THE CAUSAL EFFICACY OF CONSERVATION ACTIONS FOR PRESERVING THE SHORT-TERM EVOLVABILITY OF WILD POPULATIONS

by

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ABSTRACT

Conservation biology has prioritized the conservation of genetic variation within wild populations as the principal conservation action for preserving their short-term evolvability through rapid environmental change. The amount of heritable variation, i.e., variation due to genetic factors, within populations is often regarded as the most important causal factor in the production of evolutionary change through natural selection, as well as the only causal factor that can be intervened on through conservation action in any significant or predictable way. However, I argue that the conservation of genetic variation should be expected to have rather low causal efficacy for preserving the short-term evolvability of wild populations. Specifically, I argue that the strategy for action, namely, the maximization of heritable variation, leading to that intervention faces serious theoretical, epistemic, and operational challenges due to our uncertainty about the future environments wild populations will encounter and our lack of knowledge about the precise genetic basis of traits expected to be under selection in the future. As a result, the specific intervention the strategy recommends—namely, the conservation of *standing genetic variation*—has at best an indirect and only weakly correlative connection to the causal factors involved in the production of evolutionary change through natural selection. Furthermore, I argue that an alternative intervention—namely, the conservation of *standing phenotypic variation*—should be expected to have comparably greater causal efficacy for preserving short-term evolvability. Specifically, this intervention serves to

maximize the effect of selection, while avoiding the challenges facing the maximization of heritable variation. As a result, the connection between the conservation of standing phenotypic variation and the causal factors involved in the production of evolutionary change through natural selection is much more direct and strongly correlative than the comparable connection between the conservation of standing genetic variation and the same causal factors.

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CHAPTER 1

INTRODUCTION AND BACKGROUND

Conservation biology has prioritized the conservation of genetic variation within wild populations as the principal conservation action for preserving the ability of wild populations to evolve in the short term in response to rapid environmental change. The amount of heritable variation, i.e., variation due to genetic factors, within a population is commonly regarded as the most important causal factor in the production of evolutionary change in response to selection. Moreover, the amount of heritable variation is often viewed as the only causal factor that can be intervened on through conservation action in any significant or predictable way. However, in what follows, I argue that the conservation of genetic variation, as a conservation action aimed at preserving the short-term evolvability of wild populations, should be expected to have rather low causal efficacy for achieving that end. Specifically, I show that the strategy of maximizing amounts of heritable variation within wild populations as a way to maximize the range of possible evolutionary outcomes in response to selection faces serious theoretical, epistemic, and operational challenges due to our uncertainty about the future environments wild populations will encounter and our lack of knowledge about the precise genetic basis of traits expected to be under selection in those future environments. More importantly, these challenges suggest that the connection between standing genetic variation, i.e., present amounts of overall genetic variation, and the causal factors involved in the production of evolutionary change through natural selection is at best indirect and only weakly correlative. Furthermore, I argue that an alternative intervention, namely, the conservation of standing phenotypic variation (i.e., present amounts of overall phenotypic variation), should be expected to have comparably greater causal efficacy for preserving short-term evolvability. Specifically, this intervention serves to maximize the effect of selection, while avoiding the challenges facing the maximization of heritable variation. The implication of avoiding those challenges is that the connection between standing phenotypic variation and the causal factors involved in the production of evolutionary change through natural selection is much more direct and strongly correlative than the connection between standing genetic variation and the same causal factors.

I begin here in Chapter 1 by presenting a theoretical account of the causal structure of evolutionary change through natural selection and identifying the causal factors involved in the production of evolutionary change in response to selection. Next, I unpack the concept of evolvability, and I show how evolution through natural selection and evolvability are central to the goal of conservation biology to secure the persistence of wild populations through rapid environmental change.

In Chapter 2, I explain, using the breeder’s equation from quantitative genetics as a theoretical model of the causal structure of evolution through natural selection, why a popular strategy of maximizing heritable variation within wild populations as a way to maximize the range of possible outcomes of evolutionary change in response to selection faces serious theoretical, epistemic, and operational challenges. As I show, these challenges are due primarily to our uncertainty about the future environments wild populations will encounter and our lack of knowledge about the precise genetic basis of traits that will be under selection in those future environments. Moreover, I show that, as a result of those challenges, the specific intervention that the strategy recommends—namely, the conservation of standing genetic variation—should be expected to have rather low causal efficacy for preserving short-term evolvability, as the connection between standing genetic variation and the causal factors involved in the production of evolutionary change in response to selection is at best indirect and only weakly correlative.

In Chapter 3, I show that an alternative strategy of maximizing the effect of selection through the maximization of phenotypic variation results in an intervention—namely, the conservation of standing phenotypic variation—with a much more direct and strongly correlative connection to the causal factors involved in the production of evolutionary change in response to selection. Specifically, I show that this strategy avoids the challenges facing the maximization of heritable variation and explain why, in virtue of avoiding those challenges, the conservation of standing phenotypic variation should be expected to be more causally efficacious than the conservation of standing genetic variation for preserving the short-term evolvability of wild populations through rapid environmental change.

1.1 Evolution through natural selection

The study of biological evolution through natural selection has afforded a rich theoretical understanding of the causal processes involved in the production of evolutionary change as a result of selective influences in the environment. Much of this theory has served to explain how observed patterns of biological variation in Earth's biota have come about through processes of inheritance, variation, and causal interaction with the environment. Investigations into these processes in turn have suggested how specific causal interventions may help secure the persistence of wild populations of Earth's biota through rapid environmental change due to global climate warming.

1.1.1 The causal structure of evolution through natural selection

The traditional Darwinian account of evolution through natural selection is typically formulated in terms of three principles that collectively describe the conditions under which evolutionary change through natural selection may be expected to occur, specifically, as a causal outcome of those conditions (Godfrey-Smith, 2007). For a single trait within a population, evolutionary change resulting from natural selection may be expected to occur when: (1) there is variation in the trait among the individuals within an initial generation of the population such that different individuals exhibit different variants of the trait; (2) there are differences in reproductive success among the individuals in the initial generation that depend causally on their having specific variants of the trait; and (3) the variation in the trait is heritable between successive reproductive generations such that individuals in the subsequent generation would resemble the individuals in the initial generation that enjoyed reproductive success (Godfrey-Smith, 2007; Lewontin, 1970; Maynard Smith, 1987).

The above formulation of the conditions for evolution through natural selection offers an implicit description of what evolutionary change consists in. Specifically, this formulation implies that evolutionary change consists in the intergenerational change in the frequencies of specific trait variants within a population. Note that those individuals enjoying greater reproductive success in an initial generation as a result of their having specific variants of a trait will contribute a greater proportion of offspring to the subsequent generation than will those individuals whose reproductive success may have been negatively affected as a result of having other trait variants. Provided that the variation in the trait under selection is assumed to be heritable, a greater proportion of individuals in the subsequent generation of the population will tend to resemble those individuals who enjoyed greater reproductive success.

An alternative formulation characterizes evolution through natural selection in terms of the functional roles of the entities involved. According to this formulation, evolution through natural selection involves two distinct types of functional entities: replicators and interactors (Hull, 1980). Replicators are entities responsible for the transmission of information across generations, while interactors are entities that interact with the environment through their properties such that there may be differential proliferation of replicators (Hull, Langman, & Glenn, 2001). The information transmitted by replicators pertains to the specific properties of interactors and in part determines those properties. Differences in the properties of interactors are what make differential proliferation of replicators as a result of environmental interaction possible. Evolutionary change is thus the result of the differential proliferation and extinction of interactors as a consequence of their interaction with their environment. Similar to before, what evolutionary change amounts to under the replicator – interactor formulation is a new generation of interactors with properties that differ on average from those in the initial generation.

There is some controversy over whether the replicator – interactor formulation of evolution through natural selection is in fact general, insofar as it explicitly requires more than one type of entity, whereas the traditional tripartite formulation does not seem to require any more than one (Okasha, 2006). However, it is worth noting that an analysis of evolution through natural selection in terms of the functional roles at play does not imply that a single type of physical entity may not perform multiple functions, in this case the functions of replication and environmental interaction. While the tripartite formulation may not explicitly require more than one type of physical entity, it does imply some means of inheritance (condition 3) and some means by which trait variants are causally responsible for differential reproduction (condition 2). To this extent, the functional roles of replication and environmental interaction may be seen as implicit in the tripartite formulation. If that is the case, then the replicator – interactor formulation of evolution through natural selection is not obviously any less general than the tripartite formulation.

Nevertheless, we may notice that neither of the above formulations identifies a specific means of inheritance nor a specific means by which having certain traits or properties is causally responsible for differential reproduction or replication. These omissions are due in part to the aim for generality with these formulations (Godfrey-Smith, 2007; Okasha, 2006). However, setting aside concerns about generality, we may note that for most familiar cases of biological evolution through natural selection, those functional roles are accounted for. In such familiar cases, the means of inheritance is primarily afforded by genes and other agglomerations of genetic material, e.g., segments of DNA and chromosomes (Hull et al., 2001). Meanwhile, biological individuals like organisms afford the means by which having certain traits or properties is causally responsible for differential reproduction or replication. Specifically, this means is afforded in virtue of individuals like organisms having those traits or properties and interacting causally with the environment through those traits or properties.

1.1.2 Quantitative genetics and the evolutionary response to selection

In addition to the above qualitative characterizations of evolution through natural selection, other approaches, such as those grounded in genetics, have sought to parameterize and quantify the causal factors involved in the production of evolutionary change as a result of selection (Falconer & Mackay, 1996; Hanski & Gaggiotti, 2004; Pigliucci & Kaplan, 2006). This has been one of the primary aims of the field of quantitative genetics.

Like Mendelian genetics, quantitative genetics rests on a distinction between genotype, phenotype, and the environment. A biological individual's genotype is its genetic constitution. In typical usage, the term 'genotype' may refer to an individual's entire genetic constitution (e.g., its entire set of genes) or its genetic constitution with respect to a specific set of phenotypic traits. An individual's phenotype comprises its (nongenetic) physical and behavioral traits through which the individual interacts with its environment (Taylor & Lewontin, 2017). Phenotypic traits include any morphological, physiological, and biochemical characteristics as well as any behavioral patterns and tendencies. In typical usage, the term 'phenotype' may refer to an individual's entire set of phenotypic traits or to a specific subset of traits. Meanwhile, the environment consists of all other (nongenetic) physical factors that bear causally on an individual's genetic and nongenetic properties.

Unlike Mendelian genetics, quantitative genetics focuses on the expression, variation, and inheritance of traits within populations. As a study of populations, quantitative genetics focuses less on how particular phenotypes arise in individuals and more on how phenotypes vary within populations as the result of various genetic and environmental factors. Moreover, as its characterization suggests, quantitative genetics focuses expressly on phenotypic traits that vary *quantitatively* within populations, as opposed to those that vary qualitatively (e.g., Mendelian traits such as human blood groups). Such quantitatively variable traits are generally known as *quantitative traits*. Quantitative traits include any phenotypic traits that are in principle quantifiable, such as the physical dimensions of morphological structures, rates of physiological processes, and patterns of behavior (Falconer & Mackay, 1996).

Quantitative traits are especially germane to evolution through natural selection. For one, most of the phenotypic differences among individuals within populations are differences in quantitative traits (Allendorf & Luikart, 2007; Falconer & Mackay, 1996). In addition, most of the traits that are likely to be under intense ecological selection, such as morphological traits and life history traits, are quantitative, rather than qualitative, traits (Falconer & MacKay, 1996).

A fundamental assumption of quantitative genetics is that an individual’s phenotype with respect to some quantitative trait is the cumulative result of the individual’s genotype (i.e., the genetic basis of the trait in question), the individual’s environment, and certain other interactions between the individual’s genotype and its environment (Falconer & Mackay, 1996). Each of these factors may influence the specific phenotypic value that is expressed in an individual in various qualitative ways and to varying quantitative degrees. Expanded to populations, this fundamental assumption amounts to the assumption that the total *variation* in a phenotypic trait within a population is the cumulative result of variation due to the genetic differences among the individuals within the population, variation due to different environmental influences, and variation due to other interactions and correlations between particular genotypes and environmental factors.

As a convention within quantitative genetics, the phenotypic variation in a trait within a population is typically expressed in terms of the *variance* in the values of the trait within the population. The variance in a trait is a statistical measure of how spread out trait values are on average from the mean value of the trait within the population. The more spread out trait values within a population, the greater the variance in that trait within the population. Following this convention, the total phenotypic variance in a trait within a population may be more precisely expressed as the sum of the variance in the trait due to genetic factors, V_G, variance due to environmental influences, V_E, variance due to gene – environment interactions, $V_{G\times E}$, and variance due to other gene – environment correlations, V_{G-E}:

$$V_P = V_G + V_E + V_{G\times E} + V_{G-E}. \tag{1}$$

To be specific, gene – environment interactions ($V_{G\times E}$) involve situations in which the effects that specific environments have on phenotype differ among genotypes. In contrast, gene – environment correlations (V_{G-E}) involve situations in which different genotypes influence individuals' exposure to specific environments (Dick, 2005).

The total variance in a trait due to genetic factors (V_G) can be further partitioned into contributions from additive genetic variance, V_A, dominance genetic variance, V_D, and variance due to nonadditive epistatic interactions among genes (i.e., nonadditive gene – gene interactions), V_I:

$$V_G = V_A + V_D + V_I. \tag{2}$$

Additive genetic variance (V_A) accounts for the additive, i.e., quantitative, effects of genes on total phenotypic variance. Additive genetic variance is the main source of genetic variance for quantitative traits (Falconer & Mackay, 1996). In contrast, dominance genetic variance (V_D) is that due to the *qualitative* effects specific genes have on trait values. Dominance effects primarily affect traits whose values vary discretely in a dominant – recessive pattern of trait expression (e.g., human eye color). For such traits, the specific value of the trait expressed depends on the presence of specific alleles (i.e., alternative versions of a gene). Generally, each allele corresponds to a specific trait value. Certain alleles are dominant with respect to others such that the presence of dominant alleles (almost) entirely determines the resultant trait value. Finally, variance due to nonadditive epistatic interactions among genes (V_I) accounts for any other *qualitative* effects of gene – gene interactions on total phenotypic variance.

On the basis of these partitions, it is possible to identify a quantity H^2 that signifies the proportion of the total phenotypic variance in a trait within a population that is attributable to genetic factors:

$$H^2 = \frac{V_G}{V_P}. \tag{3}$$

This quantity is called *broad-sense heritability*. Intuitively, broad-sense heritability signifies the extent to which a subsequent generation of a population may be expected to resemble an initial generation with respect to some trait. However, since the focus for quantitative genetics is primarily on quantitative traits and the corresponding additive effects of their genetic basis, a more useful quantity is the proportion of total phenotypic variance in a *quantitative* trait within a population that is due specifically to *additive* genetic factors:

$$h^2 = \frac{V_A}{V_P}. \tag{4}$$

This is called *narrow-sense heritability*. Conceptually, narrow-sense heritability is similar to broad-sense heritability, except that narrow-sense heritability signifies the extent to which a subsequent generation of a population may be expected to resemble an initial generation with respect to the value of some *quantitative* trait.

Note that the heritability quantity in either case has occasionally been mistakenly ascribed to traits themselves, as if heritability were a property of traits simply (Sarkar, 1998). However, it is important to stress that the heritability of any trait within a population is a function of the particular population the trait is in, since genetic contributions to total phenotypic variance depend on a population's particular genetic composition, as well as a function of the particular environment the population is in, since total phenotypic variance includes environmental contributions. In addition, the heritability quantity has occasionally been mistakenly interpreted as signifying the extent to which particular individuals' phenotypes are due to genetic, rather than environmental, factors (Lewontin, 1974). However, since heritability is a statistical property of populations, it will not in general be able to describe the extent to which any particular phenotype within a population is genetically based. So, for our purposes here, we recognize that heritability is a statistical property of populations and that specific heritabilities for traits within populations are functions of those specific populations and the specific environments those populations are in.

Using narrow-sense heritability, it is possible to express the expected magnitude of evolutionary change in the mean value of a quantitative trait within a population in response to selection acting on the trait in some initial generation. Specifically, for a single quantitative trait within a population, the expected evolutionary response to selection may be expressed as:

$$R = h^2 S, \tag{5}$$

where R denotes the evolutionary response to selection, h^2 denotes the narrow-sense heritability of the trait within the population, and S denotes the selection differential, i.e., the effect of selection in the initial generation. This expression is commonly known as the *breeder's equation*, so called because of its historical use by plant and animal breeders to predict the evolutionary outcomes of selecting particular phenotypes for breeding (Falconer & Mackay, 1996).

In the breeder's equation, the evolutionary response R signifies the magnitude and direction of change in the mean value of a trait between an initial and subsequent generation of the population. The evolutionary response may have either a positive or negative value depending on the direction of change from the original population mean, i.e., depending on whether the mean value of the trait in the subsequent generation is greater than or less than the original population mean in the initial generation.

The selection differential S may be expressed as the difference in the mean value of a trait in the initial generation resulting from selection:

$$S = (\overline{z_a} - \overline{z_b}), \tag{6}$$

where $\overline{z_a}$ denotes the mean value of the trait *after selection* and $\overline{z_b}$ denotes the mean value of the trait *before selection*, i.e., the original population mean. Like the evolutionary response, the selection differential may have either a positive or negative value depending on the direction of change from the original population mean as a result of selection.

Note that the evolutionary response R and the selection differential S are related by factor h^2, the narrow-sense heritability of the trait in question within the population. Accordingly, narrow-sense heritability may be interpreted as the extent to which the effects of selection in an initial generation may be expected to appear in the subsequent generation as an evolutionary response. Notice that if heritability is zero (i.e., if $h^2 = 0$), then there will be no evolutionary response in the subsequent generation, regardless of the magnitude of the selection differential in the initial generation. In other words, the trait mean will remain at the same value in the subsequent generation as it was in the initial generation *before selection*. While selection may nevertheless occur, the change in trait mean resulting from selection in the initial generation will not appear in the subsequent generation as an evolutionary change. On the other hand, if heritability is unity (i.e., if $h^2 = 1$), then the evolutionary response will equal the selection differential. In other words, the trait mean in the subsequent generation will be the same as the trait mean in the initial generation *after selection*.

The use of the breeder's equation and similar quantitative models of evolution through natural selection (e.g., Price's equation) for *predicting* specific instances of evolutionary change has been heavily criticized. Specifically, insofar as such models rely on statistical descriptions, they cannot be used to test specific causal hypotheses, such as specific evolutionary predictions, since no specific causal inferences can be made about how the statistical parameters were causally productive of a specific evolutionary result (Pigliucci & Kaplan, 2006). Nevertheless, despite these concerns, the breeder's equation provides a useful theoretical model for characterizing evolution through natural selection in terms of two relevant and important causal factors, namely, the heritability of traits within populations and the effects of selection on those traits. While its usefulness for making or testing specific predictions of evolutionary change may be limited, the breeder's equation offers a clear picture of a general causal structure for evolutionary change through natural selection. Such a causal characterization is possible in part because the individual influence of each causal factor on the evolutionary outcome of selection is theoretically distinguishable from the other (Northcott, 2005).

1.2 Evolvability

Recent work in evolutionary biology has explored the concept of *evolvability*, or as it is sometimes called *evolutionary potential* (Frankham, Ballou, & Briscoe, 2010; Pigliucci & Müller, 2010). Most simply parsed, evolvability signifies the ability to evolve, in the sense of being capable of the production of evolutionary change. However, the scope of the concept of evolvability spans a range of scales of evolutionary change, depending on the amount of time over which evolutionary change occurs and the amount of change that is involved. As a result, the concept of evolvability encompasses more specific notions of short-term, midterm, and long-term evolvability (Pigliucci, 2008).

Short-term evolvability concerns evolutionary change on the order of reproductive generations of populations. Such short-term evolutionary change typically consists of intergenerational change in the mean value of a phenotypic trait within a population as a direct result of selective influences acting on the trait in a single generation (Houle, 1992). As indicated above, the breeder's equation provides a useful characterization of evolutionary change at this scale. According to the breeder's equation, intergenerational change in the mean value of some quantitative trait within a population depends in part on the narrow-sense heritability of the trait within the population and in part on the effect of selection on the frequency distribution of trait values within the population. Accordingly, whether a population is able to produce evolutionary change with respect to some trait in response to selection depends on the amount of heritable variation (in this case, additive genetic variation) in the trait that is present within the population.

Differently, midterm evolvability concerns evolutionary change at the scale of metapopulations, taxonomic species, and lineages (Pigliucci, 2008). At these scales, evolutionary change consists of more significant changes to phenotype and often involves significant structural modification of existing characters, e.g., changes in flower color, leaf shape, wing shape, etc. and functional modifications to existing structures, i.e., new uses for existing structures (Wagner & Altenberg, 1996). Such evolutionary changes typically occur over many reproductive generations. Moreover, unlike short-term evolvability, which depends primarily on variation present within a population at a given time, midterm evolvability depends additionally on the *variability* of traits as a function of the genetic architecture underlying them (Kirschner & Gerhart, 1998; Pigliucci, 2008; Wagner & Altenberg, 1996).

The genetic architecture of traits refers to their genetic basis and the developmental pathways leading to the their expression in individuals (Pigliucci &

Müller, 2010). The structure of the genetic architecture, sometimes referred to as the genotype – phenotype map, constrains the extent to which the expression of traits may be influenced by genetic mutation or environmental factors (Pigliucci, 2008; Pigliucci & Müller, 2010). One example of such a constraint is developmental canalization, which serves to limit the effects of genetic mutations and environmental factors on the expression of phenotypic traits during biological development (Flatt, 2005; Hendrikse, Parsons, & Hallgrímsson, 2007). Midterm evolvability then turns on the ability of the genetic architecture to overcome certain standing biological constraints, such as developmental constraints, for producing phenotypic variation (Kirschner & Gerhart, 1998; Wagner, 2005; Wagner & Altenberg, 1996). Accordingly, midterm evolvability may be more appropriately viewed as a property of the genetic architectures characteristic of more inclusive biological entities (e.g., metapopulations, taxonomic species, or lineages) rather than as a property of populations.

Finally, long-term evolvability concerns large-scale evolutionary change on the order of clades and more inclusive taxonomic groups. Such large-scale evolutionary change generally involves significant biological modifications, such as the emergence of novel morphological structures, novel physiologies, and novel behaviors (Pigliucci, 2008; Pigliucci & Müller, 2010). Evolutionary change at this scale also includes major evolutionary transitions from less to more complex biological systems, such as the transition from unicellularity to multicellularity (Szathmary & Maynard Smith, 1995). Like midterm evolvability, long-term evolvability depends on the ability of biological systems to overcome certain standing biological constraints. For long-term evolvability, however, those constraints may be more fundamental with respect to the structure and function of biological systems. For instance, such constraints may pertain to the ways that biological information is stored, processed, and transmitted (Szathmary & Maynard Smith, 1995). Accordingly, long-term evolvability might be viewed as a property of biological systems in a more general sense, rather than as a property of populations or even genetic architectures.

Certainly, the study of the causal structure of evolution through natural selection and the exploration of the concept of evolvability have additional implications besides the accumulation of theoretical knowledge about those topics. In the next section, I show how evolution through natural selection and the concept of evolvability bear on the goals of conservation biology. Specifically, I suggest how an understanding of the causal structure of evolution through natural selection could inform strategies for securing the persistence of wild populations facing rapid environmental change.

1.3 Conservation biology

Conservation biology is a goal-oriented applied science driven by values (Barry & Oelschlager, 1996; Soulè, 1985). Practitioners of conservation biology employ scientific methods and apply scientific knowledge toward the general aim of conserving Earth’s biological features—including biological individuals, groups of biological individuals, biological systems, and biological processes.

In support of this aim, conservation biology espouses three core values: biological diversity, biological complexity, and biological evolution (Soulè, 1985). Biological diversity consists in the variety of biological features at all levels of biological organization and emphasizes the differences among those features. Biological diversity includes variety at the molecular level as well as variety at much larger scales, such as differences among ecosystems. Meanwhile, biological complexity consists in the details of how biological features are composed, the biological functions they serve, and how biological features relate to one another, e.g., the anatomical organization of morphological structures, the functions of physiological systems, and the stratification of trophic levels in ecosystems (Soulè, 1985). Along with biological evolution, these values are typically regarded within conservation biology to be good as ends in themselves. However, they are also mutually supportive of one another. For instance, biological diversity may provide variation for selection. At the same time, biological diversity itself is a product of biological evolution. Similarly, biological complexity may afford novel opportunities for evolutionary change, while it is also itself a product of biological evolution. Accordingly, the core values of conservation biology may be regarded to be good as means as well as ends.

1.3.1 Crisis, uncertainty, and the causal efficacy of conservation actions

It is often noted that conservation biology deals in crisis (Soulè, 1985). Threats to Earth’s biota are often time sensitive matters that demand quick action. Consequently, decisions about what specific conservation actions to take in particular situations must often be made while lacking relevant and possibly critical information about a situation at hand (Soulè, 1985).

It is generally the case that conservation actions are taken with intentions to achieve specific conservation goals. Typically, those goals are such that they serve to achieve or exemplify one or more of the core conservation values (i.e., biological diversity, biological complexity, and biological evolution). Specific goals may include the conservation of a particular biological group (e.g., a species or a population), a particular biological or ecological community, a particular biological or ecological system, or a particular biological or ecological process (i.e., in a particular place). Recommendations for specific interventions are generally grounded in a combination of theory about the relevant causal processes involved in producing an intended outcome and empirical data about the particular biological features being affected by an intervention (Murphy, 1990). In this way, theoretical and empirical knowledge are used in concert to determine specific conservation actions that will be *causally productive* of the intended result, or at least likely to be causally productive of that result.

However, for circumstances in which data are lacking, such as situations that demand action in the present on the basis of future threats, theory has a central role in identifying possible strategies for action. At the very least, theory may provide some idea about how the relevant causal factors involved may change or relate to one another over time. Yet, without all of the relevant data, especially data pertaining to future threats, theoretical reasoning may be limited in its ability to afford much epistemic confidence in the causal efficacy of specific conservation actions for achieving specific conservation goals, i.e., in the ability of those actions to be causally productive of those goals. That is, given a theoretical understanding of the relevant causal processes, practitioners may nevertheless be uncertain about the extent to which particular conservation actions may even be likely to be causally productive of an intended result. In that case, any confidence practitioners may have in specific interventions would be afforded almost entirely in virtue of the theoretical and practical merits of the specific strategies guiding specific conservation actions.

1.3.2 Environmental change and the persistence of wild populations

Studies of Earth's climate over the last millennium predict a rapid global warming trend for the foreseeable future (Cox, Betts, Jones, Spall, & Totterdell, 2000; Jones, Osborn, & Briffa, 2001). Evidence of landscape and ecological changes worldwide in response to climate warming in the last century suggests that rapid environmental change is not only an imminent threat, but a current threat to the persistence of Earth's wild populations (McCarty, 2001; Walther et al., 2002). Rapid increases in global temperatures are expected to increase the incidence and severity of certain ecological stressors, such as extreme weather, food or nutrient shortages, and outbreaks of disease (Freedman, 1995). These increases in the incidence and severity of ecological stressors are certain to have selective influences on wild populations worldwide.

In order to avoid extinction as a result of rapid environmental change due to climate warming, wild populations must be able to cope somehow with that change. In general, wild populations may cope with environmental change in two ways: by migrating or dispersing to other areas with more favorable environments (i.e., by shifting their geographic range) or by undergoing evolutionary changes *in situ* (i.e., in place) that might allow them to endure the selective influences brought about by environmental change (Davis, Shaw, & Etterson, 2005). In most cases, it is likely to be some combination of the two (Davis & Shaw, 2001). However, the loss and fragmentation of habitats due to climate warming and anthropogenic land use suggests that many wild populations may currently have limited opportunities for migration or dispersal, and those opportunities are likely to become even more scarce in the future (Hoffmann & Sgro, 2011; Opdam & Wascher, 2004). With opportunities for migration and dispersal limited, the ability of wild populations to evolve is likely to be a key factor in their ability to cope with environmental change, now and in the foreseeable future.

In wild populations, evolutionary change in response to environmental change has been shown to occur on contemporary time scales, i.e., over decades (Stockwell, Hendry, & Kinnison, 2003). Such timescales are on the order of reproductive generations for many of the wild populations of plants and animals of concern for conservation. However, the extent to which evolutionary change can serve as a means for coping with environmental change depends on the rate at which populations are able to produce that evolutionary change (Jump & Peñuelas, 2005). Specifically, the rate at which wild populations can produce evolutionary change must be able to at least keep pace with the rate at which the environment changes. Accordingly, the *amount* of evolutionary change that wild populations can produce in the short term, i.e., on the order of generations, is critical to their ability to persist through rapid environmental change.

We may thus see how an understanding of the causal structure of evolution through natural selection has important implications for the conservation of wild populations. Specifically, with wild populations facing rapid and possibly adverse environmental change due to global climate warming, ensuring that those populations are able to evolve in the short term at a high enough rate to keep pace with environmental change is critical to securing their persistence through that change. However, since we are generally uncertain about the details of the future environments wild populations will encounter, the extent to which we could expect specific conservation actions to be causally efficacious for maintaining high enough rates of evolutionary change will depend primarily on our theoretical understanding of how the relevant causal factors involved may vary with future environments and on the extent to which we are able to intervene on or affect those causal factors.

1.3.3 Traditional approaches to preserving evolvability

Traditional approaches to preserving the ability of wild populations to evolve in the short term, i.e., preserving their short-term evolvability, have focused primarily on the conservation of the genetic resources within populations (Frankel & Soulè, 1981; Frankel, Brown, & Burdon, 1995; Schonewald, Chambers, MacBryde, & Thomas, 2003). While early approaches called for the conservation of genetic resources in general, relying on measures of overall genetic variation such as allelic richness (i.e., the total number of gene variants at a specific genetic locus) to determine how much to conserve, recent approaches drawing from quantitative genetics have emphasized the causal roles of quantitative traits in selective ecological interactions and of additive genetic variation in the production of evolutionary change in response to ecological selection (Allendorf & Luikart, 2007; Charmantier, Garant, & Kruuk, 2014; Frankel et al., 1995; Frankham et al., 2010). Nevertheless, in Chapter 2, I show why the conservation of genetic resources is generally a poor strategy for preserving the short-term evolvability of wild populations and thus a poor strategy for securing the persistence of wild populations through rapid environmental change. Specifically, I focus on a popular strategy of maximizing amounts of heritable variation within wild populations. I show how this strategy faces serious challenges in light of our uncertainty about future environments and our lack of knowledge about the precise genetic basis of many of the traits expected to be under selection in the future. Additionally, I explain why the specific conservation action that this strategy recommends, namely, the conservation of standing genetic variation, should be expected to have rather low causal efficacy for preserving the short-term evolvability of wild populations.

CHAPTER 2

THE CONSERVATION OF GENETIC VARIATION FOR PRESERVING SHORT-TERM EVOLVABILITY

The heritability of quantitative traits, i.e., the proportion of their variation that is heritable, is commonly regarded as the most important causal factor in the production of evolutionary change in response to natural selection. Consequently, conservation efforts aimed at preserving the short-term evolvability of wild populations have focused on the maximization of heritable variation as a primary strategy for determining specific conservation interventions. In this chapter, I argue that the strategy of maximizing heritable variation for preserving short-term evolvability faces serious theoretical, epistemic, and operational challenges. In light of those challenges, I argue that the specific conservation action the strategy recommends, namely, the conservation of standing genetic variation, can have only an indirect and weak affect on the relevant causal factors involved in the production of evolutionary change in response to natural selection. Consequently, we should expect the conservation of standing genetic variation to have rather low causal efficacy as a conservation action for preserving the short-term evolvability of wild populations.

I begin by showing that the task of preserving the short-term evolvability of wild populations is a quantitative, rather than a qualitative, goal. I then show, using the breeder’s equation, how a theoretical analysis of the causal factors involved in evolution through natural selection suggests strategies for determining specific interventions for preserving short-term evolvability. I then explain how our uncertainty about future environments and about the precise genetic basis of quantitative traits pose challenges to the strategy of preserving evolvability through the maximization of heritable variation. I show how, despite those challenges, the strategy recommends conserving standing genetic variation as the primary conservation intervention for preserving the short-term evolvability of wild populations. However, I suggest that those challenges raise serious doubts about the causal efficacy of conserving standing genetic variation as a conservation action for preserving short-term evolvability.

2.1 Maximizing the range of possible evolutionary outcomes

The preservation of short-term evolvability in wild populations is considered a means for securing the persistence of those populations through rapid and possibly adverse environmental change. To that end, what is at stake is not that wild populations are able to evolve in a purely qualitative sense (i.e., that they are able to evolve versus unable to evolve), but that they are able to evolve such that their chances for persistence are as great as possible, i.e., are maximal, in those environments. Those chances are expected to be maximal when the possibilities for evolutionary change are maximal (Allendorf & Luikart, 2007; Frankham et al., 2010). Specifically, the more opportunities there are for evolutionary change, the greater the chance that an evolutionary change in response to selective influences in a specific environment might be conducive to a population’s persistence in that environment. Accordingly, securing the persistence of wild populations through the preservation of evolvability may be accomplished by maximizing the possibilities for evolutionary change. Understood in this way, the preservation of short-term evolvability in wild populations may be seen as a quantitative, rather than a qualitative, goal.

Of course, the preservation of short-term evolvability as a means for securing the persistence of wild populations faces one major epistemic obstacle: our uncertainty about the future environments wild populations will encounter. Due to this uncertainty, we do not know what specific selective influences future environments will have on wild populations nor which specific phenotypes will be conducive to population persistence in those environments. Since we cannot be sure of those details, the preservation of short-term evolvability must be undertaken in a general way, namely, such that the possibilities for evolutionary change are maximized over a range of unknown and possibly adverse environments that wild populations may encounter.

One way to maximize the possibilities for evolutionary change over a range of environments is to maximize the *range* of possible outcomes of evolutionary change that can be realized within the population for any single environment. Similar to above, the idea here is that the greater the range of possible evolutionary outcomes, the greater the number of distinct possible evolutionary outcomes, and in turn the greater the chances that the population is able to persist in unknown and possibly adverse environments.

2.2 Maximizing the evolutionary response to selection

Specific interventions aimed at preserving the short-term evolvability of wild populations must follow at least in part from a theoretical understanding of evolution through natural selection. Since the preservation of evolvability is a quantitative goal, it is natural to turn to a quantitative model of evolution through natural selection, such as the breeder's equation, to see how that goal could be achieved.

As presented in Chapter 1, the breeder's equation expresses evolutionary change in response to selection with respect to a single quantitative trait within a population as the algebraic product of the narrow-sense heritability of the trait within the population and the selection differential resulting from selective influences on that trait:

$$R = h^2 S, \tag{5}$$

where R denotes the evolutionary response to selection, h^2 denotes the narrow-sense heritability of the trait in question within the population, and S denotes the selection differential. Using the breeder's equation as a model of the causal structure of evolution through natural selection, we can see how specific conservation actions aimed at preserving the evolvability of wild populations may follow from an analysis of the causal factors involved.

According to the breeder's equation, evolutionary change with respect to a single quantitative trait within a population consists in an intergenerational change in the mean value of the trait within the population. This is the evolutionary response to selection, denoted by R in the equation above. The magnitude of the evolutionary response, i.e., how great of a change there is in the mean value of the trait between the initial and subsequent generations, is a function of two causal factors: the proportion of heritable variation in the trait in the initial generation, i.e., the narrow-sense heritability of the trait within the population (h^2), and the magnitude of the effect of selection on the trait in the initial generation, i.e., the selection differential (S). Recall from Chapter 1 that the heritability of a trait within a population signifies the extent to which the effect of selection on the trait in the initial generation should be expected to appear in the subsequent generation as an evolutionary change. The sign of the evolutionary response, i.e., whether the change is in a positive or negative direction from the original population mean in the initial generation, is determined by the sign of the selection differential, i.e., by whether the effect of selection is in a positive or negative direction from the original population mean in the initial generation.

At this point, we may notice that the range of possible evolutionary outcomes in response to selection can be maximized by maximizing the *possible magnitude* of the evolutionary response R. Specifically, by maximizing the possible magnitude of the evolutionary response, we also maximize the range of possible intermediate responses between no response and a maximum possible response, since all evolutionary responses of magnitude lower than the possible maximum remain possible evolutionary outcomes. As long as intermediate responses remain possible, maximizing the possible magnitude of the evolutionary response R in the breeder’s equation above will serve to maximize the range of possible evolutionary outcomes in response to selection.

Now, since an evolutionary response in a subsequent generation may be in either a positive or a negative direction with respect to the original population mean in the initial generation, we must be sure to maximize the possible magnitude of the evolutionary response for both directions of change. In other words, we must be sure to accommodate all possible selection differentials, positive or negative, such that selection in a positive direction results in a maximum possible evolutionary response in a positive direction while selection in a negative direction results in a maximum possible evolutionary response in the negative direction. So, to maximize evolutionary response in an appropriate way, we must maximize the range of possible values, positive and negative, that R can take. That is, we must maximize the possible magnitude of the evolutionary response for selection in both the positive and the negative direction from the original population mean.

The breeder's equation suggests three possible strategies for maximizing the possible evolutionary response to selection for a single quantitative trait within a population. In each case, the possible evolutionary response is maximized by virtue of maximizing the influence of one or both of the causal factors indicated in the breeder's equation.

One strategy is to maximize the proportion of heritable variation in the trait in question within the population, i.e., maximize the heritability of the trait. As suggested above, this would serve to maximize the extent to which selection on the trait in the initial generation of the population would result in a corresponding evolutionary change in the subsequent generation. The thought is that for any selection differential in the initial generation, positive or negative, a maximal heritability would afford a maximum possible evolutionary response to that selection in the subsequent generation in the same direction.

Another strategy is to maximize the possible magnitude of the effect of selection in the initial generation for both possible directions of selection (i.e., positive or negative S). This would amount to maximizing the range of possible values of the selection differential. The underlying thought here is that for a given heritability, some maximal selection differential in either direction would afford a maximum possible evolutionary response to that selection in the same direction. In other words, if the effect of selection in the initial generation were the greatest it could possibly be (without causing the population to become extinct), then the corresponding evolutionary response in the subsequent generation would be the greatest it could possibly be, for selection in either direction from the original population mean.

A third strategy is to simultaneously maximize both the proportion of heritable variation in the trait in question as well as the possible magnitude of the effect of selection on the trait. This combined approach would serve to maximize the possible evolutionary response to selection through both of the causal channels indicated above.

Of these three possible strategies, the third option clearly seems best, seeing as it would serve to maximize possible evolutionary response through both available causal channels, i.e., through both heritability and the selection differential. However, it appears that this view is not widely held within conservation biology. Indeed, the prevailing view within conservation biology seems to regard the first option, namely, the maximization of heritability, as the preferable strategy for maximizing possible evolutionary response and thus for preserving the short-term evolvability of wild populations. While it is not obvious that the maximization of heritability is a superior strategy for maximizing possible evolutionary response, the reasons for preferring it as a strategy over the others are actually quite clear, as I explain below.

Insofar as we are concerned with evolutionary change, which consists in change across generations, it seems obvious that we should focus on factors that make that intergenerational change possible at all. With respect to evolution through natural selection, that factor is the heritability of traits under selection. Consider that any selection acting on nonheritable trait differences will presumably not result in evolutionary change, since the effects of selection in the initial generation will not be heritable. Evolutionary change in response to selection is only possible when the variation in the trait upon which selection acts is heritable. In short, without heritable variation there is no evolution, even if there is selection. On its face, this seems to be a clear reason for why we should focus on the maximization of heritability as a strategy for maximizing the possible evolutionary response to selection.

There is additionally a reason why we might prefer the maximization of heritability over either of the other strategies for maximizing possible evolutionary response. Specifically, since we are uncertain about the future environments wild populations will encounter, it is not clear how we could intervene on future selection differentials in any significant or predictable way. While we may have some general idea of the future trajectory of environmental change, as long as we do not know the details of future environments, we cannot be certain about the specific selective effects those environments could have. Consequently, it seems that the maximization of the possible selection differential as a way to maximize possible evolutionary response is simply not an available strategy. Certainly, if we can have no significant or predictable effect on the selection differential, then we should prefer the maximization of heritability as a strategy for maximizing possible evolutionary response, provided that the maximization of heritability offers better opportunities for intervention.

So, as I understand the reasoning behind the prevailing view in conservation biology, the heritability of traits is not only the most important causal factor for producing evolutionary change in response to selection; it is also seems to be the only factor that would we could intervene on in any significant or predictable way, specifically, given our uncertainty about future environments.

In the next section, I show that the strategy of maximizing heritability faces serious theoretical and epistemic challenges. I suggest that these challenges raise doubts about the practical merits of maximizing heritability as a strategy for maximizing possible evolutionary response to selection in wild populations facing rapid environmental change.

2.3 Maximizing heritability

To see how heritability may be maximized, we refer to the formal expression for the narrow-sense heritability of a single quantitative trait within a population:

$$h^2 = \frac{V_A}{V_P}, \tag{4}$$

where h^2 denotes the narrow-sense heritability of the trait within the population, V_A denotes the additive genetic variance in the trait within the population, and V_P denotes the total phenotypic variance in the trait within the population. According to this expression, the heritability of a trait is equal to the proportion of heritable variation in the trait. Specifically, the heritability of a trait is proportional to the additive genetic variance in that trait, while it is inversely proportional to the total phenotypic variance in that trait. Accordingly, heritability may be expected to be maximal when either of the following holds: when additive genetic variance is maximal for a given amount of total phenotypic variance or when total phenotypic variance is minimal for a given amount of additive genetic variance. So, to maximize the heritability of a trait within a population, we may either try to maximize additive genetic variance in the trait in relation to the amount of total phenotypic variance, or try to minimize total phenotypic variance in the trait in relation to the amount of additive genetic variance.

Now, if we are sensitive to the normative constraints of conservation biology (presented in Chapter 1), then it is clear that the preferable course of action is to try to maximize additive genetic variance in relation to total phenotypic variance, rather then the alternative of minimizing total phenotypic variance in relation to additive genetic variance. Consider that this alternative would entail the elimination of (at least some) biological diversity through the reduction of phenotypic variation. Biological diversity, however, is a core value for conservation biology, and it is one of the features we are aiming to conserve in the first place. So, if we are sensitive to the normative constraints of conservation biology, then we should try to avoid any actions that would diminish biological diversity, such as the minimization of total phenotypic variance.

Furthermore, any reduction of phenotypic variation within a population would eliminate phenotypes that could possibly be conducive to the population's persistence in future environments. To see how, consider the following scenario. Suppose we decided to reduce the total phenotypic variance in some trait within a population by eliminating the most extreme phenotypes on either side of the population mean, leaving only intermediate phenotypes. Certainly, this may result in a higher heritability for the trait within the population, since total phenotypic variance may have been decreased in relation to additive genetic variance, provided that a significant amount of additive genetic variance was not also lost. However, note that under intense directional selection, which would favor one or the other extreme phenotypes, or under intense disruptive selection, which would favor both extreme phenotypes, the population, now containing only intermediate phenotypes, faces a greater chance of extinction than it would had the extreme phenotypes been retained. The reason for this is that extreme phenotypes would have been less negatively affected by selection and so could have enjoyed comparably greater reproductive success than the intermediate phenotypes, which would have been intensely disfavored by selection.

Thus, the minimization of total phenotypic variance would both undermine a core value for conservation biology, i.e., the value of biological diversity, as well as be counterproductive to the goal of securing the persistence of wild populations through unknown and possibly adverse environments. Notice that in a comparable scenario in which we decided to *maximize* additive genetic variance in some trait within a population by simply conserving the additive genetic variation that was already present in the population, we face no such problems. Specifically, since conserving additive genetic variation would serve to conserve existing biological diversity, rather than serve to reduce any amount of it, the normative constraints of conservation biology are not violated. Moreover, since conserving additive genetic variation does not on its face affect total phenotypic variation in any way, the chances of population persistence through intensely selective environments are unaffected with respect to what they would have been otherwise. Accordingly, for any quantitative trait within a population that could possibly be under intense selection in future environments, we should prefer to maximize the additive genetic variance in the trait, rather than minimize total phenotypic variance, in attempting to maximize the heritability of the trait within the population.

Nevertheless, while we may not be permitted under the normative constraints of conservation biology to decrease total phenotypic variance in order to maximize heritability, total phenotypic variance must still somehow be constrained if the maximization of additive genetic variance is to result in some maximal heritability. Note that while heritability is proportional to additive genetic variance, the two quantities are not strictly equal. Since heritability is a fraction of variances, it is not enough to try to maximize additive genetic variance by itself, i.e., without somehow constraining total phenotypic variance, in order to maximize heritability. Specifically, this is because additive genetic variance and total phenotypic variance covary. However, they do not covary linearly.

Consider that for quantitative traits, a single genotype generally corresponds to multiple phenotypes. Primarily, this is because of the influence of environmental factors affecting the expression of phenotype via the genetic architecture and during biological development. Recall from Chapter 1 that an individual’s phenotype is the cumulative result of the individual’s genotype, the specific environment the individual is in, and various interactions between the individual’s genotype and its environment. As environmental factors may vary, so may the influence of those factors on an individual’s phenotype. What this means is that for any two amounts of additive genetic variation in some trait within a population, the greater amount of additive genetic variation will generally correspond to a disproportionately greater amount of phenotypic variation. This is precisely because each genotype contributing to additive genetic variation may interact with the environment in various ways and to varying degrees. The result is that there may be a multitude of distinct phenotypes contributing to phenotypic variation that may differ as a result of environmental influences but may nevertheless be underlain by common genotypes (for the trait or traits in question). In other words, the covariation between additive genetic variation (i.e., additive genetic variance) and total phenotypic variation (i.e., total phenotypic variance) is nonlinear. Specifically, total phenotypic variation generally increases at a greater rate than does additive genetic variation.

The consequence of all this in terms of heritability and trait variances is that heritability generally correlates with additive genetic variance *inversely* (Hansen, Pélabon, & Houle, 2011). A high amount of additive genetic variance in a trait does not necessarily imply a high heritability. Moreover, increasingly greater amounts of additive genetic variance in the same trait do not necessarily imply increasingly greater heritability. Instead, greater amounts of additive genetic variance in a trait within a population will generally correspond to *lower* heritability for that trait within the population.

For the maximization of heritability, this means that trying to maximize additive genetic variance by itself, i.e.., without constraining total phenotypic variance, will not generally serve to maximize heritability. Again, this is because with high amounts of additive genetic variance, the proportion of total phenotypic variance due to environmental factors will be correspondingly high. This inverse covariation between additive genetic variance and heritability has been shown to hold empirically for many quantitative traits subject to strong selection in wild populations, for instance, life-history traits such as age of reproductive maturity and brood size (Merila & Sheldon, 1999).

The fact that the heritability of any trait within a population is a function of the environment the population is in invites an additional consideration. As we previously noted, we do not know the details of the future environments wild populations will encounter. Consequently, we do not know the extent to which future environments will influence the expression of traits within populations nor the extent to which the heritability of those traits may vary with those environments.

Consider that the contribution of environmental factors to the total phenotypic variance in a trait within a population will generally differ in different environments (Allendorf & Luikart, 2007; Falconer & Mackay, 1996; Frankham et al., 2010). In some environments, the environmental contribution to total phenotypic variance may be small compared to additive genetic variance. In these environments, the heritability of the trait in question may be high. However, in other environments, the environmental contribution to total phenotypic variance in the same trait may be much larger, while the amount of additive genetic variance is the same. In these environments, the total phenotypic variance in the trait may be much greater, although the heritability of the trait may be correspondingly much lower.

What this suggests is that maximizing the heritability of a trait in one environment in no way implies that the heritability of that trait will remain constant across different environments. With respect to maximal heritability, what is maximal in one environment will generally differ from what is maximal in other environments—even if the absolute amount of additive genetic variance is the same in each of those environments. In general, the greater the differences among environments, the greater the differences in heritability among those environments.

A further consideration concerns the fact that the heritability of a trait within a population *before* any selection on the trait occurs does not directly bear on the heritability of the trait after selection. However, we may note that it is the heritability *after selection*, rather than the heritability before selection, that matters most for the production of evolutionary change in response to selection (Heywood, 2005). Notice that the heritability of the trait within the population *after selection* is what determines the extent to which that selection event will result in a corresponding evolutionary change in the subsequent generation. Accordingly, it is the heritability of a trait *after selection* that we should want to maximize in order to maximize the possible evolutionary response to selection.

However, as we have repeatedly noted, we do not know the details of the future environments wild populations will encounter. Consequently, we cannot be certain about the specific selective effects future environments will have on wild populations. Certainly, we do not know what specific selection differentials will result from selection. In light of these considerations, it is difficult to see how we can maximize for any trait in any clear or direct way the heritability that is most relevant to the production of an evolutionary response to selection, considering the various ways and degrees to which heritability is influenced by environmental factors. Indeed, so long as we do not know the details of future environments, any attempts to maximize the heritability of a trait within a population *in the present* in order to maximize possible evolutionary response *in the future* can at best have only an indirect affect on the heritability that actually matters most for the production of evolutionary change in response to selection—namely, the heritability *after selection*.

Interestingly, one of the main reasons we might initially prefer the maximization of heritability over other strategies for maximizing possible evolutionary response is precisely our uncertainty about future environments. However, it is now clear how our uncertainty about future environments poses a challenge for the maximization of heritability just as well.

Nevertheless, we can concede our uncertainty about future environments and hope that the maximization of additive genetic variance by itself, i.e., without constraining total phenotypic variance, can confer a sufficiently high or sufficiently close to maximal heritability, such that the possible magnitude of the evolutionary response to selection is sufficiently great or sufficiently close to maximal. We will pursue this strategy in the following section. Specifically, we will see how trying to operationalize this strategy leads to the recommendation of conserving *standing genetic variation* as a way to maximize additive genetic variance. I will also explain however why we should question the causal efficacy of that intervention for preserving the short-term evolvability of wild populations.

2.4 The conservation of standing genetic variation

In order to maximize the additive genetic variance in a trait within a population, we must have some idea of the amount of additive genetic variation for that trait within the population. This requires that we have some ability to measure additive genetic variation. However, in practice, measuring additive genetic variation in wild populations for the relevant quantitative traits may not generally be possible.

Among the genetic components of variation (i.e., additive variation, dominance variation, and epistatic variation), additive genetic variation is widely regarded as the most difficult to measure (Frankham et al., 2010). For most quantitative traits, we do not know all of the alleles or all of the loci that contribute additive effects to total phenotypic variance. So, instead of measuring additive genetic variation directly, biologists usually rely on measures of *standing genetic variation* as substitutes for estimates of additive genetic variation. Standing genetic variation is the overall genetic variation, including additive and nonadditive genetic variation, that is present within a population at a given time. Typical measures of standing genetic variation include heterozygosity, i.e., the average proportion of loci having combinations of different alleles within a population, and allelic diversity, i.e., the average number of alleles per locus within a population (Frankham et al., 2010). While such measures of standing genetic variation may be expected on theoretical grounds to correlate strongly with additive genetic variation, the actual correlation between measures of standing genetic variation and additive genetic variation has been shown to be rather weak, with measures of standing genetic variation accounting for at most 4% of additive genetic variation (Reed & Frankham, 2001). Consequently, obtaining accurate estimates of additive genetic variation within wild populations will generally require additive genetic variation to be measured directly.

Yet, as mentioned above, we do not know all of the alleles or loci contributing to additive genetic variation for most traits within wild populations. Moreover, we cannot be certain about which traits will be under the most intense selection in future environments. In that case, maximizing additive genetic variance through the conservation of additive genetic variation does not seem to be an option for most of the traits within wild populations that may be under intense selection in the future. Unless we know the specific additive genetic basis of the relevant traits, and unless we know what those traits are, we cannot conserve additive genetic variation for those traits directly. Consequently, for most quantitative traits within wild populations, we must rely on the conservation of standing genetic variation as an indirect way to maximize additive genetic variance in some trait or traits of interest.

Of course, in order to conserve standing genetic variation within wild populations, we must have access to that variation in order to determine the extent of what is to be conserved, i.e., how much standing genetic variation there actually is to conserve. For preserving the short-term evolvability of wild populations in the way described above, we should probably try at least to conserve a sampling of variation that is representative of the entire population, e.g., a representative sampling of all heterozygotic variation or all allelic variation within the population. However, there are obvious practical challenges to obtaining any representative estimates of genetic variation within wild populations. Certainly, for large or highly geographically dispersed populations, it may be not be practically feasible to get a sampling of a large enough proportion of individuals that would be representative of the genetic variation within the entire population (Frankel et al., 1995; Schonewald et al., 2003). This, on top of the technical requirements of obtaining any amount of genetic data, may make it very difficult to obtain any sufficiently representative samplings of standing genetic variation within wild populations. With respect to the conservation of standing genetic variation, these challenges may make it extremely unlikely that all, or possibly even most, of the relevant additive genetic variation would be conserved. This in turn may make it extremely unlikely that additive genetic variance in any trait within any wild population would be in any clear sense maximized.

2.5 Standing genetic variation and the evolutionary response to selection

Let us review. Recall that the maximization of heritability was initially undertaken as a strategy for maximizing a population's possible evolutionary response to selection. However, because heritability is a function of the specific environment a population is in, the heritability of a trait will vary with different environments. Moreover, because of our uncertainty about future environments, we cannot be certain about the extent to which the heritability of a trait might vary in those environments. More importantly, since the heritability most relevant to maximizing evolutionary response is that which is realized *after selection*, the maximization of heritability for some trait within a population before selection serves to maximize evolutionary response to future selection only indirectly. Again, since we are uncertain about future environments, we cannot be certain about the specific effects of selection in those environments, i.e., we cannot be certain about future selection differentials. As a result, we actually know very little about the future postselection heritabilities we should look to maximize.

Nevertheless, because the heritability of a trait is proportional to the additive genetic variance in that trait, there is still some sense in which heritability can be maximized for a range of unknown environments, despite our uncertainties about future environments and about how exactly heritabilities may vary in those environments. Specifically, we can try to maximize the amount of additive genetic variance in the trait or traits in question.

However, while the maximization of additive genetic variance appears at first to be a straightforward strategy, the implementation of that strategy as a conservation action faces several challenges. Note that the maximization of additive genetic variance requires measuring amounts of additive genetic variation within wild populations. Specifically, in order to maximize additive genetic variance, we need to have a representative estimate of the amount of additive genetic variation in order to determine the extent of that variance. However, we do not know the genetic basis for most of the quantitative traits in wild populations that may be subject to future selection. Consequently, we usually cannot measure additive genetic variation directly and must instead rely on measures of standing genetic variation, such as heterozygosity or allelic richness, as substitutes for estimates of additive genetic variation. However, as noted above, measures of standing genetic variation happen to be weakly correlated with additive genetic variation. Consequently, the conservation of standing genetic variation can serve to maximize additive genetic variation only in a very indirect and weakly correlated way. In other words, it is not clear that conserving standing genetic variation would actually serve to maximize additive genetic variance.

However, if all of this is the case, then the conservation of standing genetic variation can serve to maximize the heritability of quantitative traits only in a very indirect and weakly correlated way. If this is the case in turn, then the conservation of standing genetic variation would serve to maximize a population’s possible evolutionary response to selection only in a very indirect and weakly correlated way.

Taken together, these considerations raise serious doubts about the causal efficacy of conserving standing genetic variation as a conservation action for preserving the short-term evolvability of wild populations. As I have tried to show, the connection between standing genetic variation and the causal factors involved in evolutionary change through natural selection is at best indirect and only weakly correlative. As a result, we should regard it to be very unlikely that the conservation of standing genetic variation would actually serve to preserve the short-term evolvability of wild populations facing rapid environmental change.

In Chapter 3, I show that maximizing the possible magnitude of the *selection differential* for a given selective influence is not only an available strategy for maximizing a population’s possible evolutionary response to selection, but is also a preferable strategy for maximizing possible evolutionary response. I show that this strategy avoids the theoretical and epistemic challenges facing the maximization of heritability, and I show how it recommends an intervention—namely, the conservation of *standing phenotypic variation*—with a more direct and more strongly correlative connection to the causal factors involved in the production of evolutionary change through natural selection. Accordingly, I suggest that this intervention is a more causally efficacious conservation action for preserving the short-term evolvability of wild populations through rapid and possibly adverse environmental change.

CHAPTER 3

THE CONSERVATION OF PHENOTYPIC VARIATION FOR PRESERVING SHORT-TERM EVOLVABILITY

In Chapter 2, I showed how the maximization of heritability as a strategy to maximize the possible evolutionary response to selection recommends a conservation intervention with questionable causal efficacy for preserving the short-term evolvability of wild populations. In this chapter, I show that the maximization of the selection differential is in fact an available strategy for maximizing possible evolutionary response. Moreover, I show that maximizing the selection differential in the way I propose avoids the theoretical and epistemic challenges associated with the maximization of heritability. Because this strategy avoids those challenges, the intervention it recommends—namely, the conservation of *standing phenotypic variation*—has a much more direct and strongly correlative connection to the causal factors involved in the production of evolutionary change through natural selection. In turn, because of this direct and strongly correlative connection, we can be especially confident in the causal efficacy of that intervention for preserving the short-term evolvability of wild populations.

3.1 Maximizing the selection differential

As we noted in Chapter 2, the prevailing view in conservation biology regards the heritability of traits as the most important causal factor for producing evolutionary change in response to selection. This, along with a general uncertainty about future environments, prompted dismissing the possibility of intervening on the selection differential as an available strategy for maximizing the possible evolutionary response to selection in wild populations. However, there is a straightforward way to affect the selection differential such that the possible evolutionary response to selection is maximized: by maximizing the total phenotypic variance in the trait in question within the population. As I show below, the maximization of total phenotypic variance serves to maximize the possible magnitude of the selection differential by maximizing the effect of any selective influence acting on the trait in question. Because selection differentials of intermediate magnitude remain possible, this strategy also serves to maximize the *range* of selection differentials that are possible. In turn, maximizing the possible selection differential by maximizing total phenotypic variance also serves to maximize the *range* of possible evolutionary outcomes that may result from selection. Recall from Chapter 2 that maximizing the range of possible evolutionary outcomes was the original intention of maximizing possible evolutionary response. Importantly, the strategy presented here is unaffected by considerations of uncertainty about future environments or lack of knowledge about the precise genetic basis of traits expected to be under selection in those future environments.

To see how maximizing total phenotypic variance affects the selection differential, consider that variance is a statistical measure of the spread of trait values about a population mean. The more spread out trait values are within a population, i.e., the greater the quantitative differences among phenotypes, the greater the total phenotypic variance in that trait within the population. Because variance is a measure of the spread of trait values and not strictly a measure of trait values themselves, different populations can have identical trait means but nevertheless have different amounts of total phenotypic variance in the trait in question. Generally, populations having greater amounts of total phenotypic variance in a trait are populations in which extreme phenotypes are more common, while populations having lower amounts of total phenotypic variance are populations in which intermediate phenotypes are more common. Meanwhile, the mean value of the trait within those populations may be the same.

The consequence of having greater amounts of total phenotypic variance for a given population mean is that the effect of selection in a single generation of the population is greater than it would be were the amount of total phenotypic variance lower. In more precise terms, for any given instance of selection, the magnitude of the selection differential that results will be greater for populations with higher initial total phenotypic variance than for populations with lower initial total phenotypic variance. This is because of the residual influence of extreme trait values on the population mean after selection occurs.

To see how this bears on the preservation of short-term evolvability, consider an instance of intense directional selection, i.e., selection favoring extreme phenotypes on one side of the population mean, acting on a single quantitative trait z in two different populations, call them Population 1 and Population 2. We will set this up formally using the expression for the selection differential from Chapter 1:

$$S = (\overline{z_a} - \overline{z_b}), \tag{6}$$

where S denotes the selection differential, $\overline{z_b}$ denotes the mean value of the trait within the population before selection, and $\overline{z_a}$ denotes the mean value of the trait within the population after selection. Suppose that the populations have the same original mean trait value before selection, i.e., $\overline{z_{b1}} = \overline{z_{b2}}$, but have different amounts of total phenotypic variance in the trait, where Population 1 has the greater total phenotypic variance. Further, suppose that for both populations, the selective influence is the same such that it favors extreme trait values greater than the original population mean. In other words, the selective influence is such that individuals with more extreme trait values greater than the original population mean are less negatively affected and thus more likely to survive, while individuals with more extreme trait values less than the original population mean are more negatively affected and thus more likely to perish. Suppose now that selection occurs.

Notice that after selection occurs, the mean values of the trait in the populations will be different. Specifically, the mean in the population with greater initial total phenotypic variance, i.e., Population 1, will be greater than the mean in the population with less initial total phenotypic variance, i.e., Population 2; that is, $\overline{z_{a1}} > \overline{z_{a2}}$. As suggested above, this is because the trait mean in the population with greater initial total phenotypic variance includes a larger sampling of extreme trait values greater than the original population mean. Consequently, the selection differential will be greater in the population with greater initial total phenotypic variance, despite the selective influence on the trait in both populations having been the same. That is, $S_1 = (\overline{z_{a1}} - \overline{z_{b1}}) >$

$(\overline{z_{a2}} - \overline{z_{b2}}) = S_2$.

What the above scenario suggests is that the amount of total phenotypic variance in a trait within a population may have important implications for the persistence of that population in possibly adverse environments. Consider that the presence of more extreme phenotypes in the population with greater initial total phenotypic variance (i.e., Population 1) afforded that population a greater chance of avoiding extinction as a direct result of selection. Specifically, since the more extreme phenotypes were less likely to be negatively affected by selection, their presence meant that at least some of the population would be likely to survive. Thus, the presence of extreme phenotypes helped the population avoid extinction as a direct result of selection. In addition, since not only were extreme phenotypes present but present at higher frequencies in the population with greater total phenotypic variance, there would have been more opportunities for extreme phenotypes to breed with other extreme phenotypes. Consequently, the subsequent generation of the population would be expected to have a greater proportion of extreme phenotypes compared to that in the initial generation before selection, and the mean trait value in the subsequent generation would be expected to be more extreme. In other words, the evolutionary response to selection would be expected to be greater in the population having greater total phenotypic variance than in the population having less total phenotypic variance. Moreover, since those extreme phenotypes are less susceptible to being negatively affected by more of the same selective influences, the subsequent generation will have a greater proportion of individuals that will be less susceptible to being negatively affected by selection should those same selective influences continue.

So, for a given selective influence acting on some trait within a population, the magnitude of the selection differential will generally be larger for populations with greater total phenotypic variance than for populations with lower total phenotypic variance. Correspondingly, the magnitude of the evolutionary response to that selection will also be larger—for a given heritability. Accordingly, the maximization of total phenotypic variance offers a way to maximize the possible evolutionary response to selection. However, for this strategy to be at least as effective as the maximization of heritability for maximizing possible evolutionary response, certain conditions must be satisfied.

First, the trait under selection must be at least in some proportion heritable, such that changes in the frequency distribution of trait values in the initial generation due to selection may be passed on to the subsequent generation as an evolutionary response, regardless of what the precise heritability of the trait within the population may be. This condition is easily met, since for all quantitative traits subject to selection, we assume that they have some genetic basis that contributes additively to their expression. So long as there is some heritable variation in the trait, we can be confident that selection on that trait in an initial generation can result in an evolutionary response in the subsequent generation.

Second, the effect of selection on the frequency distribution of trait values in the initial generation due to selection, i.e., the selection differential, must be great enough to overcome any possibly low heritability associated with the trait, such that the possible evolutionary response in the subsequent generation is maximized. In other words, whatever heritability we may be losing out on by focusing on maximizing total phenotypic variance, the maximal selection differential we are securing must make up for whatever heritability is being lost out on. This second condition is likely to be met in most cases. Remember that in trying to maximize heritability, we focused on maximizing additive genetic variance rather than on decreasing total phenotypic variance in any way. Consequently, whatever maximal heritability we tried to achieve would have been a function of all of the phenotypic variance present within the population anyway. Accordingly, so long as maximizing total phenotypic variance just entails preserving all of the phenotypic variance there is within a population, no heritability is being lost out on by maximizing total phenotypic variance. Thus, in virtue of maximizing the possible selection differential alone, the maximization of total phenotypic variance serves to maximize the possible evolutionary response to selection.

Note that unlike the maximization of heritability, the maximization of the possible selection differential through the maximization of total phenotypic variance is unaffected by our uncertainty about future environments. Specifically, since what is doing the work of maximization is the total phenotypic variance in the initial generation of the population *before* any selection occurs, we do not need to be concerned with the specific details of future environments nor with the specific selective effects those environments may have on the frequency distribution of trait values. Whatever those effects may be, the magnitude of any resulting selection differential will be as great as possible. In the next section, I show how the maximization of total phenotypic variance translates straightforwardly to conservation action.

3.2 The conservation of standing phenotypic variation

Unlike the maximization of additive genetic variance, the maximization of total phenotypic variance can be operationalized as a conservation action in a comparably straightforward way—namely, by conserving *standing phenotypic variation*. The standing phenotypic variation within a population is just all of the phenotypic variation, including all additive and nonadditive variation, that is present within the population at a given time. Most importantly, unlike the connection between standing genetic variation and additive genetic variance, the connection between standing phenotypic variance and the total phenotypic variance in quantitative traits is direct and strongly correlative.

Note that measures of total phenotypic variance in a trait within a population are just measures of the standing phenotypic variation in that trait. Consequently, there is no sense in which one is a substitute for the other. So, in determining amounts of standing phenotypic variation within populations for conservation, with respect to some quantitative traits of interest, we may do so by assessing total amounts of phenotypic variance in those traits directly. With respect to specific traits within populations, the conservation of standing phenotypic variation implies the maximization of total phenotypic variance, specifically, insofar as *conserving* standing phenotypic variation just amounts to *preserving all of* the phenotypic variation that is present within the population at that time. Thus, in order to maximize total phenotypic variance with respect to quantitative traits expected to be under selection in the future, we do not require any special knowledge about the precise genetic basis of those traits beyond that they are quantitative traits.

Of course, estimating standing phenotypic variation within wild populations faces many of the same practical challenges as estimating genetic variation. For instance, it will still be difficult to obtain a representative sampling of phenotypes for large or geographically dispersed populations. However, it may be appreciated that standing phenotypic variation is in many cases more empirically accessible than genetic variation, insofar as many of the relevant quantitative traits are conspicuous morphological traits, e.g., body dimensions, as opposed to microscopic molecular characteristics. Because of this, the technical requirements for estimating phenotypic variation will often be less than those for estimating any amount of genetic variation. This means that phenotypic data may be more easy to obtain. This in turn may make it more practically feasible to assess standing phenotypic variation, compared to standing genetic variation, within large or geographically dispersed populations.

3.3 Standing phenotypic variation and the evolutionary response to selection

Thus, as I have shown, the maximization of the possible selection differential through the maximization of total phenotypic variance offers a way to maximize the possible evolutionary response to selection. Specifically, maximizing total phenotypic variance serves to maximize the possible magnitude of the selection differential for a given selective influence, which in turn serves to maximize the possible magnitude of the evolutionary response to selection for a given heritability. Notably, the maximization of total phenotypic variance bears directly on the magnitude of the selection differential that is relevant to the production of evolutionary change, and its bearing is unaffected by our uncertainty about future environments or our lack of knowledge about the precise genetic basis of traits expected to be under selection in the future. The maximization of total phenotypic variance thus avoids the challenges facing the maximization of heritability as a strategy for maximizing the possible evolutionary response to selection.

Importantly, the recommended intervention of conserving standing phenotypic variation has a direct causal connection to the maximization of total phenotypic variance. Specifically, since total phenotypic variance itself is simply a measure of standing phenotypic variation for some trait of interest, the conservation of standing phenotypic variation implies the maximization of total phenotypic variance, specifically, insofar as conserving standing phenotypic variation within a population just amounts to preserving all of the phenotypic variation in the trait that is present within the population at that time. Moreover, because of this direct connection between standing phenotypic variation and total phenotypic variance and the direct bearing of total phenotypic variance in turn on the selection differential, the connection between standing phenotypic variation and the causal factors involved in the production of evolutionary change through natural selection is demonstrably strong. For these reasons, we should expect the conservation of standing phenotypic variation to be a much more causally efficacious intervention for preserving the short-term evolvability of wild populations through rapid environmental change than we should expect the conservation of standing genetic variation to be.

CHAPTER 4

CONCLUSION

In the preceding, I have attempted to accomplish three things. First, I have attempted to show how a theoretical understanding of the causal structure of evolution through natural selection has important implications for the goal of conservation biology to secure the persistence of wild populations through rapid and possibly adverse environmental change. Specifically, I showed how this goal depends on the ability of wild populations to evolve in the short term as a way to cope with environmental change, and I showed how the preservation of this ability depends in turn on the maximization of the range of possible evolutionary outcomes in response to selection.

Second, I have attempted to show why a specific intervention aimed at preserving the short-term evolvability of wild populations, namely, the conservation of standing genetic variation, should be expected to have rather low causal efficacy for preserving evolvability. Specifically, I showed that the strategy that leads to its recommendation faces several challenges due to our uncertainty about future environments and our lack of knowledge about the precise genetic basis of most of the quantitative traits expected to be under selection in the future. I then showed that these considerations raise serious doubts about the causal connection between the conservation of standing genetic variation and the causal factors involved in the production of evolutionary change through natural selection.

Third, I have attempted to show that an alternative intervention, namely, the conservation of standing phenotypic variation, is superior to the conservation of standing genetic variation with respect to its expected causal efficacy for preserving short-term evolvability. Specifically, I showed how the conservation of standing phenotypic variation has a more direct and strongly correlative connection to the causal factors involved in the production of evolutionary change through natural selection. Moreover, I showed that the strategy leading to the recommendation of that intervention is unaffected by considerations of uncertainty about future environments or lack of knowledge about the specific genetic basis of any quantitative traits.

Meanwhile, I have highlighted several aspects of how specific conservation actions for achieving specific conservation goals follow from a theoretical understanding of the causal processes involved in achieving those goals. Specifically, I have shown how in light of uncertainty, theory has a central role in determining strategies for action. However, I also showed that the merits of specific strategies may be called into question, specifically, in light of uncertainties and the extent to which specific causal factors can be intervened on or affected directly. These considerations, I suggested, greatly influence the extent to which specific conservation actions may be expected to be causally productive of their intended results.

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