation regardless of the fact that for individual organisms, offspring tend to resemble their parents.

So, we have a point of contrast in the explanandum. Quetelet wants to discover the causal factors that determine why a feature in a population exhibits a particular mean and error, despite variation among individuals. In contrast, Galton wants to explain why, despite the general tendency of offspring to resemble their parents, features across all human populations conform to the curve of error *across generations*—i.e. the normal curve is intergenerationally stable. In addition to this difference in target explananda, it is also important to notice the difference in Galton's and Quetelet's use of statistics within the *explanans*. Namely, it is important to see what Galton did not do in utilizing statistical laws to explain reversion: appeal to causality.

Quetelet believed that a large-scale distribution pattern, such as a stable average or a bell-shaped curve, is what we would expect to see if individuals in a very large population shared some common causal features. Quetelet's technique was a primitive form of what we now call an "analysis of variance" or an ANOVA. Quetelet demonstrates a practical application of his causal interpretation of statistical distributions in his attempts to explain a surprising worldwide sex ratio favoring boys that appeared in censuses of over 14 million people. The first step was to distribute the census information of the whole population into groups according to a variety of categories. Quetelet then looked for differences between the averages of the whole and averages of various subsections. He eventually settled on a conjecture, that the cause of the skew of sex ratios favoring boys is the difference of ages between males to females.[6] In this way, Quetelet used statistical analysis to try to discover the cause of the explanandum.

[6] It is this work that Darwin references in his notebook entries on Quetelet.

But, this is not what Galton did. Galton did not interpret the statistical distribution as favoring a non-probabilistic causal hypothesis. The question concerning reversion to mediocrity was to explain the stability of variation (specifically the Normal distribution) in a population over generations despite inheritance mechanisms that are expected to change the contours of the statistical distribution. As Sober notes, "For Galton, variability is not to be explained away as the result of interference with a single prototype. Rather, variability within one generation is explained by appeal to variability in the previous generation and to facts about the transmission of variability" (Sober, 2006, 342-43).[7] Sober recognizes the key point. The explanation for a complex inheritance pattern, reversion to mediocrity, is explained by certain facts. For Galton, there is no presumption that these facts are causal or otherwise non-probabilistic. So whereas Quetelet assumed the explanatory facts had to be causal facts, this is precisely what Galton did not do—for Galton facts about the deductive properties of statistical distributions could provide a sufficient explanation.

This discussion of what Galton did not do helps us understand why Weismann's mice experiments are irrelevant to the kind of autonomous statistical explanation provided by Galton. In explaining the large-scale regularity characterized by a stable Normal curve, Galton would have no need for the sort of experimental manipulations involved in Weismann's mice experiment. It would do no good for Galton to trace out or hold fixed any of the factors that are involved in the life histories of any individuals, including their development or causal processes involved in their inheritance. Therefore, the kinds of "holding fixed" experiments on individuals within the population advocated by S/S are irrelevant to the kind of explanation Galton sought.

Galton explained his target explanandum by appealing to idealized statistical "laws". The structure of the explanation is as follows. First, he made idealizing assumptions about the distribution of quantitative frequencies and their independence—that is, he assumed that the traits of interest approximately conformed to the Normal curve (Hacking, 1990). These idealizing assumptions allowed Galton to derive—from a mathematical representation of the parameters that determine a Normal curve—that in a second generation there will be (ideally)

[7] It is also important to notice that the facts about the transmission of variability that Galton appeals to are *statistical facts* that follow from the Normal curve—they are not facts about the causal processes operating within the population.

a Normal curve of the same mean and variance. That is, Galton's explanation appeals to the following statistical fact: a normal curve and variation in a population deductively entails a normal curve of the same mean and variance in later generations. In addition, these derivations showed why the exceptional members of the second generation will not necessarily be descendant from the exceptional members of the previous generation.

In his discussion of the nature of Galton's explanation, Hacking calls it "autonomous". It is autonomous, according to Hacking, because the nature of the phenomena to be explained calls for a general law. Hacking writes: "Galton wanted to explain what he believed were curious phenomena of a thoroughly regular and law-like sort, about the distribution of hereditary genius in gifted families" (Hacking, 1990, 182). Galton's target explanandum is a highly general statistical regularity—and this kind of explanandum requires a particular kind of explanans. Galton's explanation is an autonomous *statistical* explanation because in order to explain the population-level regularity, Galton appeals to the deductive consequences of laws that involve the statistical properties of the population; i.e. the mean and variance of the normal curve. As Hacking points out, "The typical [statistical] laws are those which most nearly express what takes place in nature generally; they may never be exactly correct in any one case, but at the same time they will always be approximately true and *are always serviceable for explanation*" (Hacking 1990, 181, emphasis added). The structure of this kind of autonomous statistical explanation involves two general steps: (1) assume the population conforms to the properties of an idealized statistical distribution (e.g. the normal curve), then (2) deduce the explanandum from statistical regularities that "govern" statistical distributions.

There is another key lesson to be learned from the contrast between Galton's and Quetelet's respective uses of statistics in explanation. Quetelet is using statistics to confirm causal hypotheses that, in turn, explain trends. This use of statistics to uncover causes, such as the use of ANOVA analyses, is an important and legitimate statistical technique that can be used to determine the cause of an event.[8] For Galton, however, the statistical generalizations are sufficient for explanation—there is no need to make further assumptions about the reducibility of the statistical generalizations to any causal process. The lesson is that statistical

[8] Of course, an ANOVA analysis can also potentially be used to establish type-level causal claims.

generalizations can serve both purposes.[9] Therefore, to point out that some uses of statistical information aim to reveal underlying causal processes and mechanisms is not an objection against WALM's explanatory doctrine. WALM's view does not entail that statistical information can never reveal information about causal processes and mechanisms; rather, they argue that the modern genetical theory of natural selection provides autonomous statistical explanations (of the kind provided by Galton).[10]

Most importantly, however, Galton's case shows how a statistical model can be used to provide an autonomous explanation independent of any claims about what causal relationships are present in the world. At this point we should recall the distinction between the claim (A) that natural selection is not a cause and the claim (B) that the modern genetical theory of natural selection explains by citing a statistical model. The important lesson is that "selection and drift can be explanatory...without being causes of evolutionary change" (Matthen and Ariew, 2009, 16). And this is precisely the reason why WALM's explanatory doctrine does not require any kind of epiphenomenalism. *The explanation does not need to cite causes*.

This shows that there are at least two types of explanations that appeal to statistics. The first kind uses statistics to uncover the most likely cause of the target explanandum and thereby provide a causal explanation. The second kind of explanation uses statistics to provide an autonomous statistical explanation that is independent of causal claims. Which kind of explanation is preferable will depend on the target explanandum. As we will argue below, autonomous statistical explanations are often appealed to when the explanandum is a large-scale regularity that occurs within a "complex system".

**6. A second example: the statistical explanation of the diffusion of gases.**

WALM's prior use of the analogy with the kinetic theory of gases is worth elaborating as a second case study of an autonomous statistical explanation. Our discussion of gases largely

[9] It is important to note that both kinds of explanation—statistical and causal mechanical—are important to evolutionary biology. Thanks to Jim Lennox for suggesting that we emphasize this point—see Lennox and Wilson (1994) for a discussion of the causal mechanisms involved in the Darwinian struggle for existence. The key question we are raising in this paper is how are these styles of explanation different and in what contexts do they apply.

[10] Lewontin often criticizes population genetics for failing to adequately reveal causal processes and mechanisms for natural selection. He is neglecting the alternative scientific virtues that statistical idealizations possess. For a more complete account (but not complete enough), see Weisberg (2007).

follows Woodward's exposition in his Stanford Encyclopedia of Philosophy entry, “Scientific Explanation”, and his book, *Making Things Happen* (Woodward, 2003, 2011).[11] Evolving systems are, like the diffusion of gases, what Woodward calls "complex" or "higher order" systems.[12] In such complex systems, we often find large-scale regularities at the level of the population that do not hold at the level of individuals. Identifying this kind of target explanandum is key to understanding the context in which autonomous statistical explanations are intended to apply. What calls out for an explanation is how the large-scale regularity emerges from the relative chaos that describes the actions and interactions of the individual constituents. We contend that, in many cases, the deductive consequences of a statistical model are sufficient to explain such large-scale regularities in complex systems.

Woodward's discussion of gas behavior intends to demonstrate shortcomings with Salmon's "causal mechanical" account of explanation, whereby events are explained by demonstrating how the event fits into a causal nexus of processes and interactions leading up to the event occurring. One important parallel has to do with why statistical explanations are sometimes preferable to causal process explanations in the case of complex systems. To us, this is the relevant similarity between natural selection, the kinetic theory of gases, and Galton’s statistical explanation that should drive the analogy that supports WALM’s explanatory doctrine: in each case the target explanandum is the existence of an ubiquitous and stable population-level pattern. And, in each case the explanans is “statistically autonomous” involving two general steps: assumptions that allow for the use of an idealized statistical model and then deduction from that model. Finally, as with all statistically autonomous explanations, this deductive procedure is sufficient for explanation—no further appeal to causal processes is necessary.

[11] S/S cite Woodward’s work on explanation to support their view that WALM advocates a wrong-headed epiphenomenalism. But, to us, Woodward’s work on complex systems better suits our point, that statistically autonomous explanations are sometimes better than individual-level causal explanations for complex systems.

[12] We will use the analogy with the explanation of diffusion of gases to highlight some important similarities with the explanations provided by the modern genetical theory. Namely, the target explanandum is a large-scale regularity, the statistical explanation is independent from the causal explanation, and the statistical explanation is sometimes preferable for similar reasons. Of course, there are also several disanalogies between the two cases (e.g. see Fisher, 1930). Moreover, we do not intend to claim that all equilibrium explanations are instances of statistical explanation--only that there are important parallels between these two kinds of explanation.

Suppose we want to explain the overall change of equilibrium pressure for a mole of gas placed in a container and allowed to expand in volume while holding the temperature fixed. Salmon's causal mechanical account of explanation would prescribe explaining by tracing out the causal processes and interactions. Presumably the spatio-temporally connected causal processes and interactions take place at the individual molecular level and the explanation would cite each of the $6 \times 10^{23}$ individual-level causal processes. But there is another way to explain the overall change of equilibrium pressure that involves citing one of a number of standard statistical mechanical laws. And here we can see the same two-step explanation found in Galton.

Accordingly**,** the first step is to make generalized assumptions about the distributions of molecular velocities and the forces involved in their collisions. The assumptions (along with other laws of mechanics) allow us to derive and solve a differential equation for a mole of gas: PdV + VdP = RdT. The behavior of gases is then explained by *citing deductive consequences of this differential equation*.

This statistical explanation is distinct from the explanation that cites the individual level causal processes. Indeed, it "abstracts radically from the details of the causal processes involving particular individual molecules and instead focuses on identifying higher level variables that aggregate over many individual causal processes and that figure in general patterns that govern the behavior of the gas" (Woodward, 2003, p. 354). This is, for WALM, the relevant similarity that drives the analogy with natural selection. Accordingly, the modern genetical theory of evolution explains without tracing the causal processes and interactions represented by the multitude of individual life histories (life, death and reproductive events). Instead, modern evolutionary theory explains by abstracting radically from the details of the causal processes involving particular individual life histories. Indeed, like Galton's explanation of reversion and the explanation of the diffusion of gases, the explanations provided by modern evolutionary theory do not refer to causal processes at all—even if we consider causal processes at the level of the population. Evolutionary explanations of this kind focus on identifying higher-level statistical variables that aggregate over many individual causal processes and that figure in general patterns that govern evolutionary events.

Since neither the statistical treatment of gas nor the modern genetical evolutionary theory cite any causal processes, Salmon's "causal mechanical" account fails to account for them. In addition, Woodward argues that there are many reasons why one may prefer the "higher-level" (i.e. in our terms the autonomous statistical) explanation to the causal mechanical explanation. For one thing, the computation involved in tracing each of the individual molecular processes and interactions is impossible. Even if it were possible, the computation would be far too complex to be followed by the human mind. Still, in the case of natural selection sometimes tracing individual life histories down a lineage is possible and in some cases not too complex to be followed by a human mind (or computer).

However, there is a more important reason for favoring the statistical treatment of gas over the causal mechanical explanation—and this reason clearly applies to the case of evolutionary systems. The statistical account of gases' macro-behavior provides us with extra explanatory information that the causal mechanical explanation does not provide. Namely: "There are a very large number of different possible trajectories of the individual molecules in addition to the trajectories actually taken that would produce the macroscopic outcome—the final pressure P2—that we would want to explain" (Woodward, 2011). The content of the last quote parallels Elliott Sober's claim that, "Where causal explanation shows how the event to be explained was in fact produced, equilibrium explanation shows how the event would have occurred regardless of which of a variety of causal scenarios actually transpired" (Sober, 1983, 202).[13] However, nothing in Sober's account of equilibrium explanation, nor anything in Woodward's account of gas laws commits either of them to defending epiphenomenalism. The important parallel is that statistically autonomous explanations demonstrate that *no matter what the arrangement of individual-level causes are, a particular ensemble level trajectory is highly likely*.[14] In Galton's case, it is the stability of the normal curve. In the case of thermodynamics, it is the trajectory in which entropy increases. In the case of natural selection,

[13] In a similar spirit, Jackson and Pettit (1992, 177) claim that, the macro-level explanation tells us that, "if the actual history described by the microcausal explanation had not obtained, the explanandum would still have occurred."

[14] How likely depends on how well the idealized statistical model reflects the real world conditions. For instance, how likely height in human populations will revert depends on how well the distribution of height in human populations approximates the curve of error.

it is the trajectory in which the fitter trait type increases. These trajectories are detectable only at the ensemble level.[15]

We argue that the explanations provided by the modern genetical theory of natural selection do not cite detailed information about the lives, deaths, and reproductive events of individuals because the *explanandum* is the highly likely trajectory at the level of ensembles in which fitter trait types increase.[16] The general theory of selection explains by providing only the general conditions for adaptive evolution. What is more, since the modern genetical theory of natural selection is meant to explain most (or all) instances of organic evolution, natural selection explanations are able to apply to the conditions common to all life forms, from prokaryotes to eukaryotes, from Venus flytraps to mammoths (Ariew, 1998; Matthen and Ariew, 2002). We do not intend to claim that one type of explanation, causal explanations or autonomous statistical explanations, is always better than the other. In addition, we are not claiming that there isn't a causal story to tell about how the confluence of a variety of life histories lead to the evolution of a particular population. Our claim here is only that causal historical explanation and statistically autonomous explanations are distinct and epistemically irreducible forms of explanation (Ariew, 1998; Sober, 2003).

**7. Distinguishing Two Theories of Evolution by Natural Selection.**

So far we've articulated two cases, diffusion of gas and reversion to mediocrity, in which statistics can explain without citing causes that determine any particular instance of the general phenomena that constitutes the explanandum. What makes the statistical explanation appropriate is that the phenomena to be explained are "large-scale regularities" that arise out of "complex systems". The point of our discussion of these cases is to defend WALM's explanatory doctrine by showing how this kind of autonomous statistical explanation is possible by comparing it with other statistical explanations.

As S/S correctly point out, a central motivation for WALM's view is to provide an alternative to Sober's account of evolution as a "theory of forces" whereby the "causes of evolution behave in some ways like Newtonian forces. If two forces promote the evolution of a

[15] Thanks to an anonymous reviewer for suggesting this to us.
[16] Thanks to an anonymous reviewer for suggesting this to us.

trait, it will increase in frequency at a faster rate than if just one of them were in place" (Shapiro and Sober, 2007, 249). On this framework, drift, mutation, migration, and matting patterns are possible causal forces of evolution. In contrast, WALM argue that statistical models provide a more appropriate framework upon which to view the distinction between selection and drift. Rather than a sum of forces, it is better to view selection as a statistical consequence of variation in trait fitness (defined as a statistical distribution of types) and drift as sampling error.

So which view, a theory of forces or a statistical theory, is a better characterization of how natural selection explains large-scale regularities at the level of ensembles? The answer depends on which version of the theory of natural selection is under consideration. In order to settle which theory is being used in a particular case, we must pay close attention to what we are trying to explain and what sort of explanans would satisfy it. Unfortunately, a lot of confusion about the nature of natural selection explanations arises from the failure to distinguish Darwin's version of natural selection from the modern genetical theory of natural selection first developed by mathematicians associated with population genetics in the 20th century.

For Darwin, ensembles change because each individual, regardless of their place in the economy of nature, is governed by a natural tendency to reproduce at a high geometrical rate of increase and external forces that check population growth. This is the basis of his universal struggle for existence, which is the natural analogue to an artificial selector. As a result, Darwin's theory explains changes in *lineages*—i.e. differences in the number of offspring of particular individuals within the population. This kind of theory explains ensemble change by citing the aggregation of difference in the reproduction of individuals. Darwin's theory does not cite (or involve) the statistical properties of the population; e.g. the mean and variance of the distribution of trait types. As a result, Darwin's theory cannot explain changes in the relative frequencies of trait types since this explanation requires reference to the statistical properties of the population.

In contrast to Darwin's theory, the genetical theory of natural selection operates under a distinct understanding of ensemble change: ensemble changes (i.e. large-scale regularities) are a mathematical consequence of a model equation that refers to the *statistical properties* of

a population. This kind of statistical explanation can explain changes in the relative frequencies of trait types by citing statistical properties of the population—e.g. its mean and variance. In the modern genetical theory, the condition of a struggle for existence is replaced by a parameter, the reproductive rate, that appears as a scalar quantity that can be calculated without having to determine the natural properties, inherent tendencies, or external interfering forces acting on individuals (Ariew and Lewontin, 2004).

It is important to bear in mind that the statistical method at the basis of the modern genetical theory was motivated by the need to "re-animate" evolutionary theory as Darwin's version was facing insurmountable difficulties. For one, Darwin's theory relied on the universality of the struggle for existence as the lawful outcome of external checks against an inherent tendency to reproduce at a high rate. He relied on it because he believed, consistent with the Newtonian philosophy of science espoused by Herschel and Whewell, that a good theory of ensemble changed required the existence of a real cause to instigate change. But, it quickly became apparent among demographers and naturalists that there is no fixed universal reproductive rate. Even under Malthus's logic—from which Darwin derived inspiration—a type's reproductive rate is proportional to its mortality rate. Hence, a high reproductive rate, geometrical, or otherwise, is contingent upon a high mortality rate, and not fixed independently as Darwin purported it to be. Consequently, Darwin's "struggle for existence" appears to be more metaphoric than the inevitable consequence of a fixed law of population growth.[17]

Secondly, in *On the Origin of Species*, Darwin provides insufficient answers to questions about the dynamics of an evolving population that vary along factors other than individual differences. For instance, Fleeming Jenkin asks in his seminal review of *On the Origin of Species*: what happens when the population is large, mating is effectively random, and the variant most suited for the struggle for existence is rare? What will insure that the rare variant is not 'swamped by number'? (Gayon, 1998, 94). Jenkin's objections strike at the heart of Darwin's Newtonian fixed law approach because by focusing on the causal contribution of individuals to effect ensemble change Darwin was neglecting the important role the structure of population-

[17] Indeed, based on human demographic statistics, theorists as early as the 1830s thought "struggle" and "war" were exaggerated terms to describe the conditions of dense populations.

level variation plays in the explanation of complex ensemble change. This is especially problematic for Darwin's theory that required variation to be sustained in a population while the struggle for existence is supposed to act to eliminate it. In the face of these objections, Darwin's theory needed an overhaul that replaced the focus on the struggle for existence involved in individual lineages with an alternative that could account for the importance of the population-level distributions of variants.

The solution to such problems came in the earliest part of the 1900s in the form of a mathematical equation formulated independently by British mathematician G. H. Hardy and German physician, W. Weinberg. They modeled the conditions upon which Mendelian inheritance rules allow for the maintenance of genetic diversity. The model is a statistical idealization; it operates under random mating (infinite population size), but it is also approximated by natural populations. The seminal investigations of R. A. Fisher, J. B. S. Haldane and S. Wright contributed to the development of the mathematical framework that describes the modern genetical theory of natural selection. This theory was able to unify the Mendelian inheritance model with Galton's biometry. Moreover, this theory is able to explain changes in the relevant frequencies of trait types by citing statistical properties of the distribution. This is the "modern genetical theory of natural selection" to which WALM are referring in the defense of their explanatory doctrine.

In support of this interpretation, Margaret Morrison has demonstrated that Fisher's contribution to the development of the genetical theory of natural selection depended on modeling Mendelian populations in the same fashion as molecular models of statistical mechanics (Morrison, 2002, 64). This is one key feature that makes the modern genetical theory of natural selection so distinct from Darwin's version. For Darwin, populations are comprised of individual organisms. In contrast, on the statistical framework, populations are represented in terms of statistical distributions.

Therefore, while Sober's "theory of forces" is able to account for Darwin's strategy of explaining the dynamics of the economy of nature, it fails to capture what is distinctive about modern versions. These two theories operate under completely different characterizations of complex ensemble change (Ariew, manuscript). As a result, whereas Sober's theory of forces is

an adequate description of Darwin's theory, WALM's explanatory doctrine is required to understand how the modern genetical theory of natural selection explains large-scale statistical regularities.

In the 21st century, the statistical character of natural selection introduced by Fisher, Haldane, and Wright remains prevalent but the specifics of the modern genetical theory change. This is because nature is so varied there is likely no one "most general" theory of natural selection that is able to apply to every population (Ariew and Lewontin, 2004).[18] As a result, the modern genetical theory of evolution is, today, represented by a variety of statistical models, each invoking very general conditions of evolution: variation, inheritance, fitness differences (Ariew and Lewontin, 2004). Of course, current biological theorizing uses both Darwin's theory and the modern genetical theory—indeed, both kinds of explanation, causal mechanical and statistical are prevalent in biological theorizing. The important point to recognize is that they are distinct and irreducible forms of explanation.

**8. A Possible Objection: Are Statistical Laws Sufficient for Explanation?**

We have now defended WALM's explanatory doctrine by distinguishing autonomous statistical explanations from causal mechanical explanations and identifying the kind of explanatory context in which they apply. At this point, one might object that mere deduction from statistical laws is insufficient to provide an explanation. Indeed, the many critiques of Hempel's (1965) account of explanation have clearly established that mere deduction is insufficient for explanation. In response to this challenge, we argue that autonomous statistical explanations are not merely deductions, but are sufficient explanations when they are also able to provide counterfactual information that reveals the salient relationships of dependence.

Our emphasis on counterfactual information parallels Woodward's claim that, "[an] explanation must enable us to see what sort of difference it would have made for the explanandum if the factors cited in the explanans had been different in various possible ways" (Woodward 2003, 11). Although Woodward develops his account in terms of causal dependence, he suggests that perhaps not all scientific explanations are causal:

[18] Moreover, there is no necessary requirement that natural selection is statistical in this sense—Darwin's theory, for example, is not.

> One natural way of accommodating these examples is as follows: the common element in many forms of explanation, both causal and noncausal, is that they must answer what-if-things-had-been-different questions. When a theory tells us how Y would change under interventions on X, we have (or have the material for constructing) a *causal* explanation. When a theory or derivation answers a what-if-things-had-been-different question but we cannot interpret this as an answer to a question about what would happen under an intervention, we may have a noncausal explanation of some sort. (Woodward 2003, 221)

We retain Woodward's emphasis on providing information about counterfactual dependence relations without requiring that these be *causal* counterfactual relations. Statistical explanations are often sufficient, not merely because they allow us to deduce the explanandum, but because they also tell us about how things would have been different in various counterfactual situations. Namely, the statistical explanation tells us how the large-scale regularity would have been different if the statistical properties of the population had been different. In addition, autonomous statistical explanations can provide counterfactual information about what *is not* important for understanding the target phenomenon. Statistical explanations provide this additional information by showing us that within a certain range of possibilities, "the event would have occurred regardless of which of a variety of causal scenarios actually transpired" (Sober, 1983, 202). In the instance of natural selection explanations, the statistical explanation provided by the modern genetical theory show us that the large-scale regularities we want to understand would have occurred regardless of various changes in the detailed life histories of individuals within the population.

**9. Conclusion.**

S/S argue that WALM would have done better to support their explanatory doctrine by adopting an empirical demonstration of epiphenomenalism such as the one offered by Weismann in his mice experiment. We have shown that Weismann's mice experiments are ill-suited for WALM's purposes. WALM's explanatory doctrine does not require a defense of epiphenomenalism. Indeed, WALM's claims about how the theory of natural selection explains are independent of any metaphysical claims involved in epiphenominalism. After distinguishing these two positions, we have defended WALM's explanatory doctrine by arguing that the explanations provided by the modern genetical theory of natural selection are autonomous statistical explanations analogous to Galton's explanation of reversion to mediocrity and the

explanation of the diffusion of gases. A key distinguishing feature of this kind of autonomous statistical explanation is the nature of the target explanandum: these statistical models aim to explain large-scale regularities that arise from complex systems. Furthermore, we have bolstered the case for WALM's explanatory doctrine by distinguishing the modern genetical evolutionary theory, from Darwin's. Whereas Sober's theory of forces is an adequate description of Darwin's theory, WALM's explanatory doctrine is required to understand how the modern genetical theory of natural selection explains large-scale statistical regularities. Such autonomous statistical explanations are sufficient when they provide the right kind of counterfactual information about the occurrence of the explanandum.

**References**


Ariew, A. (1998).”Are Probabilities Necessary for Evolutionary Explanations?“. *Biology and Philosophy*, 13(2), 245-253.

Ariew, A. (2007). “Under the Influence of Malthus’s Law of Population Growth Darwin Eschews the Statistical Techniques of Aldolphe Quetelet”, *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 38(1), 1-19.

Ariew, A. (manuscript). *How Statistics Changed Natural Selection.*

Ariew, A. and Lewontin, R. (2004). “The Confusions of Fitness“. *British Journal for the Philosophy of Science*, 55, 347-363.

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

Fisher, R. A. (1953). The expansion of statistics. *Journal of the Royal Statistical Society: Series A,* CXVI, 1–10.

Galton, F. (1889). *Natural Inheritance.* MacMillan Press.

Gayon, J. (1998). *Darwinism's struggle for survival: heredity and the hypothesis of natural selection.* Cambridge: Cambridge University Press.

Hacking, I. (1990). *Taming of Chance.* Cambridge: Cambridge University Press.

Hempel, C. (1965). *Aspects of scientific explanation*. New York: Free Press.

Jackson, F., and Pettit, P. (1992). In defense of explanatory ecumenism. *Economics and Philosophy, 8*(1), 1-21.

Lennox, J.G., and Wilson, B.E. (1994). Natural selection and the struggle for existence. *Studies in History and Philosophy of Science, 25,* 65-80.

Matthen, M. and Ariew, A. (2002). “Two Ways Of Thinking About Fitness and Natural Selection“. *Journal of Philosophy*, 49(2), 55-83.

Matthen, M., and Ariew, A. (2009). Selection and causation. *Philosophy of Science, 76*, 201-224.

Morrison, M. (2002). Modelling Populations: Pearson and Fisher on Mendelism and Biometry. *British Journal for the Philosophy of Science*, 53(1), 39-68.

Northcott, R. (2010). Walsh on causes and evolution. *Philosophy of Science*, 77(3): 457-467

Nowak, M. (2006). *Evolutionary Dynamics: Exploring the Equations of Life.* Harvard University Press.

Reisman, K., and Forber, P. (2005). “Manipulation and the Causes of Evolution”, *Philosophy of Science 72*, 1113-1123.

Salmon, W. (1984). *Scientific Explanation and the Causal Structure of the World.* Princeton, N.J.: Princeton University Press.

Shapiro, L, and Sober, E. (2007). "Epiphenomenalism -- the Do's and the Don'ts." In G. Wolters and P. Machamer (eds), *Studies in Causality: Historical and Contemporary*. University of Pittsburgh Press, 235-264.

Sober, E. (1983) “Equilibrium Explanations”. *Philosophical Studies*, 43, 201-210.

Sober, E. (2003). "Two Uses of Unification" in F. Stadler, ed., *Institute Vienna Circle Yearbook*, Kluwer, 205-216.

Sober, E. (2006). ‘‘Evolution, Population Thinking, and Essentialism.’’ In E. Sober (ed.), *Conceptual Issues in Evolutionary Biology*, 3rd ed. Cambridge, Mass.: MIT Press.

Walsh, D. (2000). Chasing Shadows--Natural Selection and Adaptation. *Studies in the History and Philosophy of Biological and Biomedical Sciences*, 31, 135-53.

Walsh, D. M. (2007). The pomp of superfluous causes: The interpretation of evolutionary theory. *Philosophy of Science, 74*(3), 281-303.

Walsh, D. M. (2010). Not a sure thing: Fitness, probability, and causation. *Philosophy of Science, 77*(2), 147-171.

Walsh, D. (manuscript). Variance, Invariance, and Statistical Explanation.

Walsh, D. M., Lewens, T., and Ariew, A. (2003). “Trials of Life: Natural Selection and Random Drift“. *Philosophy of Science*, 69, 58-83.

Weisberg, Michael. 2007. “Three Kinds of Idealization.” Journal of Philosophy, 104: 639-659.

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

Woodward, J. (2011). "Scientific Explanation", The Stanford Encyclopedia of Philosophy (Winter 2011 Edition), Edward N. Zalta (ed.), URL = <http://plato.stanford.edu/archives/win2011/entries/scientific-explanation/>.