Chapter 3

Natural selection

Natural selection is a mechanism for generating an exceedingly high degree of improbability. Ronald Fisher

3.1 The language of population genetics

So far we have assumed a monomorphic population, i.e., that all individuals in the population are identical. But individuals in a population generally differ in many ways. For instance, they differ in traits that can easily be observed, such as size, weight, or strength. Individuals are also likely to differ in traits that cannot be directly observed, such as blood pressure, ability to break down lactose, or personality and preferences. Differences in the expression of a given trait can both result in fitness differences and have a genetic basis. The purpose of this chapter is to understand how natural selection drives the evolution of the genetic component of traits, and to that aim it is useful to introduce some population genetics terminology. In particular, it is important to distinguish the four following fundamental concepts (see also section 1.1, "The organism and basic genetic concepts", of Chapter 1).

• **Phenotype**. Any morphological, physiological or behavioral trait expressed by an individual. It ranges from the acceleration rate of a reaction by an enzyme, to individual preferences of chocolate flavors. Everything that is directly or indirectly observable about an individual is part of its phenotype, including its state of mind.[1]

• **Genotype**. Set of molecular determinants of the phenotype (encoded in DNA), each of which is transmitted independently of the environment. In humans, the genotype is inherited by the **zygote**, the initial cell of an organism formed by the fertilization event

[1] Preferences and beliefs as considered in Microeconomics and Game Theory are part of an individual's phenotype.

between a sperm and an egg.

• **Gene**. A non-recombining element of the genotype. Roughly put, it the region of DNA that codes for an amino acid chain and thus for a single and simple protein.

• **Allele**. A variant of a gene.

A **Mendelian trait** is a trait (or phenotype) that is controlled by a single gene. Most traits, however, are likely to be **polygenic** or **multifactorial**, which means that their expression depends on the effects of several interacting genes and possibly the environment. But Mendelian traits are probably the simplest starting point to study natural selection, what we now turn to.

3.2 Natural selection as an evolutionary force

3.2.1 Allele frequency and fitness

We now consider that the population is **polymorphic** so that different individuals express different phenotypes. We assume that the phenotype of an individual is faithfully transmitted from a parent to its offspring, which is virtually equivalent to saying that the trait is entirely genetically determined. In this case, there is no need to distinguish between genotype and phenotype.

In order to understand how natural selection operates on faithfully transmitted traits, we assume that only two alleles can segregate in a population where individuals are haploid. We call one allele A and the other B. Since different alleles may encode for different phenotypes (physiological, morphological, or behavioral), their expression may result in different survival and reproductive outcomes. We denote by w_{A} the fitness of an individual carrying allele A and by w_{B} the fitness of an individual carrying allele B (recall eq. 2.3 for the definition of fitness).

We further denote by n_{A} the number of individuals carrying A and by n_{B} the number of individuals carrying B, which must sum up to the total population size

$$n = n_{\mathrm{A}} + n_{\mathrm{B}}, \tag{3.1}$$

since individuals in the population either carry allele A or B.

We are now ready to introduce the key quantity featuring in evolutionary analysis. This is the **frequency** of individuals in the population that carry A:

$$p = \frac{n_{\mathrm{A}}}{n}. \tag{3.2}$$

Thus, p is the relative number (or proportion) of individuals in the population having allele A and it varies between zero and one ($p \in [0, 1]$). For instance, suppose there is a population

with $n = 1000$ individuals in which $n_{\mathrm{A}} = 250$ individuals carry allele A, then the frequency of this allele in the population is $p = 250/1000 = 0.25$. The complementary proportion of individuals $1 - p$ in the population carry allele B, hence we have

$$1 - p = \frac{n_{\mathrm{B}}}{n}. \tag{3.3}$$

3.2.2 Change in allele frequency

Formally, **evolution** is defined as the change in allele frequency in a population. Let us evaluate this change over one demographic time period. Using the notation we introduced to describe the change of a variable over a single discrete time period (recall eq. 2.2), the change in the frequency of A over one demographic time period is

$$\Delta p = p' - p, \tag{3.4}$$

where p' is the frequency of allele A in the descendant generation, when p is its frequency in a given parental generation. How do we determine p'? Since by definition p' is the relative number of A individuals in the descendant generation, we have

$$p' = \frac{n'_{\mathrm{A}}}{\underbrace{n'_{\mathrm{A}} + n'_{\mathrm{B}}}_{n'}} = \frac{w_{\mathrm{A}} n_{\mathrm{A}}}{w_{\mathrm{A}} n_{\mathrm{A}} + w_{\mathrm{B}} n_{\mathrm{B}}}. \tag{3.5}$$

The numerator in the right-hand side is the fitness, $w_{\mathrm{A}} n_{\mathrm{A}}$, multiplies the number of individuals carrying A in the parental generation by their fitness w_{A}. By the definition of fitness, this gives the number n'_{A} of A individuals in the descendant generation. Likewise, $w_{\mathrm{B}} n_{\mathrm{B}}$ is the number n'_{B} of B individuals in the descendant generation so that the denominator in eq. (3.5), $w_{\mathrm{A}} n_{\mathrm{A}} + w_{\mathrm{B}} n_{\mathrm{B}}$, equals the total number n' of individuals in the descendant generation. Now multiply the numerator and denominator of the right-hand side of eq. (3.5) by the factor $1/n$ and use the expression for allele frequency $p = n_{\mathrm{A}}/n$ in the parental generation (eqs. 3.2–3.3). We can write the frequency of allele A in the descendant generation as

$$p' = \frac{w_{\mathrm{A}}}{\bar{w}} p, \tag{3.6}$$

where

$$\bar{w} = p w_{\mathrm{A}} + (1 - p) w_{\mathrm{B}} \tag{3.7}$$

is the **average fitness**; that is, the fitness of an individual randomly sampled in the population.

In eq. (3.6), $w_{\mathrm{A}}/\bar{w}$ is the relative fitness of a A individual, which measures how well it survives and/or reproduces relative to the average (or a randomly chosen individual) in the population. Hence, the interpretation of eq. (3.6) is that the A allele frequency in the descendant generation is equal to allele frequency in the parental generation times the relative fitness of that allele. As a consequence, an allele should increase in frequency, i.e., get a higher share in the population, if its fitness is larger than that of an average individual.

In order to obtain a more intuitive interpretation of allele frequency change, we substitute eq. (3.6) and eq. (3.7) into eq. (3.4), whereby the change $\Delta p = p' - p$ in the frequency of allele A over one demographic time step is

$$\begin{aligned}
\Delta p &= \frac{w_{\mathrm{A}}}{\bar{w}} p - p \\
&= \frac{w_{\mathrm{A}}}{\bar{w}} p - p \frac{\bar{w}}{\bar{w}} \\
&= \frac{1}{\bar{w}} \left[w_{\mathrm{A}} p - p \bar{w} \right] \\
&= \frac{1}{\bar{w}} \left[w_{\mathrm{A}} p - p \left(p w_{\mathrm{A}} + (1-p) w_{\mathrm{B}} \right) \right] \\
&= \frac{1}{\bar{w}} \left[w_{\mathrm{A}} p (1-p) - p (1-p) w_{\mathrm{B}} \right] . \qquad (3.8)
\end{aligned}$$

Factoring out $p(1-p)$, the change in the frequency of allele A can now be written as

$$\Delta p = \frac{p(1-p)s}{\bar{w}}, \qquad (3.9)$$

which consist of two quantities. First, the **variance** $p(1-p)$ in the frequency of A in individuals in the population. The variance[2] $p(1-p)$ is a measure of diversity in the population. It is equal zero when the allele is either fixed in the population ($p = 1$) or absent from it ($p = 0$), and takes a maximal value at $p = 1/2$, when half the individuals carry A. Hence, it is dome shaped as a function of p. Second, the **selection coefficient**

$$s = w_{\mathrm{A}} - w_{\mathrm{B}} \qquad (3.10)$$

on the A allele, which is the difference in fitness between expressing allele A and B. The selection coefficient can be thought of as the fitness "gains from switching" to an individual from the expressing allele A instead of B.

Two things should be noted about eq. (3.9):

• Allele frequency change by natural selection occurs only if there is genetic variability in the population $v > 0$. And the change is proportional to the variance v in the frequency of the allele under selection.

• The direction of allele frequency change (whether it increases or decreases) is determined by the selection coefficient s, which is the change in an individual's fitness brought by expressing the A allele instead of the B allele. If expressing A results in a benefit, i.e., $s > 0$, selection favors A that will henceforth increase in frequency in the population over a demographic time step. To the contrary, if A results in a cost, i.e., $s < 0$, selection disfavors the A allele, which will decrease in frequency in the population.

[2]The fact that $p(1-p)$ is the variance can be seen from the definition of the variance, i.e., the average of the squared deviation relative to the average, which is $(1-p)^2 \times p + (0-p)^2 \times (1-p) = p(1-p)$, where $(1-p)$ is the deviation from the average in A individuals, since the frequency of A in such individuals is "1″, and $(0-p)$ is the deviation from the average in B individuals, since the frequency of A in such individuals is "0″

Eq. (3.9) is thus a formal encapsulation of Darwin's idea of natural selection and describes how selection operates on any *replicator*, defined as a unit that exhibits (1) faithful heredity, (2) variation, and (3) replication.

3.2.3 Evolutionary equilibria

Importantly, eq. (3.9) allows to evaluate evolutionary equilibria (analogously to finding equilibria in population dynamic models as we did in Chapter 2, recall also Appendix 1 therein). Evolutionary equilibria are the equilibrium values of p (denoted by p^*), such that there is no change in allele frequency: $\Delta p = 0$. We can see from eq. (3.9) that there are two types of equilibria. Firstly, two **boundary equilibria**: either A is fixed in the population ($p^* = 1$) or it is extinct ($p^* = 0$). Secondly, **interior equilibria**: each of which must satisfy $s = 0$.

3.3 Modes of selection

The equation for allele frequency change (eq. 3.9) describes evolutionary change over one demographic time step, but so far it does not allow us to determine where evolution will go. Indeed, s may depend on allele frequency as well (i.e., it may depend on p) and may thus change from one time step to the next. In order to reach more definite predictions we need to make more specific assumptions. To that end, we now investigate three different modes of selection: **constant**, **density-dependent**, and **frequency-dependent** selection.

3.3.1 Constant selection

Suppose fitnesses (w_A and w_B) are constant at all times, then the selection coefficient s (eq. 3.9) will also be constant for all time, which in turn implies that the change in allele frequency Δp will be of constant sign. If $s > 0$, then $\Delta p > 0$ as long as the variance is larger than zero ($p(1-p) > 0$). This implies that A will go to **fixation** in the population, since it is favored at all allele frequencies. Conversely, if $s < 0$, then $\Delta p < 0$ as long as the variance is larger than zero ($p(1-p) > 0$), which implies that A will go to **extinction** in the population, since it is disfavored at all allele frequencies.

For constant selection (constant s), we can further explicitly solve for the frequency p_t as a function of time t. Without loss of generality, we assume that the fitness of B individuals is one ($w_\mathrm{B} = 1$, the baseline fitness at an ecological equilibrium, recall the previous chapter) and write $w_\mathrm{A} = 1 + s$ for some constant s. Using explicit time indices, eq. (3.6) is given by

$$p_{t+1} = \left(\frac{1+s}{1+sp_t} \right) p_t \tag{3.11}$$

owing to the fact that $\bar{w} = p_t w_\mathrm{A} + (1-p_t) w_\mathrm{B} = 1 + sp_t$ (when $w_\mathrm{B} = 1$ and $w_\mathrm{A} = 1 + s$). By

iterating this equation (like in Derivation 3 in the Appendix of Chapter 1), one obtains that

$$p_t = \frac{1}{1 + \frac{1-p_0}{p_0}\left(\frac{1}{1+s}\right)^t}, \tag{3.12}$$

where p_0 is the initial frequency of the allele. The frequency of allele A as a function of time (Fig. 3.1) shows that the increase in frequency of A under positive selection follows a sigmoidal curve, which encapsulates two features.

• The favored allele **initially increases at exponential rate**. This can be understood by considering the change in allele frequency over one demographic time step when the allele is initially **rare** ($p \approx 0$). In this case, the population mean fitness $\bar{w}$ is simply that of the B allele, since the effect of A individuals on population average fitness can be neglected. Therefore, we can approximate the denominator of eq. (3.11) by $\bar{w} = 1 + sp_t \approx w_B = 1$ to get

$$p_{t+1} = (1+s)\,p \qquad \Longrightarrow \qquad p_t = (1+s)^t\,p_0. \tag{3.13}$$

The frequency of allele A changes at an exponential rate given by its fitness w_A. If $s > 0$, the frequency of A increases exponentially while if $s < 0$ it goes to extinction. In the case of invasion, the variance in allele frequency is reduced and the change in allele frequency must decrease giving rise to the sigmoidal nature of allele frequency dynamics (Fig. 3.1).

• Constant selection results in **invasion implies substitution**, since when $s > 0$, the frequency of A not only increases when rare (invasion) but goes to one ($p \to 1$) for any initial positive frequency $p_0 > 0$ so that A replaces B (in eq. 3.12, $(1/(1+s))^t \to 0$ as $t \to \infty$ when $s > 0$). By contrast, extinction befals when $s < 0$ (Fig. 3.1).

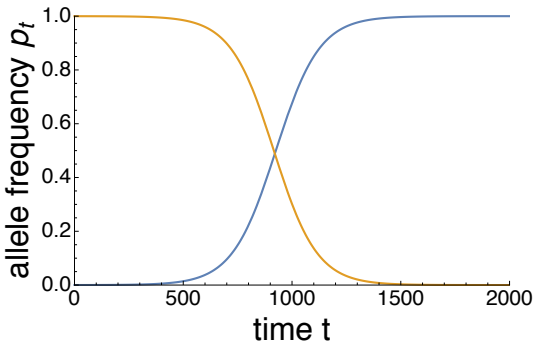

Figure 3.1: Frequency p_t of A as a function of time t (eq. 3.12) . Parameters: $p_0 = 10^{-4}$ and $s = 0.01$ (blue), $p_0 = 1 - 10^{-4}$ and $s = -0.01$ (yellow).

3.3.2 Density-dependent selection

Both the fitness of A and B allele are unlikely to be constant over time, if only because of competition for resources. Let us now assume that the fitness of the A and B allele are, respectively, given by

$$w_{\mathrm{A}}(n) = \frac{f_{\mathrm{A}}}{1+\gamma_{\mathrm{A}} n} \quad \text{and} \quad w_{\mathrm{B}}(n) = \frac{f_{\mathrm{B}}}{1+\gamma_{\mathrm{B}} n}, \tag{3.14}$$

where $n = n_{\mathrm{A}} + n_{\mathrm{B}}$ is the total number of individuals carriers of the A and B allele in the population. These fitnesses are those of a Beverton-Holt model of density-dependent competition (recall eq. 2.7), but here parameters depend on the genotypes. The fitnesses imply individuals of different genotypes have different fecundities (f_{A} for A and f_{B} for B) hence, are subject to different strengths of density-dependent competition (γ_{A} for A and γ_{B} for B).

From eq. (3.14) and eq. (3.10), the selection coefficient for this model is

$$s(n) = w_{\mathrm{A}}(n) - w_{\mathrm{B}}(n), \tag{3.15}$$

where the n emphasizes that selection is *density-dependent*, since fitness depends explicitly on n. Substituting this selection coefficient into eq. (3.9) allows to track the change in allele frequency as

$$\Delta p = \frac{p(1-p)s(n)}{\bar{w}(n)}, \tag{3.16}$$

but here we also need to track population dynamics, since both the selection coefficient and mean fitness $\bar{w}(n) = pw_{\mathrm{A}}(n) + (1-p)w_{\mathrm{B}}(n)$ depend on it. The number of individuals in the descendant generation is

$$n' = w_{\mathrm{A}}(n)n_{\mathrm{A}} + w_{\mathrm{B}}(n)n_{\mathrm{B}}, \tag{3.17}$$

which, using eqs. (3.2)–(3.3), can be formulated as

$$n' = \underbrace{\left[w_{\mathrm{A}}(n)p + w_{\mathrm{B}}(n)(1-p)\right]}_{\bar{w}(n)} n. \tag{3.18}$$

Hence, population dynamics are determined by mean fitness so that if $\bar{w}(n) > 1$, then the population size increases. This is intuitive as it says that if the average individual in the population produces more than one offspring, then the population size increases.

Note that eq. (3.16) and eq. (3.18) define a coupled dynamical system of equations, where selection affects population dynamics, which in turn affects the dynamics of the allele frequency. This is called an **eco-evolutionary feedback**. In order to investigate such feedback, let us first consider the case $\gamma_{\mathrm{B}} = \gamma_{\mathrm{A}} = \gamma$ so that alleles affect only fecundity, and not the strength of density-dependent selection. Substituting this into $s(n)$ (eq. 3.15) and $\bar{w}$ (see eq. 3.18) shows that $s(n) = (f_{\mathrm{A}} - f_{\mathrm{B}})/(1+\gamma n)$ and $\bar{w} = \bar{f}/(1+\gamma n)$, where

$\bar{f} = pf_A + (1-p)f_B$ is the average fecundity in the population. Then, from eq. (3.9), the change in the frequency of A reduces to

$$\Delta p = p(1-p)\frac{(f_A - f_B)}{\bar{f}}. \tag{3.19}$$

Hence, the change in allele frequency is independent of population size. We have density-independent selection and can study selection without considering population dynamics.

Independence of selection on population size is indeed an attractive simplifying feature. However, in a more general case, how does natural selection operate if different alleles also affect density-dependent competition, so that $\gamma_A \neq \gamma_B$ in the Beverton-Holt model? To answer this question let us denote the carrying capacities in a monomorphic A and B population, respectively, by

$$n_A^* = \frac{(f_A - 1)}{\gamma_A} \qquad \text{and} \qquad n_B^* = \frac{(f_B - 1)}{\gamma_B}. \tag{3.20}$$

Here, we thus use the equilibrium population sizes from the demographic Beverton-Holt model (eq. 2.11), which are specific for each allele.

Suppose now we introduce a single A individual into the population at the demographic equilibrium of the B allele, given by n_B^*. The fitness of a carrier of B is therefore $w_B = 1$ (otherwise the population would not be at equilibrium, recall eq. 2.10). Allele A will be favored by selection if its fitness w_A, in a population of B individuals, is larger than that of a B individual in a monomorphic B population; that is, if

$$w_A = \frac{f_A}{1 + \gamma_A n_B^*} \quad > \quad w_B = 1, \tag{3.21}$$

which is equivalent to

$$\frac{f_A}{1 + \gamma_A n_B^*} > 1. \tag{3.22}$$

This means that the fitness of a A individual in a B population must be larger than one. This holds only if[3]

$$n_A^* > n_B^*. \tag{3.23}$$

In other words, a rare A allele is favored by selection if its carrying capacity (in an otherwise monomorphic population) is larger than that of the alternative B allele. If this condition holds, then A will not only invade the population when rare but also go to fixation (see Appendix 1 of this chapter). Hence, initial invasion implies fixation of the allele in the population, and the equilibrium population size after selection will be larger than before (see Fig. 3.2). Here, competition brought by natural selection increases the productivity in the population, as measured by its carrying capacity.

[3]We have that $\frac{f_A}{1+\gamma_A n_B^*} > 1 \iff f_A > 1 + \gamma_A n_B^* \iff (f_A - 1)/\gamma_A > n_B^*$, which is equivalent to $n_A^* > n_B^*$, by the definition given in eq. 3.20.

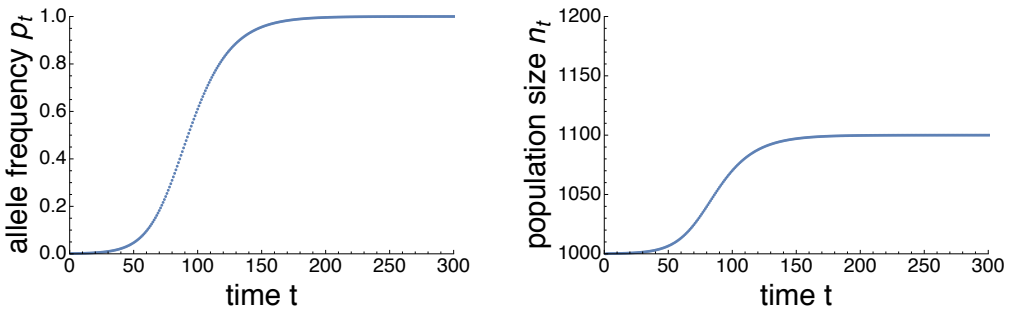

Figure 3.2: Frequency of A and total population size as a function of time (using eq. 3.18 and eq. 3.9 along with eq. 3.14) for $p_0 = 10^{-4}$ and $n_0 = (f_B - 1)/\gamma_B$ (carrying capacity of B) with $f_B = 2$, $\gamma_B = 0.001$, $f_A = 6.5$, and $\gamma_A = 0.005$.

3.3.3 Frequency-dependent selection

Under both constant and density-dependent selection, we saw that invasion implies substitution. Namely, when the A allele is favored by selection, it not only increases when rare (invasion) but goes to fixation in the population and thus replaces the B allele (substitution). But this outcomes was obtained in situations where the vital rates of an individual were independent of the frequency of the alleles in the population. In general, however, the fecundity and/or survival of each individual is likely to depend on allele frequencies in the population.

Suppose for instance that individuals in the population are randomly paired to play a symmetric "Hawk-Dove" **game**. A game is a situation where individuals can express different actions with consequences depending on the action of others. In the Hawk-Dove game individuals compete over a resource of value V and can express one of the following two actions (phenotypes): playing Hawk or playing Dove. If the two interacting individuals play Dove, then they share the resource, each receiving a payoff of $V/2$. If the two individuals play Hawk, then they fight over the resource. On average each individual is assumed to get half of the resource, but pays a cost C for fighting. Hence, the payoff to each individual playing Hawk is $V/2 - C$. Playing Hawk thus seems at first glance to result in a lower payoff than playing Dove. But when one individual plays Hawk and the other plays Dove, then the Hawk is assumed to get all the resource, thus getting a payoff of V, while the Dove gets a payoff of zero.

Suppose now that the A allele programs its carrier to play Hawk and allele B to play Dove. We can see these two actions as pure strategies in the Hawk-Dove game. Then, assuming random matching of individuals for competitive interactions, such that a Hawk or

Dove interacts with a Hawk with probability p and with a Dove with probability $1-p$, we can write the fitness of a carrier of allele A and B, respectively, as

$$\begin{aligned} w_{\mathrm{A}}(p) &= 1 + p\left(\frac{V}{2} - C\right) + (1-p)V \\ w_{\mathrm{B}}(p) &= 1 + p \times 0 + (1-p)\frac{V}{2}, \end{aligned} \tag{3.24}$$

where the "1" is the baseline fitness of an individual. One defining feature of this model is that the fitnesses depend on the frequency p of the A allele. Hence, the selection coefficient (eq. 3.10) is given by

$$s(p) = w_{\mathrm{A}}(p) - w_{\mathrm{B}}(p), \tag{3.25}$$

which exhibits **frequency-dependence**, since it depends on p.

Substituting eq. (3.24) into eq. (3.25), and simplifying, we get that

$$s(p) = \frac{V}{2} - Cp. \tag{3.26}$$

Thus, switching to play Hawk instead of playing Dove results in an assured benefit of $V/2$ and a cost that depends on the frequency of Hawk players in the population.

Under frequency-dependent selection, in addition of the two boundary equilibria ($p^* = 1$ and $p^* = 0$), interior evolutionary equilibria arise and these are found by setting $s(p^*) = 0$. Substituting eq. (3.26) and solving for p^* yields

$$p^* = \frac{V}{2C} \tag{3.27}$$

as the (unique) interior equilibrium in the Hawk-Dove game. This equilibrium can be stable or unstable, depending on the value of the parameters. In fact, three evolutionary outcomes are possible in the Hawk-Dove game (and this qualitatively applies to any two player two action game):

• **One allele dominates the other (constant selection)**. If $V/2-C < 0$ and $V/2 < 0$, then $s(p) < 0$ for all $p > 0$. Hence, allele A is counter-selected at all frequencies and driven to extinction by selection ($p \to 0$). If $V/2 - C > 0$ and $V/2 > 0$, then $s(p) > 0$ and allele A is favored at all frequencies and goes to fixation ($p \to 1$).

• **Stable equilibrium**. If $V/2 - C < 0$ and $V/2 > 0$, then A is favored when rare ($s(p) > 0$ when $p \approx 0$) and counter-selected when frequent ($s(p) < 0$ when $p \approx 1$). Hence, there will be an internal stable equilibrium given by eq. (3.27). This is a state where there is *polymorphism* in the population since the two alleles coexist. This polymorphism is sometimes called a *protected polymorphism* since it is stable.

• **Unstable equilibrium**. If $V/2 - C > 0$ and $V/2 < 0$, then A is counter-selected when rare ($s(p) < 0$ when $p \approx 0$) and favored when frequent ($s(p) > 0$ when $p \approx 1$). Hence, eq. (3.27) characterizes an unstable equilibrium and the endpoint of the evolutionary dynamics depends on the initial conditions.

We have investigated density-dependent and frequency-dependent selection separately. In general, since resources are limited and individuals interact, a population will display both density-dependent and frequency-dependent selection. We will study more in detail the consequences of the resulting eco-evolutionary feedback when we turn to adaptive dynamics, the gradual, step-by-step transformation of trait values in a population by selection. But before doing, so we need to introduce the concept of mutation as an evolutionary force: this is the topic of the next chapter.

3.4 Appendix

Appendix 1. To prove that invasion implies substitution in the evolutionary model specified by eq. (3.14), we note that at equilibrium, allele frequency change Δp^* and population number change Δn^* must satisfy the following system of equations (from eq. 3.18, eq. 3.15, and eq. 3.9):

$$\begin{aligned} \Delta p^* &= \frac{p^*(1-p^*)}{\bar{w}} \left(w_{\mathrm{A}}(n^*) - w_{\mathrm{B}}(n^*)\right) = 0 \\ \Delta n^* &= \left(p^* w_{\mathrm{A}}(n^*) + (1-p^*) w_{\mathrm{B}}(n^*)\right) n^* = 0, \end{aligned} \tag{3.28}$$

where fitness is given by eq. (3.14). It can then be checked, that the only non-trivial solutions (i.e., involving a population size different than $n^* = 0$) are

$$(p^* = 1, n^* = \frac{f_{\mathrm{A}} - 1}{\gamma_{\mathrm{A}}}) \quad \text{and} \quad (p^* = 0, n^* = \frac{f_{\mathrm{B}} - 1}{\gamma_{\mathrm{B}}}). \tag{3.29}$$

Hence, there can be no interior solution with $p \in (0, 1)$. This means that the mutant either goes extinct or invades the population from any initial frequency. Therefore invasion implies substitution.