Towards a standard model for teaching the process of biological evolution

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Abstract

Evolution is widely considered to be one of the cornerstones of the biological sciences. Despite this importance, the process of biological evolution remains widely misunderstood among students, illustrating that evolution education is in need of an educational synthesis. The current paradigm for teaching the evolutionary process revolves around using population genetics models to illustrate the evolutionary change. While this convention describes the consequences of evolutionary changes, it conflates these consequences with the actual process of biological evolution. In addition to increased cognitive load, the pedagogical cost of this conflation is general misconceptions about the drivers of evolutionary change that at best create an overly simplistic view and at worst hinder understanding of the evolutionary process. To help address this issue, here we suggest evolution education should focus on trait-based models of evolution, and propose that Fisher's geometric model may provide a more useful model for centering education. We start by describing Fisher's geometric model and show how it can be used to effectively illustrate the fundamental evolutionary process (mutation, drift, selection, and migration/gene flow). We then mathematically show how Fisher's geometric model generalizes several widely used models in evolutionary biology, as well as how it can be connected to conventional population genetics. We conclude that future work should focus on empirically evaluating the efficacy of teaching Fisher's geometric model in the classroom.

Introduction

Evolution is widely accepted to be one of the foundations of the biological sciences, as it explains how and why various other topics in biology came to be [1]. Despite this importance, the process of evolution remains highly misunderstood among both students and the general public [2–5]. This issue is not only pertinent to students, as the field of evolutionary biology notoriously disagrees on the drivers of evolutionary change and has a history of creating adaptive stories to explain biological evolution [6–8]. This discourse among the field and frequent misunderstanding among students illustrates that evolutionary biology stands to greatly benefit from an educational synthesis that provides a standard model for teaching the process of biological evolution.

While there is debate about its specifics, it is generally accepted that some form the Modern Synthesis provides our most general description of biological evolution. The Modern Synthesis was born from the reconciliation of Darwin's theory of natural selection with Mendelian genetics, and in it's most general form, describes how mutation and gene flow can introduce genetic variation into a population, while drift, selection, and the rate of continued gene flow drive its change in frequency over time. Since its initial description, the Modern Synthesis has implicitly and explicitly served as the basis of education in evolutionary biology, which tends to focus on describing the process of evolution via changes in allele/genotype frequencies (Hardy-Weinberg model, Wright-Fisher model, selection coefficients, etc.) [9]. Similarly (and perhaps causally), the field of evolutionary biology largely focuses on explaining patterns of genetic/genomic evolution [7, 8, 10]. While this convention may describe the genetic mechanisms that facilitate evolutionary change, it conflates these mechanisms with the actual process of evolution. That is, evolutionary change is driven by traits and changes in allele frequencies are simply the genetic consequences, which is obscured by the current convention for teaching evolution. This perspective may be less intuitive given the educational emphasis on population genetics, but a breakdown of why traits are more important to consider for understanding evolutionary change is provided in Box 1.

The pedagogical cost of conflating the process of evolutionary change with its genetic mechanisms leaves students with a less accurate and less general picture of the evolutionary process. This is because changes in allele frequencies represent the *outcome* of drift, selection, and migration/gene flow, not the processes themselves. For example, it is common to include selection coefficients for different genotypes in a Hardy-Weinberg model and have students calculate the changes in genotype frequencies. This is problematic because the driver of selection is not some selection coefficient that is an intrinsic property of an allele/genotype. The driver of selection is differential reproductive success conferred by heritable traits. Similarly, allele frequencies change stochastically (drift) *because* the traits of individuals do not confer heritable differential reproductive success. While these points might seem pedantic, focusing evolution education on calculating changes in allele/genotype frequencies leaves students with a picture of the consequences of evolutionary change as opposed to an intuitive understanding what the different evolutionary process actually are and how they operate. In attempt to paint a more comprehensive picture that links genotypes and traits, it is common to assume a genotype-phenotype map and call on specific empirical examples where said map is applicable. The most common example is the classic heterozygote advantage in a population burdened by sickle cell disease and malaria. While this and similar examples may help students connect genetics to evolution, they typically require emphasis on additional genetic complexities, such as diploidy, dominance, and recombination. While these are real phenomena, they are features of specific genetic architectures, not fundamental features of the evolutionary process (most organisms are not diploids, do not have sex, etc.). Including the complexities of specific genetic architectures forces students to have to simultaneously learn about said specific genetic architecture and the evolutionary process. This increases cognitive load during learning without adding conceptual clarity about what evolution fundamentally is, which can result in student frustration and ineffectual learning outcomes [11]. Furthermore, focusing on specific genetic architectures can distort student understanding into thinking very niche situations are common and fail to illustrate the other evolutionary processes that act simultaneously.

Box 1. The evolutionary process

Mutation: Mutation is the process that generates heritable variation in traits. This is predominately associated with genetic mutations, which from a literal standpoint is not incorrect. However, mutations may be produced by any number of kinds of genetic and epigenetic changes, and the fundamental aspect of mutation that is important for the process of evolution is its effects on traits.

Selection: Selection is the process by which individuals with certain traits experience elevated reproductive success relative to those with other traits. Population genetics tends to assign fitness values or selection coefficients to genotypes/alleles. However, this practice incorrectly reflects causality, as different genotypes or alleles are only selected for because of their associated traits. In other words, changes in genotype/allele frequencies is the consequence of selection, not its driver, and emphasizing selection at the level of traits better reflects causality.

Drift: Similar to selection, drift is the result of changes that are not related to differential reproductive success. The process of drift is typically referred to as *genetic drift*, reflecting stochastic changes in the frequencies of different genetic variations. However, it is important to emphasize that drift is the *consequence* of stochastic sampling driven by small population sizes and/or a lack of reproductive differences conferred by a heritable trait (neither of which are genetic properties). This means genetic drift is the pattern that emerges from the process of drift, which is driven by changes that occur without respect to fitness.

Gene flow: Gene flow is perhaps the least intuitive to separate from the underlying genetic mechanisms, as the word "gene" is in the name. However, it is important to emphasize that the most fundamental property of gene flow that determines its evolutionary importance is determined by what traits are being brought into population interest from elsewhere. Gene flow also plays a role in shaping the relative importance of selection and drift (via its effects on effective population sizes), both of which are driven by how individuals are sampled with respect to their traits.

Instead of centering evolution education in models of population genetics, here we suggest centering education in a model of phenotypic change, which is both more reflective of our best understanding of the natural world and stands to be more pedagogically useful [12]. To make this shift away from population genetics-centered evolution education, we suggest emphasis on Fisher's geometric model [13]. In summary, Fisher's geometric model considers a multi-dimensional trait space where individuals are points within this space. For example, in a 2-dimensional case, one dimension (axis) could be beak width and the other could be beak length. Here, the phenotype of an individual, which is the cumulation of its traits, is given by its coordinates within the trait space. There is also an optimal phenotype within the trait space, which represents the combination of traits that increases the probability of reproductive success. Individuals that are closer to this optimum (have a more suitable beaks) are more likely to serve as parents of offspring the next generation. Offspring then inherit the traits of their parents, but mutation introduces slight deviations to the

trait values (a parent with a beak length of 9 mm could have an offspring with a beak length of 9.5 mm because of a mutation that increased beak length). This process repeats, and one can track how the population's trait values evolve over time.

Because Fisher's geometric model deals in traits, not alleles, it allows for easier integration of visual depictions of how traits evolve, a tool that can be useful for teaching evolution [12]. In doing so, it provides both a more accurate depiction of evolutionary change and a pedagogically useful platform for teaching said process. To facilitate the integration of Fisher's geometric model into evolution education, here we begin by providing an overview of the model in both non-mathematical and mathematical terms. We then demonstrate how Fisher's geometric model can illustrate the fundamental evolutionary process (mutation, drift, selection, and migration/gene flow) to students, as well as how it can provide students with a conceptual unification of several properties of the evolutionary process. We then mathematically show that Fisher's geometric model generalizes several other models that are frequently employed in the field of evolutionary biology (we also provide a conceptual overview), therefore creating a natural bridge for students to engage with more complex topics. Among the models that are encompassed in Fisher's geometric model is the breeder's equation, and we use this to show how teaching Fisher's geometric model can help students bridge the evolutionary process and quantitative genetics, which then allows for consideration of other sources of phenotypic variation. Finally, we show how Fisher's geometric model can be connected to population genetics and allele frequency dynamics under specific assumptions (that are not more extreme than assumptions routinely made in other models used for teaching). We conclude that Fisher's geometric model has the potential serve as a more pedagogically useful starting point for education in evolutionary biology relative to population genetic models, and briefly introduce an open-source software for helping bring Fisher's geometric model into the classroom.

An introduction to Fisher's geometric model

The following subsections provide general overviews of Fisher's geometric model. The section titled "Conceptual overview" focuses more on the conceptual basis of Fisher's geometric model without relying on a mathematical description. The section titled "Quantitative overview" provides the mathematical and statistical description of Fisher's geometric model. Readers can read either section without loss of continuity.

Conceptual overview

The most basic form of Fisher's geometric model begins by considering a fitness landscape defined in a multi-dimensional trait space. For example, if we consider Darwin's finches, one dimension could be beak width and another could be beak length. Individuals are represented as points within this space, and the coordinates of a point defines the corresponding individual's phenotype (Figure 1A). Within the trait space, there is an optimal phenotype, which represents the trait combination that maximizes reproductive success. Therefore, the fitness of each individual is defined by measuring their distance from the optimum in the phenotype space. Fitness of an individual declines with increasing distance from this optimum, and the rate of this decline determines the strength of selection. For example, is beak width is not under strong selection, the decline in fitness associated with having a non-optimal beak width is more gradual. Similarly, if beak width is

under strong selection, even minor deviations from the optimal beak width results in a sharp fitness penalty. The fitness values of each individual are then summed to calculate the relative fitness of each individual with respect to the total fitness of the population. The parent of any given individual in the next generation is decided by sampling parents of the current generation, where the probability of being sampled as a parent is proportional to an individual's relative fitness.

Offspring inherit the traits of their parents, but mutations can cause trait values to change. For example, if an individual's parent had a beak length of 9 mm, they might have a beak length of 8.5 mm because a mutation resulted in a change of -0.5 mm. These mutational changes are drawn from what is called the mutational effect distribution, which is typically a normal distribution with a mean of 0 (mutation is not directional) and some standard deviation (Figure 1B). It is important to emphasize that mutation here does not refer to the genetic basis of the mutation itself, but rather the change in the trait value. For example, the previously described -0.5 mm reduction in beak length could be generated by any number of single nucleotide changes, genomic rearrangements, etc. Therefore, the standard deviation of the mutational effect distribution corresponds to the mutational input, where a higher standard deviation could be due to either a greater mutation rate (more genetic mutations) or mutations with larger effects on the phenotype. Offspring of the current generation then become the parents of the next, and this process repeats. It is typical that population size is held constant, and the standard deviation of the mutational effect distribution is kept small. However, these parameters can certainly be changed to explore how properties like a greater mutation rate or larger population size impact evolutionary dynamics. This process is graphically summarized in Figure 1 and step-by-step breakdown of the evolutionary algorithm is provided in Box 2.

Box 2. Steps for implementing Fisher's geometric model

1. Define a fitness landscape in a trait space by setting an optimal phenotype (point in the trait space that maximizes reproduction probability) and defining a function that describes how fitness declines with distance from the optimum.

2. Initialize a population by defining points within the trait space. Most members of the population may be far away from the optimum (and will then undergo directional selection) or near the optimum (where they will be under stabilizing selection).

3. Calculate the relative fitness of each individual in the population. This is done by dividing each individual's fitness by the sum of all fitness values in the population.

4. For each individual in next generation, sample a parent, where the probability of being chosen is given by the individual's relative fitness.

5. Once parents have been selected, add mutations to their trait values to determine the traits of the offspring. Mutations are generated by sampling values from a normal distribution with a mean of 0 and a standard deviation of σ_m (typically small relative to trait value).

6. Set the mutated offspring as the current generation, and repeat steps 3–6.

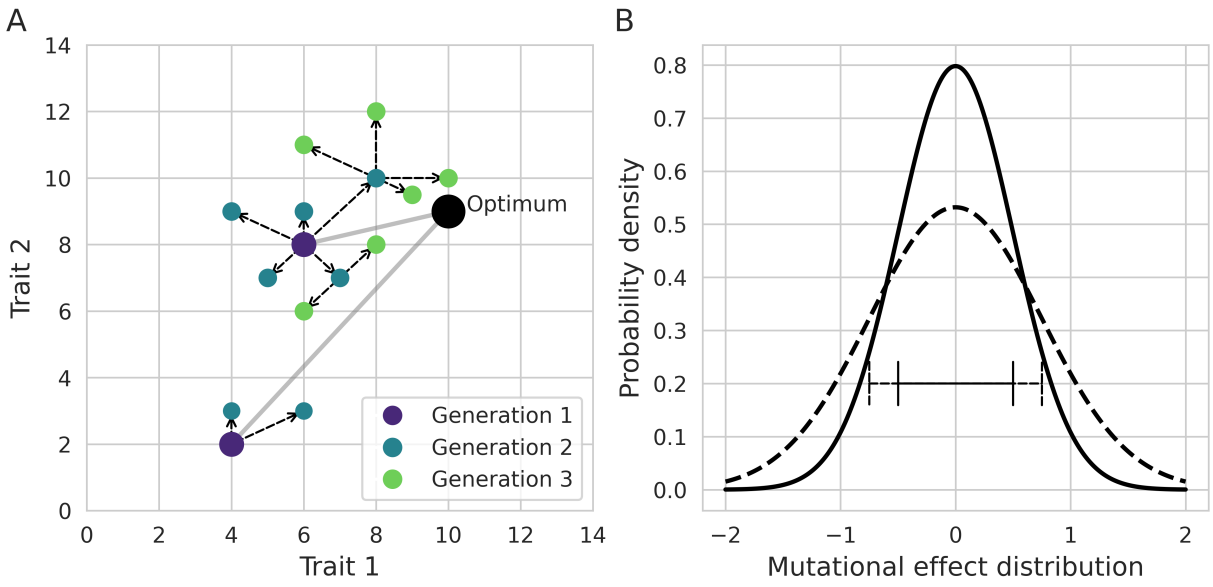

Figure 1: A graphical summary of Fisher's geometric model. A) A 2-dimensional trait space with an optimal phenotype designated as the large black point. Individuals from the first generation, which are depicted in a darker purple, are some distance from the optimum, which is depicted by the lighter gray lines. The individual in generation 1 that is further from the optimum only produces 2 offspring, while the individual that is closer produces 5 offspring. This process repeats to generate the third generation. Each member of subsequent generations have similar trait values to their parent, but have also mutated, where mutations were drawn from the mutation effect distribution depicted in B. B) The mutational effect distribution used to sampled mutation. The x-axis denotes the change in offspring trait from their parent's, and the y-axis the probability density of the corresponding change.

Quantitative overview

Fisher's geometric model considers a fitness landscape defined in an n-dimensional trait space. Each dimension represents a different trait, designated as z. For example, if we consider Darwin's finches, one dimension could be beak width and another could be beak length. Within this space, there exists an optimal phenotype defined by a point P_{opt}. Letting $z_{\text{opt},i}$ represent the optimal value for trait z_i, the optimal phenotype P_{opt} is

$$P_{\text{opt}} = [z_{\text{opt},1}, z_{\text{opt},2}, \ldots, z_{\text{opt},n}], \tag{1}$$

where n is the number of traits under consideration. Fitness of an individual is then defined with respect to its distance from this optimum. For an individual with a phenotype of $P = [z_1, z_2, \ldots, z_n]$, the squared distance d^2 from the optimum, is

$$d^2 = \sum_{i=1}^{n} (z_i - z_{\text{opt},i})^2. \tag{2}$$

Fitness declines with increasing distance from this optimum, and the rate of this decline is determined by the strength of selection, denoted as σ_s. Therefore, the fitness function takes the Gaussian form

$$W = \exp\left(-\frac{d^2}{2\sigma_s^2}\right). \tag{3}$$

In Equation 3, σ_s defines the strength of selection, but it may be more intuitive to think about σ_s as scaling the width of the fitness peak. For example, $\sigma_s = 3$ corresponds to a wider fitness peak, meaning individuals that are further from the optimum may still achieve some fitness. Conversely, $\sigma_s = 1.5$ corresponds to a very narrow fitness peak, indicating selection is acting strongly on the population, and individuals with traits that place it far from the optimum are less able to achieve higher fines. This property is illustrated in Figure 2, which shows theoretical fitness landscapes defined by beak length and width in Darwin's finches.

Once a fitness landscape has been defined, a population then evolves on said landscape. First, imagine a population of individuals that vary in their traits, placing them at different points across the fitness landscape. Returning to Figure 2, one can visualize where a finch with a beak width of 10 mm and a beak length of 8 mm would be situated on the fitness landscape, and contrast this positioning with a finch with a beak width of 8 mm and a beak length of 10 mm. The position of individuals on the fitness landscape then determined their fitness (Figure 2). It is standard practice to convert the absolute fitness W_i of each individual into relative fitness, which would be $w_i = \frac{W_i}{\frac{1}{n}\sum_{j=1}^{n} W_j}$, where w_i is the relative fitness of individual i. However, implementing Fisher's geometric model is typically done in a Wright-Fisher-like framework, where for each offspring in the next generation, a parent is selected with a probability that is proportional to their relative fitness. Therefore, we instead need to calculate the probability p that an individual i will be selected to parent any given offspring in the next generation, which is

$$p_i = \frac{W_i}{\sum_{j=1}^{n} W_j}. \tag{4}$$

In other words, the sampling probability of an individual is given by dividing their absolute fitness by the total fitness of the population. Letting N_{t+1} be the number of individuals in the next

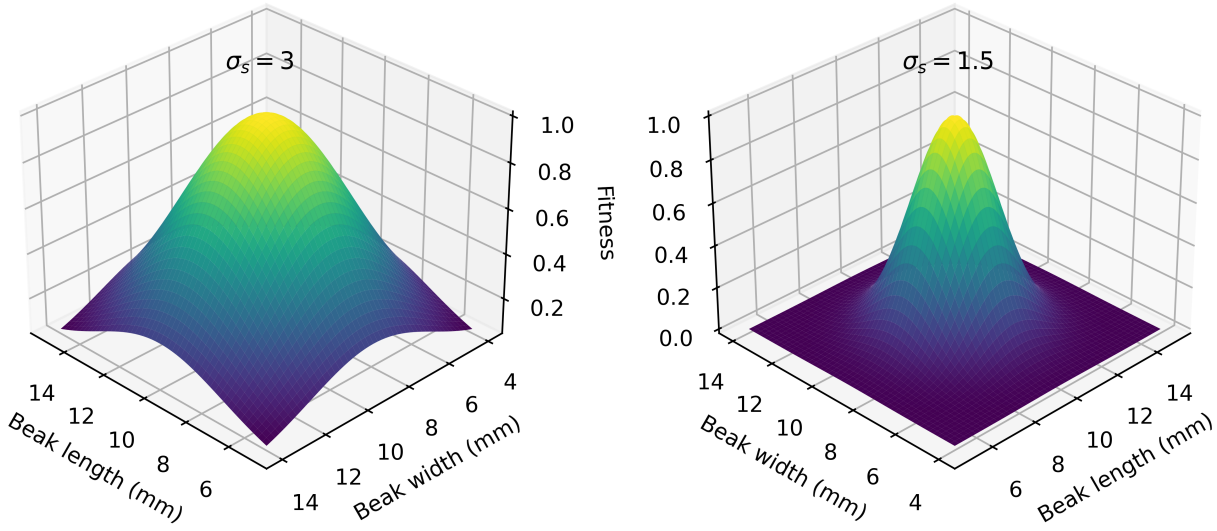

Figure 2: Theoretical fitness landscapes defined by the beak length (z-axis) and beak width (x-axis) trait space in Darwin's finches. The y-axis in each panel represents fitness, where brighter colors also correspond to higher fitness. The difference between panels illustrates the effect of σ_s (strength of selection) in shaping the fitness landscape. In the left panel, $\sigma_s = 3$ corresponds to relatively weak selection, meaning individuals that are further away from the optimum can achieve relatively higher fitness. Conversely, $\sigma_s = 1.5$ in the right panel represents stronger selection, where individuals that are further from the optimum have significantly lower fitness.

generation, the next generation is produced by using the parental sampling probabilities to populate a multinomial distribution that is sampled N_{t+1} times. Put more simply, for each offspring, a parent is sampled with a probability defined in Equation 4. Note that the same parent can be chosen multiple times, allowing for greater reproductive success. This illustrates how reproduction is a stochastic sampling process, but individuals with higher fitness still have a greater chance of producing more offspring.

Once an individual has been sampled to parent an offspring, they then pass their traits on to said offspring. If offspring were simply clones of their parents, they would keep the same trait values as their parents. However, offspring also receive mutations, which slightly change their trait values from those of their parents. To understand how mutations are modeled, consider a parental phenotype $P_{\text{parent}} = [z_1, z_2, \ldots, z_n]$. The phenotype of their offspring would be determined by adding random deviations (mutations) to their traits, which is accomplished by drawing mutations from a normal distribution with a mean of 0 and a variance of σ_m^2. Let $\delta_i \sim \mathcal{N}(0, \sigma_m^2)$ represent a mutation to the i^{th} trait, the offspring phenotype with respect to that of their parent would be

$$P_{\text{offspring}} = [z_1 + \delta_1, z_2 + \delta_2, \ldots, z_n + \delta_n]. \tag{5}$$

Put more simply, offspring traits are their parents traits plus noise (mutation) that is drawn from a normal distribution. It is important to note that the distribution from which mutations are drawn is centered on 0, indicating that most mutations have small effects and are not inherently directional in how they impact the trait value. Likewise, the variance of this distribution is typically small

relative to the values of the traits, but can be altered to represent higher or lower mutation rates. The offspring of the current generation then become parents of the next, and this process then repeats, allowing the population's traits to change over time.

Fisher's geometric model illustrates fundamental evolutionary processes

Mutation, drift, and selection

The four fundamental processes that drive evolution are mutation, drift, selection, and gene flow. Therefore, it is important that a central model on which education in evolutionary biology is based is able to effectively illustrate the nature of these processes to students.

One of the most fundamental relationships in evolutionary biology is between population size and the importance of drift in driving evolutionary dynamics. Fisher's geometric model illustrates this well, as depicted in Figure 3A, which shows a number of lineages evolving via drift. It is clear that the mean trait value of lineages with a population size of 50 stochastically change more than populations with 500 individuals, which stochastically change more than populations with 1000 individuals (Figure 3A). This mirrors the more commonly employed depictions of allele frequencies stochastically varying over time across different population sizes.

Fisher's geometric model also paints an intuitive depiction of how selection operates. Figure 3B shows a number populations evolving to reach a new optimum. Within this, different populations are subject to different strengths of selection (σ_s), which changes the rate at which they reach the new optimum. Once populations reach the new optimum after a period of directional selection, the mean trait values stabilize around said value, showing the populations shifting to regimes of stabilizing selection (Figure 3B). This shift is of pedagogical importance, and is not as clearly communicated via population genetic models. In evolutionary biology education, selection is often presented categorically, where different outcomes of selection are described as a taxonomy. However, Fisher's geometric model more readily illustrates to students that the different outcomes of selection (stabilizing, directional, etc.) are emergent properties of the population's location on the fitness landscape, thus providing a more unified and explicit view of the different outcomes of selection. This is not to say that the standard taxonomy is wrong. Rather, it might obscure the underlying process of selection, which is better clarified in Fisher's geometric model.

Another fundamental aspect of biological evolution that is more readily illustrated by Fisher's geometric model is the balance between mutation, drift, and selection. Further inspection of Figure 3B shows that after the populations have reached the new fitness optimum, the mean trait values still vary around the optimum across generations. Stabilizing selection may keep the populations near the optimum but mutation and drift allow them to move away, creating a classic tension that is fundamental for understanding the process of evolution. Fisher's geometric model illustrates the selection-mutation-drift balance in a more intuitive way than population genetic models, which would need to show either the frequency dynamics of many different alleles or the dynamics of genetic diversity. The former is less visually clear and both require additional layers of mechanistic understanding which adds cognitive load and decreases pedagogical value particularly during earlier stages of education.

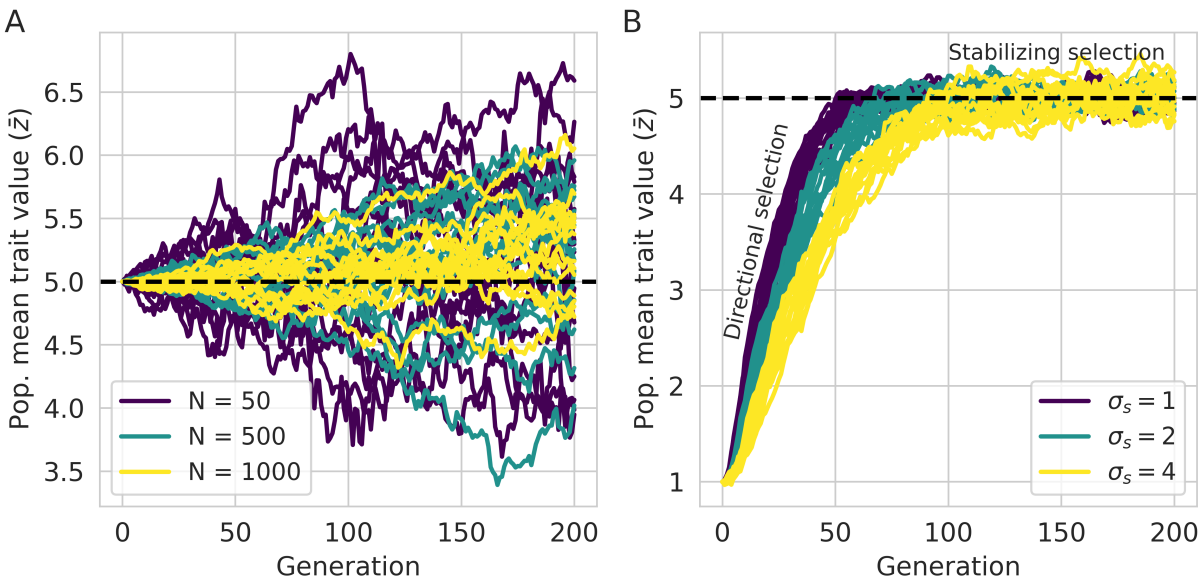

Figure 3: Fisher's geometric model more clearly illustrates the fundamental properties of mutation, drift, and selection that are central to understanding the process of biological evolution. A) Populations evolving purely via mutation and drift. The y-axis represents the population mean trait value, and the x-axis denotes evolutionary time in generations. Different colored lines represent different population sizes, and replicate lines of the same color represent replicate simulations. The black dashed line represents the initial trait values. These dynamics show that smaller populations are more sensitive to drift and therefore vary more in the mean trait value over time. B) Populations evolving via mutation, selection, and drift. Different colored lines represent different strengths of selection (lower σ corresponds to stronger selection), and the black dashed line represents the optimal trait value. The populations initially start far away from the fitness optimum and undergo a period of directional selection that fuels adaptation. Once the populations reach the fitness optimum, subsequent dynamics reflect the balance between mutation, drift, and selection.

Migration/gene flow

Gene flow is a somewhat more complicated evolutionary process to teach. It isn't a direct extension of Fisher's geometric model, but it isn't exactly a direct extension of standard population genetic models that are used for earlier teaching either (e.g., Hardy-Weinberg, Wright-Fisher, etc.). Therefore, what specific aspects of gene flow need to be included depends on the goal of the educational exercise. For simplicity, here we will focus on showing how Fisher's geometric model (with a brief extension) can illustrate how gene flow can both mitigate and facilitate adaptation, as this is a commonly discussed property of gene flow earlier in education.

A simple way to include migration/gene flow into the Fisher's geometric framework is to replace a fraction m of the focal sub-population each generation with immigrants from a different sub-population. This other sub-population may have trait distribution that is different from that of the focal population, and this difference is key to understanding the implications of gene flow for the focal population. To illustrate this, consider a focal population with an initial mean trait value of $\bar{z}_{\text{init}} = 5$, as illustrated in Figure 4A. Then consider a fitness landscape shift, placing the new op-

timum at $z_{\text{opt}} = 15$ (Figure 4A). If the sub-population experiences no immigration (or immigration from a sub-population with the same trait distribution), it will adapt to the new fitness landscape under standard dynamics (Figure 4B). In contrast, we can consider a scenario where individuals from a different sub-population migrate into the focal sub-population. The trait distribution in the migrating sub-population is centered at a value of $\bar{z}_i = 10$, which initially and rapidly pulls the trait distribution of the focal population towards the optimum (Figure 4B). In other words, immigration initially facilitates more rapid adaptation. However, if the sub-population that the immigrants came from is not undergoing selection to reach the same optimal value of 15, this immigration eventually constrains adaptation in the focal sub-population because it pulls the trait distribution towards that of the immigrating population, as illustrated in Figure 4B. Demonstrating all of the possible outcomes of migration/gene flow is not practical for the purposes of this manuscript. However, the previously described example illustrates how the complexities of migration/gene flow can still be easily explored using Fisher's geometric model as a base.

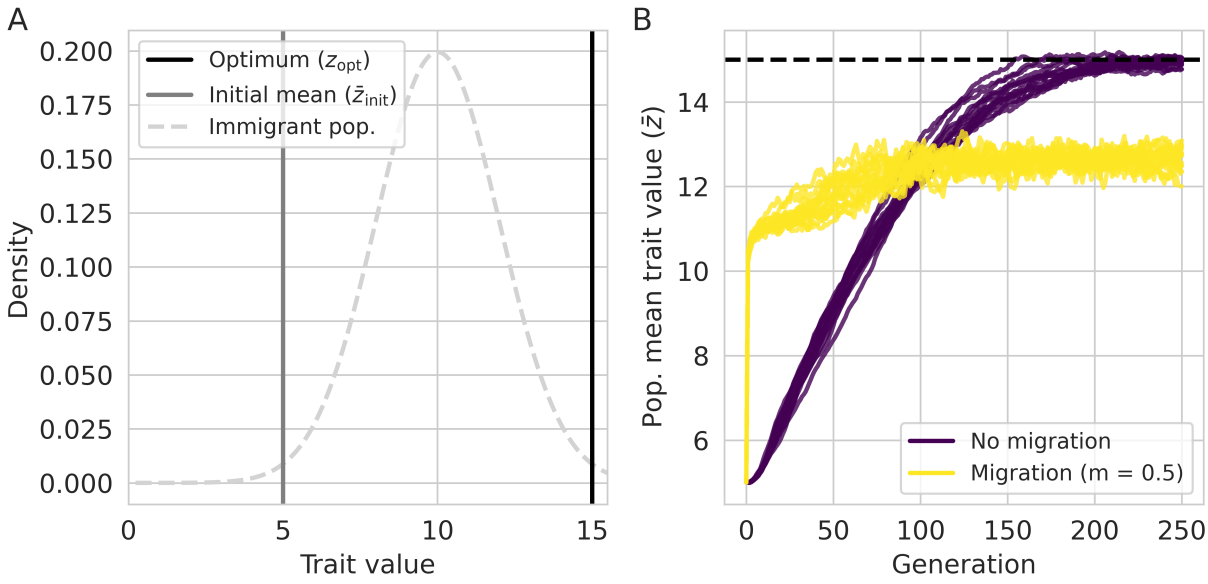

Figure 4: The complex effects of migration/gene flow can be illustrated using Fisher's geometric model. A) The darker gray vertical line represents the initial mean trait value for a focal sub-population, and the vertical black line represents a new optimal trait value. The distribution depicted by the lighter gray dashed line represents the trait distribution of a different sub-population that is immigrating into the focal population. B) Evolutionary dynamics for scenarios where there is no immigration into the focal sub-population, (darker lines) and migration ($m = 0.5$) from a different sub-population with a trait distribution depicted in A (lighter lines). The solid black line denotes the new optimum for the focal population.

Fisher's geometric model generalizes well-established models in evolutionary biology

The following subsection develops the mathematical connection between Fisher's geometric model, the breeder's equation, the Ornstein-Uhlenbeck process, and Brownian motion, all commonly used models in evolutionary biology. Readers primarily interested in the conceptual implications may skip the subsection titled "The mathematical connection" to the proceeding section titled "The conceptual connection" without loss of continuity.

The mathematical connection

A primary advantage of focusing on Fisher's geometric model as a unifying framework for teaching the process of evolution is that several other important and well-established models in evolutionary can be derived from it under general assumptions. These include the breeder's equation, the Ornstein-Uhlenbeck process, and Brownian motion. To make these links, first consider a 1-dimensional case of Fisher's geometric model, where z_i represents the trait value of the i^{th} individual within a population. The population mean $\bar{z}$ at time t is therefore

$$\bar{z}_t = \frac{1}{N}\sum_{i=1}^{N} z_i, \tag{6}$$

where N is the number of individuals in the population. Fitness of the i^{th} individual is then

$$W_i = \exp\left(-\frac{(z_i - z_{\text{opt}})^2}{2\sigma_s^2}\right). \tag{7}$$

The probability of being selected to be the parent of any given offspring is then

$$p_i = \frac{W_i}{\sum_{j=1}^{N} W_j}. \tag{8}$$

Assuming $z_i \sim \mathcal{N}(\bar{z}_t, V)$ and $V \ll \sigma_s^2$, the expected trait value of a chosen parent is

$$\mathbb{E}[z_{\text{parent}}] = \sum_{i=1}^{N} p_i z_i. \tag{9}$$

Letting $\delta_i = z_i - z_t$, the second order Taylor expansion of W_i around z_t gives

$$W_i \approx \bar{W}\left(1 - \frac{(\bar{z}_t - z_{\text{opt}})}{\sigma_s^2}\delta_i - \frac{\delta_i^2}{2\sigma_s^2}\right), \tag{10}$$

where $\bar{W} = \frac{1}{N}\sum_{j=1}^{N} W_j$. Letting $\sum_i \delta_i = 0$ and $\frac{1}{N}\sum_i \delta_i^2 = V$, substituting this into $\mathbb{E}[z_{\text{parent}}]$ gives

$$\mathbb{E}[z_{\text{parent}}] \approx \bar{z}_t - \frac{V}{\sigma_s^2}(\bar{z}_t - z_{\text{opt}}). \tag{11}$$

Therefore, the expected change in the mean due to selection each generation is

$$\mathbb{E}[\Delta \bar{z}^s] = -\frac{V}{\sigma_s^2}(\bar{z}_t - z_{\text{opt}}) \tag{12}$$

Note that Equation 12 is exactly the breeders equation, where $h^2 S = \frac{V}{\sigma_s^2}(\bar{z}_t - z_{\text{opt}})$.

The next generation mean also changes due to drift (finite sampling) and mutation. The N offspring are drawn multinomially from parents with probabilities p_i, giving a variance of the sample mean of approximately V/N. Mutations adds independent deviations from $\mathcal{N}(0, \sigma_m^2)$ to each offspring, thus adding σ_m^2/N to the variance of the mean. Therefore, the total variance per generation is

$$\text{Var}(\Delta \bar{z}) \approx \frac{V + \sigma_m^2}{N}. \tag{13}$$

Under the infinitesimal model, V is approximately constant across generations, making this variance effectively constant.

Combining the expected change in the mean and the variance in the mean gives the discrete time difference equation update

$$\bar{z}_{t+1} = \bar{z}_t - \frac{V}{\sigma_s^2}(\bar{z}_t - z_{\text{opt}}) + \epsilon_t, \quad \epsilon_t \sim \mathcal{N}\left(0, \frac{V + \sigma_m^2}{N}\right) \tag{14}$$

Scaling time so that each generation corresponds to the interval Δt, the deterministic term scales as Δt and the stochastic term scales as $\sqrt{\Delta t}$ (standard diffusion scaling). Letting $\alpha = \frac{V}{\sigma_s^2}$, $\sigma = \sqrt{\frac{V+\sigma_m^2}{N}}$, and dW_t be a Weiner process, taking $\Delta t \to 0$ gives

$$d\bar{z}_t = -\alpha(\bar{z}_t - z_{\text{opt}})dt + \sigma dW_t, \tag{15}$$

which is the stochastic differential equation for the Ornstein-Uhlenbeck process. Here, it is useful to note that α is the ratio of phenotypic variance to the width of the fitness peak (how strongly selection pulls the mean back), and σ is how much the mean wanders per generation due to drift and mutation. Setting $\sigma_s^2 \to \infty$, which produces a flat fitness landscape (no selection), $\alpha \to 0$ and the deterministic term vanishes, leaves

$$d\bar{z}_t = \sigma dW_t, \quad \sigma = \sqrt{\frac{V + \sigma_m^2}{N}}, \tag{16}$$

which is Brownian motion with the diffusion coefficient $\sigma = \sqrt{\frac{V+\sigma_m^2}{N}}$.

The conceptual connection

As shown above, Fisher's geometric model provides a unifying framework for teaching the process of evolution because it encompass several well established evolutionary models. Specifically, we first showed that the expected change in the trait mean due to selection each generation in Fisher's geometric model is exactly the breeder's equation (Equation 12). We will revisit this point more generally in the following section focused on the link between Fisher's geometric model and quantitative genetics.

Extending this process over time and including the effects of drift and mutation, we then showed how the Ornstein-Uhlenbeck process can be derived from Fisher's geometric model under the assumption of many small-effect loci (Equation 15). Finally, we showed that removing the effects of selection gives rise to Brownian motion, which is a model of pure mutation and drift (Equation 16). Taken together, these findings show that several foundational models in evolutionary biology can be derived from Fisher's geometric model, illustrating its utility as a more foundational base for teaching the evolutionary process. To further this point, it is important to note that these models describe the evolution of population mean trait values, rather than individual-based dynamics that are modeled by Fisher's geometric model, which provides a useful pedagogical bridge between micro- and macro-evolutionary dynamics. While these models might not be essential or common in an early introduction to the evolutionary process, focusing initial education on Fisher's geometric model would likely prepare students to engage with them in more advanced topics and when encountered in the literature.

Fisher's geometric model creates a natural bridge to quantitative genetics

Quantitative genetics is often a central aspect education in evolutionary biology because it explains where variation in traits comes from. In the most basic form, total phenotypic variance V_P is partitioned into variance explained by genetics (V_G), variance explained by the environment (V_E), and variance explained by genetic by environmental interaction (V_{GE}). Within this, variance explained by genetics can be further partitioned into variance explained by dominance effects (V_D), variance explained by epistatic effects (V_I), and variance explained by additive effects (V_A). Therefore, total phenotypic variation V_P is

$$V_P = \underbrace{V_A + V_D + V_I}_{V_G} + V_E + V_{GE}. \tag{17}$$

From Equation 17, it is clear that Fisher's geometric model does not describe each of these sources of variation. There is not a source of variation due to the environment and there are not complex genetic interactions, which is a point that is pedagogically useful. Fisher's geometric model implicitly assumes an additive architecture, and subsequently discussing quantitative genetics shows students additional sources of phenotypic variation. This sets the stage for students to engage in questions like "*where does phenotypic variance actually come from*?" and "*how do additional sources of variation impact evolutionary dynamics*?"

Because of this natural link, Fisher's geometric model also connects cleanly to the breeder's equation, as we previously showed. More conceptually, broad sense heritability is $H^2 = V_G/V_P$, which describes all genetic contributions to phenotypic variance. Narrow-sense heritability is $h^2 = V_A/V_P$, which represents the additive portion. This distinction is important because only V_A predictably responds to selection, while dominance and epistatic effects are not reliably transmitted from parent to offspring in the same manner. Because Fisher's geometric model implicitly assumes additivity, $H^2 = h^2$. The breeder's equation, which predicts the evolutionary response R of a trait mean due to selection, is

$$R = h^2 S, \tag{18}$$

where S is the selection differential. This is exactly the same logic that governs the rate at which population mean trait moves towards the optimum in Fisher's geometric model, illustrating that

Fisher's geometric model embeds the breeder's equation. Stemming from this discussion, it is also pedagogically useful to point out that in reality, $H^2 > h^2$ (usually) because epistasis and dominance effects add additional phenotypic variance beyond additivity, which can aid in student understanding of the breeder's equation and quantitative genetics as a whole.

Fisher's geometric model can be naturally connected to population genetics

Teaching the evolutionary process has been predominately done through population genetic models, making it important to connect Fisher's geometric model to said frameworks. To achieve this, we must first define a genotype-phenotype map, which we will for simplicity assume is linear and additive. If there are L loci each with two alleles, and each allele contributes an additive effect a_l, the trait z is

$$z = \sum_{l=1}^{L} a_l x_l + \epsilon, \tag{19}$$

where $x_l \in \{0, 1, 2\}$ counts the number of derived alleles at locus l and ϵ is residual noise. Under this map, the fitness of an individual is still determined by how far their phenotype z is from the optimum z_{opt}. This is somewhat of a departure from how selection is typically introduced in simple population genetic models, which involves assigning selection coefficients (s) to alleles. However, it is important to make this departure, as selection coefficients represent an abstraction that oversimplifies how the process of evolution works.

To connect the previously described population genetic model to Fisher's geometric model, we need a way for the additive genotype-phenotype map to produce continuous traits. This link is provided by Fisher's infinitesimal model, which models continuous phenotypic variation by assuming traits are produced by many small effect loci. Allele frequencies can then evolve under Wright-Fisher dynamics, with the exception that fitness is determined as described in Equation 3, rather than a fixed selection coefficient. The aggregate change in allele frequencies across all loci then produces the phenotypic dynamics that are well described by Fisher's geometric model. This is illustrated by Figure 5, which shows how allele frequency dynamics translate into population mean phenotype dynamics, as well as how this compares to the Fisher geometric model dynamics. It is important to note that the initial standing genetic variation in the multilocus model can result in slight deviation from standard Fisher's geometric model dynamics due to their discreteness.

Note that the simulations depicted in Figure 5 do not strictly enforce the infinitesimal limit but they do approximate this regime sufficiently to illustrate the connection. Nonetheless, this shows how Fisher's geometric model can still be linked to population genetic models, and therefore, serve as a link between micro- and macro-evolutionary process. The phenotypic dynamics described by Brownian motion and the Ornstein-Uhlenbeck process emerge from Fisher's geometric model dynamics, which are consistent with, and can be derived as diffusion limits of (under certain assumptions) from the previously described allele frequency dynamics under a Wright-Fisher-like model. Under the infinitesimal model (and its approximation), additive genetic variation (V_A) is also maintained by many small effect loci, thus completing the link to quantitative genetics as well. It is important to note that these connections break down under non-linear/non-additive genotype-phenotype maps. However, this is an important pedagogical point that connects back to the previously described discussion of quantitative genetics.

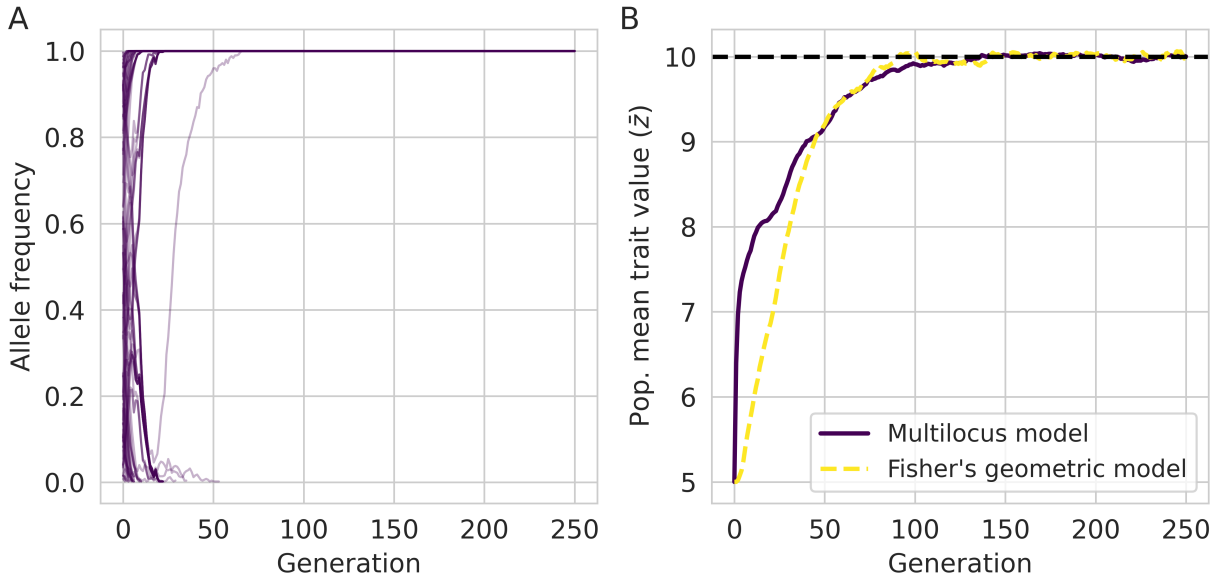

Figure 5: Fisher's geometric model dynamics recapitulate the mean trait value dynamics of Fisher's infinitesimal model under Wright-Fisher-like dynamics. A) Changes in allele frequencies over time as a population adapts to a new optimum. The population starts with standing genetic variation, which are alleles at various frequencies with phenotypic effects that are drawn from a normal distribution. Each line represents the dynamics of an individual allele. B) The population mean trait dynamics ($\bar{z}$) over time as populations adapt to a new optimum, which is denoted as the dashed black line. The purple line shows the emergent phenotypic dynamics from the allele frequency dynamics depicted in A, and the yellow dashed line shows Fisher's geometric model predictions for a population adapting to the same landscape.

Future directions

Population genetic models have long served as the corner stone for teaching students the process of evolution. However, this is problematic because it conflates the process of biological evolution with its genetic consequences, thus obscuring the process that drive evolutionary change. To begin addressing this issue, here we suggest re-focusing evolution education on trait-based models, and propose that Fisher's geometric model may be useful to make this transition. We showed how Fisher's geometric model illustrates each of the fundamental process that drive biological evolution, as well theoretical justification for how it can connect a variety of topics in evolutionary biology education. To help facilitate integration into the classroom, we have developed FGMLab (https://gabe-dubose.github.io/FGMLab/), an open-source interactive web application for running Fisher's geometric model and visualizing model dynamics using interactive animations. This application can be used by students to help illustrate the evolutionary process, but also by instructors to serve as a basis for designing problem sets.

Despite this theoretical justification and apparent utility, it will be important for future work to empirically assess the efficacy of teaching the evolutionary process using Fisher's geometric model. This goal has several empirical challenges. Most notably, population genetic models are so

integrated in evolution education that standard forms of assessing student understanding are based on population genetics. For example, it is common to assess student understanding of genetic drift [2], but assessing the efficacy of teaching via Fisher's geometric model would require assessing student understanding of stochastic changes in traits. Therefore, new forms of assessment may need to be devised to accurately compare our proposed trait-centric approach to the conventional population genetics-centric approach. Nonetheless, we believe future work should focus on conducting these evaluations, as Fisher's geometric model has the potential to serve as a standard model for teaching the process of biological evolution.

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