Supplementary material to:

Chapter 12: Evolutionary Invasion Analysis

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Supplementary Material 12.1: Further examples of class-structured evolutionary invasion analyses

In this section we present three further examples of evolutionary invasion analyses involving class structure. The purpose of these examples is two-fold. First, because there are a very large number of different types of class-structured models that are possible, it is useful when first learning these techniques to see as many different examples as possible. Nevertheless, for each example, the general approach is the same (Recipe 12.1). Second, these three examples allow us to illustrate different shortcuts that can be taken to determine the direction of evolution and the value of ESS.

<u>**Example: Evolution of the Sex Ratio**</u>

In many populations females devote equal resources to the production of sons and daughters. A question of long-standing interest is why, from an evolutionary standpoint, this is so (Bulmer 1994; Charnov 1982; Fisher 1958). One idea that comes to mind is that the sex ratio is even because many species have sex chromosomes, which segregate 50:50 among the gametes that determine sex (e.g., 50:50 X and Y sperm in humans). This explanation is not fully satisfying, however, because the sex ratio is nearly 50:50 even in organisms like turtles and crocodiles in which sex is environmentally determined (e.g., by temperature). Furthermore, there are many known mechanisms by which the proportion of successful X- and Y-bearing sperm could differ from 50:50 if such a deviation were favored by selection (e.g., meiotic drive, differential sperm viability or motility). Here, we use an evolutionary invasion analysis to determine the selective forces acting on the sex ratio of a population.

In the model we consider, the sex ratio of offspring is assumed to be under maternal control. For example, the success of X- and Y-bearing sperm might depend on the physical and chemical environment inside a female. The trait of interest is the fraction of available resources that a female devotes to the production of sons, which is sometime called a female's *sex allocation strategy*. This is the decision variable of Step 1, Recipe 12.1. We use s to denote the resident value of this trait and s_m to denote the value in females who carry a mutant allele that alters her sex allocation strategy.

Moving on to Step 2, Recipe 12.1, let's suppose that generations are discrete, that the species in question is diploid, and that the gene altering the sex ratio is on an autosome. In our model, we also incorporate density-dependence by assuming that the amount of resources that a female can devote to reproduction, R, decreases as the number of females in the population increases. We will assume that, provided there are some males in the population, all females will obtain a mate. Finally, we suppose that it takes the same amount of resources to produce a son or a daughter and that R is measured in units that represent the amount of resources required to produce one offspring.

As in previous examples, we begin by specifying a model for the dynamics of the population in the absence of the mutant allele. If the population currently contains a resident allele coding for sex allocation strategy s, then we suppose that the population dynamics of males and females is given by

$$
\begin{aligned}
F(t+1) &= F(t)R_0\, e^{-\alpha F(t)}(1-s) \\
M(t+1) &= F(t)R_0\, e^{-\alpha F(t)}s
\end{aligned}
\tag{S12.1.1}
$$

where $F(t)$ and $M(t)$ represent the number of females and males at time t, and we have used the function $R = R_0\, e^{-\alpha F(t)}$ to model density-dependent resource acquisition in a manner equivalent to the Ricker model (R_0 is a positive parameter representing the amount of resources that a female acquires for reproduction when the number of females in the population is very low, and α measures the strength of density dependence). The only non-zero equilibrium of model (S12.1.1) is

$$\hat{F} = \frac{\ln\left(R_0(1-s)\right)}{\alpha} \quad \hat{M} = \frac{s}{1-s}\frac{\ln\left(R_0(1-s)\right)}{\alpha}.$$
(S12.1.2)

A linear stability analysis reveals that this equilibrium is locally stable provided that $1 < R_0(1-s) < e^2$.

Assuming that the above condition for stability holds, imagine introducing a rare mutant allele into the population. We want to derive an expression for the reproductive factor of this mutant allele. This reproductive factor can be derived from the full model that keeps track of all six different kinds of individuals: resident homozygote males and females, heterozygote males and females, and mutant homozygote males and females. Constructing the full model is not difficult, but it requires that we be careful with our bookkeeping when counting up the various ways in which each type of individual might be produced. This can be quite tedious, and given that we only want to use the model for an invasion analysis while the mutant allele is rare, we don't actually need the full model. Rather, all we really need is the local stability matrix for the equilibrium where the mutant allele is absent. But how can we calculate this local stability matrix without constructing the full model in the first place?

As we have seen, all invasion analyses result in a local stability matrix that has a block upper triangular form:

$$\begin{pmatrix} \mathbf{J}_{res} & \mathbf{V} \\ \mathbf{0} & \mathbf{J}_{mut} \end{pmatrix},$$
(S12.1.3)

where $\mathbf{0}$ is a sub-matrix containing all zeros. The eigenvalues of matrix (S12.1.3) are therefore given by the eigenvalues of sub-matrix, $\mathbf{J}_{res}$, and the eigenvalues of sub-matrix $\mathbf{J}_{mut}$. The eigenvalues of sub-matrix $\mathbf{J}_{res}$ describe the stability of the resident equilibrium in the absence of the mutant strategy. Therefore, the long-term reproductive factor of the mutant allele is always given by the leading eigenvalue of the mutant sub-matrix, $\mathbf{J}_{mut}$. Sometimes, we can use reason to determine what the elements of this sub-matrix are without specifying the full model. This shortcut is commonly used by practicing modelers, so let's see how it might work for our model of sex ratio evolution.

When the mutant allele is rare, there will be virtually no homozygous mutant individuals in a randomly mating population. Therefore, we need only keep track of mutant heterozygotes. Thus, although the full local stability consists of a 6×6 matrix (describing the frequency of the three genotypes, *AA*, *Aa*, and *aa*, in the two sexes), the mutant sub-matrix used in the invasion analysis is only a 2×2 matrix, which tracks how mutant heterozygous males and females produce mutant heterozygous males and females in the next generation.

To construct this mutant sub-matrix, first consider the number of mutant heterozygous offspring produced by a mutant heterozygous mother. All mutant females obtain a mate (by assumption), and these mates will almost always be resident males because the mutant is rare (mutant-mutant matings will be exceedingly rare and can thus be ignored). Again because the mutant allele is rare, the effects of density dependence will be determined almost exclusively by the resident population of females, $\hat{F}$, so that mutant females produce about $R_0 \exp\left(-\alpha \hat{F}\right)$ offspring. Now because mothers determine the sex ratio of their offspring (by assumption), a proportion, $1 - s_m$, will be daughters and the remainder, s_m, will be sons. Finally, because the mutant mother is a heterozygote and her mate is a resident homozygote, only half of these offspring carry the mutant allele. Thus, the total number of mutant daughters produced by a mutant mother is $R_0 \exp\left(-\alpha \hat{F}\right)(1 - s_m)/2$, and the total number of mutant sons is $R_0 \exp\left(-\alpha \hat{F}\right)s_m/2$.

Next, let's consider the number of mutant heterozygous offspring produced by a mutant heterozygous father. Not all males necessarily obtain a mate, and in fact, the number of mates that any male can expect to have is given by the ratio of the number of females to males in the population, because all females are assumed to mate. For example, if there are twice as many females as males, then, on average, a male will obtain two mates. Conversely, if there are twice as many males as females then, on average, only every other male will obtain a mate. Thus, because the mutant allele is rare, the expected number of mates that a mutant male will obtain is very nearly $\hat{F}/\hat{M}$, the ratio of resident females to competing resident males. Any mate that a mutant male does obtain will almost always be a resident female, and she will produce a total of $R_0 \exp\left(-\alpha \hat{F}\right)$ offspring. The sex ratio of their offspring will be determined by the resident

mother's sex allocation strategy, so a fraction $1 - s$ will be daughters and s will be sons. Finally, because the mutant father is a heterozygote and his mate will almost always be a resident homozygote, only half of the offspring produced will carry the mutant allele. Thus, the total number of mutant daughters produced by a mutant father is $\frac{\hat{F}}{\hat{M}} R_0 \exp(-\alpha \hat{F})(1-s)/2$, and the total number of mutant sons is $\frac{\hat{F}}{\hat{M}} R_0 \exp(-\alpha \hat{F}) s/2$. These arguments are summarized in Table S12.1.1.

Table S12.1.1: Offspring produced by mutant parents with selection on the sex ratio. Calculations assume that the mutant allele is rare and that all matings by mutant mothers are with resident males and that all matings by mutant fathers are with resident females.

	# Mutant Daughters Produced	# Mutant Sons Produced
Mutant Mothers	Per mutant mother = $R_0 e^{-\alpha \hat{F}}$ % female = $1 - s_m$ % mutant = 50% Total = $R_0 e^{-\alpha \hat{F}} \frac{(1 - s_m)}{2}$	Per mutant mother = $R_0 e^{-\alpha \hat{F}}$ % male = s_m % mutant = 50% Total = $R_0 e^{-\alpha \hat{F}} \frac{s_m}{2}$
Mutant Fathers	Per mutant father = $\frac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}}$ % female = $1 - s$ % mutant = 50% Total = $\frac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \frac{(1 - s)}{2}$	Per mutant father = $\frac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}}$ % male = s % mutant = 50% Total = $\frac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \frac{s}{2}$

According to the above logic, the dynamics of the mutant females and males in the population are given by

$$F_m(t+1) = F_m(t)R_0 \exp\left(-\alpha \hat{F}\right)\frac{(1-s_m)}{2} + M_m(t)\frac{\hat{F}}{\hat{M}}R_0 \exp\left(-\alpha \hat{F}\right)\frac{(1-s)}{2}$$

$$M_m(t+1) = F_m(t)R_0 \exp\left(-\alpha \hat{F}\right)\frac{s_m}{2} + M_m(t)\frac{\hat{F}}{\hat{M}}R_0 \exp\left(-\alpha \hat{F}\right)\frac{s}{2}$$

(S12.1.4)

Equation (S12.1.4) can be written in matrix form as

$$\begin{pmatrix} F_m(t+1) \\ M_m(t+1) \end{pmatrix} = \mathbf{J}_{mut}\begin{pmatrix} F_m(t) \\ M_m(t) \end{pmatrix}$$

(S12.1.5a)

where

$$\mathbf{J}_{mut} = \frac{R_0 \exp\left(-\alpha \hat{F}\right)}{2}\begin{pmatrix} 1-s_m & \dfrac{\hat{F}}{\hat{M}}(1-s) \\ s_m & \dfrac{\hat{F}}{\hat{M}}s \end{pmatrix}.$$

(S12.1.5b)

$\mathbf{J}_{mut}$ is the mutant sub-matrix that we need to determine the growth rate of the mutant allele. Equation (S12.1.5b) can be simplified further by substituting in the values for $\hat{F}$ and $\hat{M}$ from (S12.1.2) to give

$$\mathbf{J}_{mut} = \frac{1}{2}\begin{pmatrix} \dfrac{1-s_m}{1-s} & \dfrac{1-s}{s} \\ \dfrac{s_m}{1-s} & 1 \end{pmatrix}.$$

(S12.1.5c)

At this point there are two checks that we should perform in order to ensure that no mistakes have found their way into the analysis. The first check is to make sure that the elements of the mutant sub-matrix do not contain any of the mutant variables (here, F_m or M_m). Recall from Chapter 8 that the local stability matrix represents a linear system of equations that approximately describe the dynamics near an equilibrium. Therefore, the local stability matrix should be a constant that does not depend on the number of mutants. Matrix (S12.1.5c) passes this check.

The second check is to make sure that, when we set the mutant allele to code for the same trait value as the resident allele, the matrix has a leading eigenvalue of one in a discrete-time model (zero in a continuous-time model). This requirement follows from the fact that the mutant

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allele should then be completely neutral, neither increasing nor decreasing in frequency. For matrix (S12.3.5c), setting $s_m = s$ gives

$$\frac{1}{2}\begin{pmatrix} 1 & \dfrac{1-s}{s} \\ \dfrac{s}{1-s} & 1 \end{pmatrix},$$

(S12.1.5d)

☐ whose eigenvalues are zero and one. Therefore, the mutant sub-matrix (S12.1.5c) passes this check as well.

Recipe S12.1: Checking the Derivation of the Mutant Sub-matrix

After deriving the mutant sub-matrix whose leading eigenvalue describes the growth of a mutant allele, two checks help guard against errors.

Check 1: The mutant sub-matrix should not contain any of the mutant *variables*. The reason is that this sub-matrix specifies a linear system of equations that approximates the dynamics of the mutant allele when it is rare (i.e., it is a local stability analysis; see Chapter 8).

Check 2: When the mutant allele codes for the same trait value as the resident allele, the leading eigenvalue of the mutant sub-matrix should equal one for discrete-time models and zero for continuous-time models (assuming that the mutant is introduced into a population of residents at a stable equilibrium). In this case, the mutant allele is completely neutral, neither increasing nor decreasing in number.

Deriving the mutant sub-matrix in this way often saves a lot of time and avoids unnecessary headache. Of course, if you want to know what happens after the mutant allele becomes common, you will need the full set of equations. Plus, if homozygous mutant carriers are no rarer than heterozygous mutant carriers (e.g., when parents can self-fertilize), then again you must derive the full set of equations. Once the mutant sub-matrix is derived, we proceed with the analysis as usual. The conditions under which a mutant allele can invade are determined by the leading eigenvalue of the mutant sub-matrix, (S12.1.5c). If the resident population has an ESS sex allocation, s^*, then this eigenvalue will satisfy $\lambda(s_m, s^*) < \lambda(s^*, s^*)$ for any mutant

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allocation strategy, s_m that differs from s^*. In particular, an ESS must satisfy the first derivative condition (12.15c), $\partial\lambda/\partial s_m\big|_{\substack{s_m=s^*\\s=s^*}}=0$, in Recipe 12.2 of Chapter 12.

Rather than calculate the ESS from the leading eigenvalue (see Problem S12.2), let's again illustrate the technique presented in Recipe 12.5. Using matrix (S12.1.5c), the first derivative condition for an ESS is equivalent to setting the fitness gradient to zero:

$\bar{v}^{\mathrm{T}}\dfrac{\partial \mathbf{J}_{mut}}{\partial s_m}\bar{u}=0$, where $s_m=s^*$ and $s=s^*$. To use this condition, we need to calculate the left and right eigenvectors of matrix (S12.3.5c) when $s_m=s^*$ and $s=s^*$, at which point the leading eigenvalue is one. The eigenvectors are $\bar{v}=\begin{pmatrix} s^* \\ 1-s^* \end{pmatrix}$ and $\bar{u}=\begin{pmatrix} 1-s^* \\ s^* \end{pmatrix}$ (or some multiple thereof).

We also need to calculate $\dfrac{\partial \mathbf{J}_{mut}}{\partial s_m}\bigg|_{\substack{s_m=s^*\\s=s^*}}$, which equals

$$\frac{\partial \mathbf{J}_{mut}}{\partial s_m}\bigg|_{\substack{s_m=s^*\\s=s^*}}=\frac{1}{2}\begin{pmatrix} -\dfrac{1}{1-s^*} & 0 \\ \dfrac{1}{1-s^*} & 0 \end{pmatrix}. \qquad\qquad (S12.1.6)$$

Carrying out the matrix multiplication, $\bar{v}^{\mathrm{T}}\dfrac{\partial \mathbf{J}_{mut}}{\partial s_m}\bar{u}=0$, gives $-\dfrac{s^*}{2}+\dfrac{1-s^*}{2}=0$. Thus, the ESS sex ratio in our model is $s^*=1/2$. At ESS, we expect 50% males and 50% females. We leave it as an exercise to examine the second derivative condition and convergence stability condition in Recipe 12.2 (see Problem S12.2).

The above analysis indicates that sex ratios other than $s^*=1/2$ can be invaded by mutant strategies. Indeed, we can use the mutant sub-matrix $\mathbf{J}_{mut}$ to show that any mutant allele that increases the proportion of males ($s_m>s$) can invade when the sex ratio is female-biased ($s<1/2$), while any mutant allele that decreases the proportion of males ($s_m<s$) can invade when the sex ratio is male-biased ($s>1/2$) (see Problem S12.3). Intuitively, if there are more males in a population than females, any mutant allele that increases sex allocation to females benefits by reducing competition among sons for access to the rarer sex. Conversely, if there are more females in a population than males, any mutant allele that increases sex allocation to males

benefits because there is relatively little competition among males for access to the more common sex. Thus, selection favors mutations that increase the frequency of the rarer sex and brings a population toward an even sex ratio. (See Hamilton 1967 for interesting situations under which a non-even sex ratio can evolve, as well as Problem S12.5)

Example: Intralocus sexually antagonistic selection

Sexually antagonistic selection occurs when different values of a trait are favored in males versus females. For example, in many species females and males tend to have very different sizes. In mammals, males are typically larger than females whereas in many insects and fish the opposite pattern occurs. One explanation for this sexual size dimorphism is that natural or sexual selection favors different body sizes in females versus males. Sexually antagonistic selection is termed "intralocus" when the same locus controls the trait of interest in both males and females. Conversely, it is termed interlocus if there are different loci coding for the trait of interest in males and females (Rice and Chippindale 2001). Here we construct a model for the evolution of body size, focusing on a single body-size locus subject to sexually antagonistic selection. The following example then explores a case of inter-locus sexually antagonistic selection.

Imagine an ancestral population in which males and females have the same body size, and in which natural selection favors larger females than males. Specifically, let's suppose that the probability of survival of a female offspring, p_f, increases with the amount, x, by which her body size is above some minimum, whereas that of a male offspring, p_m, decreases with x. Now imagine that a mutant allele coding for a different body size, x_m, in both males and females appears in the population. We are interested in determining the conditions under which this allele will spread.

Intuitively, the mutant allele should spread if the benefit it confers to females more than outweighs the cost borne by males. This conclusion is relatively straightforward to understand if the mutant allele is found on an autosome, but many sexually antagonistic alleles are found on the X chromosome in species with XY sex determination (Gibson *et al.* 2002). Although the cost-benefit argument should still apply, understanding exactly how the costs and benefits balance for X-linked genes is more difficult for two reasons. First, 2/3 of all the X chromosomes

in the population will be found in females (because females are XX and males are XY). It seems plausible that this will affect the cost-benefit balance, but does it simply weight it by 2/3? Second, we might expect the effects of dominance of the mutant allele to have an important impact on its ability to invade because the mutant allele will be fully expressed in males (assuming the Y chromosome lacks a homologous copy of the gene), whereas its level of dominance will affect its expression in females. But how, exactly, do all of these factors fit together? This is where a mathematical model becomes crucial (Rice 1984).

Again, following Recipe 12.1, we begin by specifying the dynamics of the population in the absence of the mutant allele. For simplicity, let's follow the same assumptions used in model (S12.1.1) for sex ratio evolution. We suppose that all females always obtain a mate and that the total offspring production of a female is density-dependent and is given by $R_0 \exp(-\alpha F(t))$. We further suppose that the sex ratio is 50:50 at birth, in which case our resident model takes the form

$$
\begin{aligned}
F(t+1) &= F(t)R_0 \exp(-\alpha F(t)) \tfrac{1}{2} p_f(x) \\
M(t+1) &= F(t)R_0 \exp(-\alpha F(t)) \tfrac{1}{2} p_m(x)
\end{aligned}
\tag{S12.1.7}
$$

where $p_f(x)$ and $p_m(x)$ are the sex-specific probabilities of survival, which are functions of the resident body size, x. The equilibrium number of adult females and males is given by

$$
\hat{F} = \frac{\ln\left(\tfrac{1}{2} p_f(x) R_0\right)}{\alpha} \qquad \hat{M} = \frac{p_m(x)}{p_f(x)} \frac{\ln\left(\tfrac{1}{2} p_f(x) R_0\right)}{\alpha}.
\tag{S12.1.8}
$$

We then imagine introducing an X-linked mutant allele that codes for a different body size. We need to derive an expression for the reproductive factor of this mutant allele.

Rather than specifying a complete model for the joint dynamics of the two different alleles, we proceed directly to constructing the mutant sub-matrix as we did for the model of sex ratio evolution. Because the mutant allele is rare and we assume random mating, we need only keep track of mutant heterozygotes. Thus, the mutant sub-matrix will again be a 2×2 matrix, whose elements describe the number of heterozygous mutant males and females produced by each heterozygous mutant male and female in one time step.

The process of counting up the number of mutant females and males produced by a mutant mother, as well as those produced by a mutant father, is very similar to that for the sex ratio model (see Table S12.1.2 and Figure S12.1.1). First consider the number of mutant heterozygous offspring produced by a mutant heterozygous mother. All mutant females mate with a resident male and produce a total of $R_0 \exp(-\alpha \hat{F})$ offspring, 1/2 of which are daughters and 1/2 of which are sons. Furthermore, because the mutant allele is X-linked, only 1/2 the daughters and 1/2 the sons will inherit the mutant allele (Figure S12.1.1a). Finally, offspring survive with a probability that depends on their body size. We can describe the body size of a female carrying one mutant and one resident allele as $x_{het} = h x_m + (1-h)x$, where h is the degree of dominance of the mutant allele, so that female mutant offspring survive with probability $p_f(x_{het})$. Because males are hemizygous at an X-linked body-size locus (Figure S12.1.1), the body size of mutant males is just x_m, and such mutant sons survive with probability $p_m(x_m)$.

Table S12.1.2: Offspring produced by mutant parents with sexually antagonistic selection on body size. Calculations assume that the mutant allele is rare and that all matings by mutant mothers are with resident males and that all matings by mutant fathers are with resident females. See also Figure S12.1.1.

	# Mutant Daughters Produced	# Mutant Sons Produced
Mutant Mothers	Per mutant mother = $R_0 e^{-\alpha \hat{F}}$ % female = 50% % mutant = 50% survival probability = p_f Total = $\frac{1}{4} R_0 e^{-\alpha \hat{F}} p_f$	Per mutant mother = $R_0 e^{-\alpha \hat{F}}$ % male = 50% % mutant = 50% survival probability = p_m Total = $\frac{1}{4} R_0 e^{-\alpha \hat{F}} p_m$
Mutant Fathers	Per mutant father = $\frac{\hat{F}}{M} R_0 e^{-\alpha \hat{F}}$ % female = 50% % mutant = 100% survival probability = p_f Total = $\frac{1}{2} \frac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} p_f$	Per mutant father = $\frac{\hat{F}}{M} R_0 e^{-\alpha \hat{F}}$ % male = 0% % mutant = NA survival probability = NA Total = 0

Figure S12.1.1: Inheritance of mutant sex chromosomes. The patterns of inheritance are shown for matings between (a) mutant females and resident males and (b) resident females and mutant males. White and black blocks represent resident and mutant X chromosomes, respectively. Males are XY and therefore always pass their X chromosome to daughters and their Y chromosome to sons. Females are XX and have a 50:50 chance of passing any one of their X chromosomes to an offspring, regardless of the sex of the offspring.
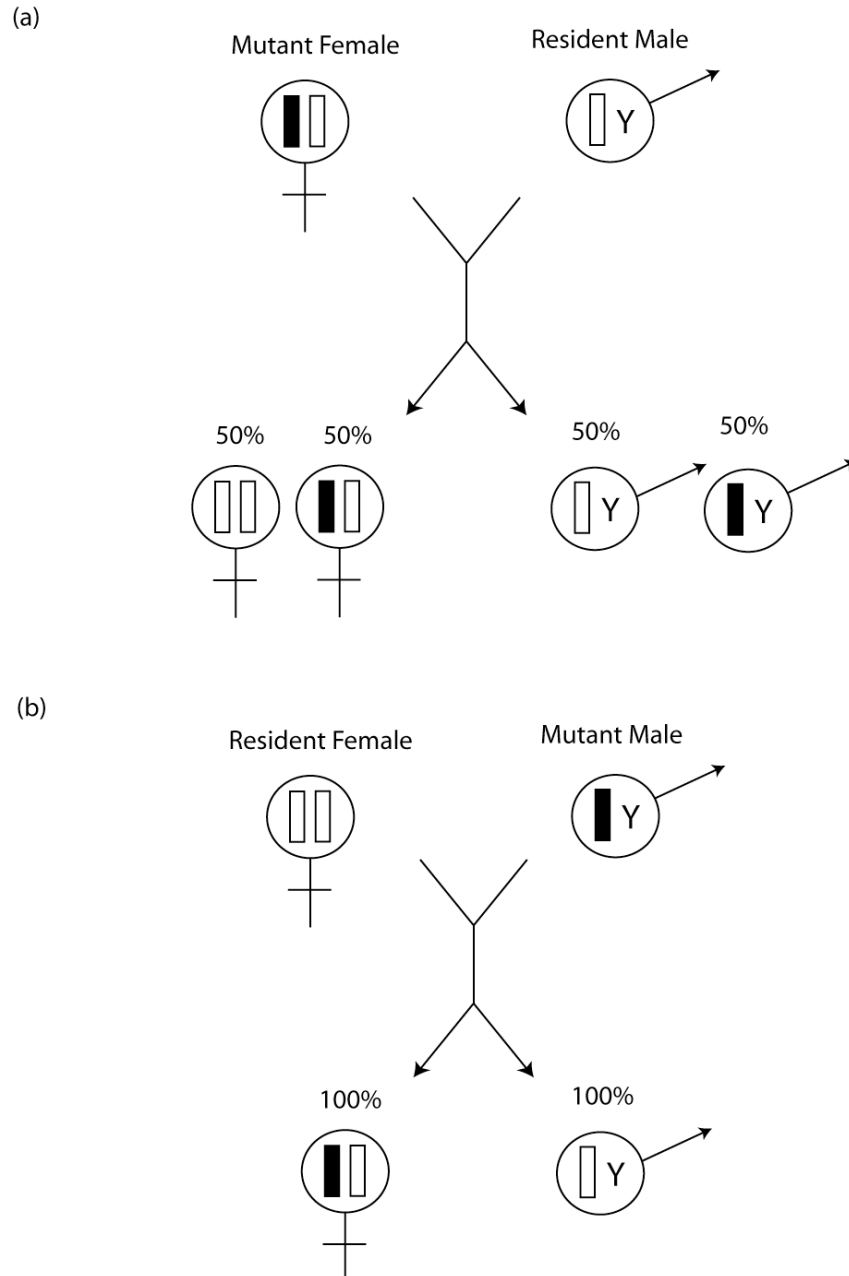
Similar considerations apply to a mutant father except that the expected number of mates that a mutant male will obtain is given by $\hat{F}/\hat{M}$, as it was in the sex ratio model. Now however, because mutant fathers will almost certainly mate with resident mothers, all of their female offspring will carry the mutant allele, because daughters are created from sperm carrying their father's X chromosome (Figure S12.1.1). Conversely, none of the male offspring produced by a rare mutant father will carry the mutant allele, because sons are created from sperm carrying their father's Y chromosome (Figure S12.1.1b). Finally, the mutant daughters survive to adulthood with probability $p_f(x_{het})$, as before.

The above patterns of offspring production are summarized in Table S12.1.2 and result in the following equations for the number of mutant females and males, F_m and M_m:

$$F_m(t+1) = F_m(t)R_0 \exp(-\alpha\hat{F})\frac{1}{4}p_f(x_{het}) + M_m(t)\frac{\hat{F}}{\hat{M}}R_0\exp(-\alpha\hat{F})\frac{1}{2}p_f(x_{het})$$

$$M_m(t+1) = F_m(t)R_0\exp(-\alpha\hat{F})\frac{1}{4}p_m(x_m)$$

(S12.1.9)

After substituting the values of $\hat{F}$ and $\hat{M}$ from equations (S12.1.2) into equations (S12.1.9), the dynamics can be written in matrix form as

$$\begin{pmatrix} F_m(t+1) \\ M_m(t+1) \end{pmatrix} = \mathbf{J}_{mut}\begin{pmatrix} F_m(t) \\ M_m(t) \end{pmatrix}$$

(S12.1.10a)

where

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{1}{2}\dfrac{p_f(x_{het})}{p_f(x)} & \dfrac{p_f(x_{het})}{p_m(x)} \\ \dfrac{1}{2}\dfrac{p_m(x_m)}{p_f(x)} & 0 \end{pmatrix}.$$

(S12.1.10b)

Matrix (S12.1.10b) is the mutant sub-matrix for this model. Before proceeding to the analysis of the matrix, let's first perform the two checks in Recipe S12.1. First, we must ensure that matrix (S12.1.10b) does not contain either of the variables F_m or M_m, which it does not. Second, we must ensure that matrix (S12.1.10b) has a leading eigenvalue of one when the mutant

body size equals the resident body size. Setting $x_m = x$ in $x_{het} = hx_m + (1-h)x$ and then in (S12.1.10b) gives

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{1}{2}\dfrac{p_f(x)}{p_f(x)} & \dfrac{p_f(x)}{p_m(x)} \\[2mm] \dfrac{1}{2}\dfrac{p_m(x)}{p_f(x)} & 0 \end{pmatrix}, \tag{S12.1.10c}$$

whose eigenvalues are $-1/2$ and 1. Therefore, matrix (S12.1.10b) passes the second check as well.

We would now like to determine the conditions under which a mutant allele can invade. The long-term reproductive factor of the mutant allele is given by the leading eigenvalue of matrix (S12.1.10b). This eigenvalue can be calculated using the methods of section P2.8 in Primer 2, but let's again use Recipe 12.5 to calculate the fitness gradient for x.

From Recipe 12.5, we need to calculate $\vec{v}^{\,\mathrm{T}} \left.\dfrac{\partial \mathbf{J}_{mut}}{\partial x_m}\right|_{x_m=x} \vec{u}$, where $\vec{v}$ and $\vec{u}$ are the leading left and right eigenvectors associated with the leading eigenvalue of one of $\mathbf{J}_{mut}(x,x)$. These eigenvectors evaluate to $\vec{v} = \begin{pmatrix} p_m(x) \\ p_f(x) \end{pmatrix}$ and $\vec{u} = \begin{pmatrix} 2p_f(x) \\ p_m(x) \end{pmatrix}$. We can also calculate $\left.\dfrac{\partial \mathbf{J}_{mut}}{\partial x_m}\right|_{x_m=x}$ and put these results together to obtain

$$\left.\frac{\partial \lambda}{\partial x_m}\right|_{x_m=x} \propto 2h\, p_m \frac{\mathrm{d}p_f}{\mathrm{d}x} + p_f \frac{\mathrm{d}p_m}{\mathrm{d}x}. \tag{S12.1.11}$$

Therefore, a mutant allele that increases body size will invade only if

$$h\left(\frac{1}{p_f}\frac{\mathrm{d}p_f}{\mathrm{d}x}\right) > -\frac{1}{2}\left(\frac{1}{p_m}\frac{\mathrm{d}p_m}{\mathrm{d}x}\right). \tag{S12.1.12}$$

Condition (S12.1.12) can be better interpreted by noting that $(\mathrm{d}p_f/\mathrm{d}x)/p_f$ is the proportional increase in survival probability of females that comes from a unit increase in body size. Similarly, $(\mathrm{d}p_m/\mathrm{d}x)/p_m$ is the proportional decrease in survival probability of males that comes

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from a unit increase in body size. Therefore, introducing the simpler notation $b_f = (dp_f/dx)/p_f$ for the benefits to females and $c_m = -(dp_m/dx)/p_m$ for the costs to males of increasing body size, condition (S12.1.12) can be re-written as $hb_f > c_m/2$. This reveals that, if the costs to males are roughly equivalent in magnitude to the benefits to females ($b_f = c_m$), then the mutant allele can invade only if it is dominant (i.e., only if $h > 1/2$). This suggests that, all else being equal, female-benefit alleles that are found on the X chromosome should tend to be dominant (Rice 1984). Conversely, we can carry out the same line of reasoning for an allele that reduces body size and come to the conclusion that male-benefit alleles that are found on the X chromosome should tend to be recessive. The simplicity of these predictions is very pleasing, and newly available techniques in molecular biology are now allowing these predictions to be tested (Parisi *et al.* 2003; Ranz *et al.* 2003).

Example: Interlocus sexually antagonistic selection in water striders

Our final example introduces a new feature, in which we suppose that individuals of different classes exhibit different traits, each coded for by its own locus. We are interested in the coevolution of these two traits. Similar techniques can also be used to model the coevolution of interacting species (see Problem S12.9).

Water striders are insects that live on the surface of many ponds, streams and marshes. Their name stems from the fact that they are able to walk on the top of water using its surface tension (Denny 2004; Hu *et al.* 2003). The mating system of water striders has been the subject of considerable research, and their behavior and morphology have been well characterized. Interestingly, the males of some species have long and elaborate grasping appendages that they use to hold on to females during mating attempts, and the females of these species have long spines on their abdomens that keep these males at a particular distance from their bodies. In species where males have short grasping appendages, the females tend to have short spines. As a result, the effectiveness of male grasping appendages at holding females during mating is roughly the same for different species despite there being large differences in the sizes of male graspers and female spines (Arnqvist and Rowe 2002; Rowe and Arnqvist 2002). It appears as though the relative size of male and female traits has evolved so that they match one another regardless of their absolute sizes.

Recently, however, a less harmonious explanation for this pattern has been proposed. The mating system of many species of water striders is characterized by intense competition among males for access to females, such that males incessantly harass females with mating attempts (Rowe *et al*. 1994). When males are successful at mating, they hold the female with their graspers for a period of time, and this places the female at a higher risk of predation from aquatic predators. Because females typically require very few matings to fertilize all of their eggs and because mating places females at greater risk of predation, female water striders prefer to mate less often and for shorter periods of time than males. As a result, it has been hypothesized that males evolve longer and more elaborate grasping appendages in order to increase their mating success when in competition with other males, despite the fact that these male traits impose a cost to females. Females have then evolved longer spines as a counter adaptation to reduce the likelihood that males will be able to hold on to them.

This coevolutionary arms race provides an interesting potential explanation for the patterns seen in many water strider species. There is, however, one nagging thought about this explanation. All genes will find themselves in males 1/2 of the time and in females 1/2 of the time (assuming a 50:50 sex ratio). Does the fact that genes spend equal amounts of time in males and females prevent the coevolutionary arms race? To see whether or not the explanation is plausible, let's formalize the hypothesis mathematically using an evolutionary invasion analysis. In this example, let's suppose that two genes are involved; one coding for the length of male grasping appendages and the other coding for the length of female spines. Both sexes carry both genes, but only the male trait gene is expressed by males and vice versa. We will suppose that the sex ratio is fixed at 50:50 and not worry about how the sex of an individual is determined.

Let's begin by specifying a model for the dynamics of the population when fixed for a resident allele. We use x to denote the length of female spines coded for by the resident allele at the spine locus, and y to denote the length of grasping appendages coded for by the resident allele at the grasper locus. Suppose that each year all females mate at least once, then they produce offspring and die. We also suppose that the total number of offspring produced by a female declines as the population size increases as a result of density dependence, and we model

this using the function $R_0 e^{-\alpha F(t)}$ where R_0 is a positive constant, F is the density of females, and α measures the strength of density dependence.

A female's offspring production will depend on her spine length relative to the length of the grasping appendages of males in the population. If her spines are relatively short, then males of the population will be readily able to hold on to her. This will result in a higher predation risk for the female. On the other hand, if a female's spines are extremely long, then very few males will be able to mate with her, and again her reproductive output will be low owing to a lack of fertilized eggs. As a result, we expect female offspring production to be highest when spine length "matches" the length of male grasping appendages. We use the function, $\phi(d)$, to represent how female offspring production depends on the difference, d, between female spine length and male appendage length, $d = x - y$. We assume that the function $\phi(d)$ reaches its maximum when the spines and graspers are perfectly matched ($d = 0$).

With these assumptions, female fecundity during a single season is a function of her spine length, the average male appendage length, and the female population size: $R_0 e^{-\alpha F(t)} \phi(x - y)$. The dynamics of the resident population is therefore given as

$$\begin{aligned} F(t+1) &= \frac{F(t)R_0 e^{-\alpha F(t)} \phi(x - y)}{2} \\ M(t+1) &= \frac{F(t)R_0 e^{-\alpha F(t)} \phi(x - y)}{2} \end{aligned}, \qquad \text{(S12.1.13)}$$

where $F(t)$ and $M(t)$ represent the number of females and males at time t, and the 1/2 stems from the assumption that the sex ratio is 50:50. The only non-zero equilibrium of model (S12.1.13) is given by

$$\hat{M} = \hat{F} = \frac{1}{\alpha} \ln\left(R_0 \phi(x - y)/2\right). \qquad \text{(S12.1.14)}$$

A local stability analysis shows that this equilibrium is stable provided that
$2 < R_0 \phi(x - y) < 2e^2$.

The next step in an invasion analysis is to introduce a mutant allele. Except for the model of dominance in section 12.5 of Chapter 12, all of the examples considered so far have had only a single gene. Even in the two-gene example of dominance, there was still only one gene at which we introduced a mutant allele. The second gene simply served to create a form of class structure. Now, however, we are interested in introducing mutant alleles at both genes because we are interested in the evolution of both traits.

Conceptually, there are two types of invasion analyses that can be performed to understand the evolution of two traits that are coded for by separate loci. One approach is sequential: mutants are introduced at only one locus at a time, and loss or fixation of the mutant occurs before the next mutant appears. A sequential analysis allows the state of one trait to change and to alter the subsequent evolution of the other trait, but it does not allow genetic associations to develop between alleles at the two loci (i.e., linkage disequilibrium). A second approach is simultaneous: mutants are allowed to segregate at the same time at both loci. This second approach requires a more sophisticated analysis (i.e., you must consider genetic associations among the alleles, and you must specify the relative timing at which mutations appear at the two loci). Here we focus on a sequential analysis, which essentially involves following the steps of Recipe 12.1 separately for each trait.

Let's first consider the male side of the story, and imagine introducing a rare mutant allele into the population that codes for longer grasping appendages at the grasping locus. In doing so we assume that an allele coding for spine length x remains fixed at the spine locus. We want to derive an expression for the reproductive factor of the mutant allele. We proceed to construct the 2×2 mutant sub-matrix that tracks how mutant heterozygous males and females produce mutant heterozygous males and females in the next generation, just as we did in previous examples. We suppose that there is competition between males for access to females such that the number of mates that a male can expect in his lifetime is given by the ratio of the number of females to males in the population. In addition to this, however, we also want to model the fact that males whose graspers are relatively long compared with those of other males in the population will have an increased likelihood of successfully mating. We do this by assuming that the number of mates that a mutant male with appendage length y_m obtains is given

by $\dfrac{\hat{F}}{\hat{M}} g(y_m - y)$. The function $g(y_m - y)$ is a factor specifying how the number of mates the mutant obtains depends on its relative grasper length. If the mutant's graspers are the same length as those of resident males ($y_m = y$), then the function g should equal one because all males are equivalent. On the other hand, if the mutant's graspers are relatively short ($y_m < y$), then g should be less than one whereas if they are relatively long ($y_m > y$), then g should be greater than one.

With these assumptions we can now construct the mutant sub-matrix. Mutant males obtain $\dfrac{\hat{F}}{\hat{M}} g(y_m - y)$ mates, almost all of which will be resident females, each of which produces $R_0 \, e^{-\alpha \hat{F}} \phi(x - y)$ offspring. Here we assume that female reproductive success depends on the resident male grasper length rather than the mutant grasper length because nearly all the other mates of this female will be resident males (because the mutant is rare), and it is the overall number of mating events that determines her fitness. (This is not the only plausible assumption, and other assumptions could be explored by altering $\phi(x - y)$ in this equation.) One half of the offspring produced will be male and one half female, and because the mutant father is a heterozygote and his mates are resident homozygotes, only 1/2 of these will carry the mutant allele. Thus, the total number of mutant daughters and sons produced by a mutant father are both equal to

$$\frac{1}{4} \frac{\hat{F}}{\hat{M}} g(y_m - y) R_0 \, e^{-\alpha \hat{F}} \phi(x - y).$$

Females also carry unexpressed copies of the mutant allele, and all mutant females will obtain a mate (by assumption). These mates will primarily be resident males, and they will therefore produce a total of $R_0 \, e^{-\alpha \hat{F}} \phi(x - y)$ offspring. Again one half of these offspring will be male and one half female, and only 1/2 of the offspring will carry the mutant allele. Thus, the number of mutant daughters and sons produced by a mutant mother both equal $R_0 \, e^{-\alpha \hat{F}} \phi(x - y)/4$.

Putting all of these terms together, the dynamics of a rare mutant that alters the male grasping trait is described by:

$$\begin{pmatrix} F_m(t+1) \\ M_m(t+1) \end{pmatrix} = \mathbf{J}_{mut} \begin{pmatrix} F_m(t) \\ M_m(t) \end{pmatrix},$$
(S12.1.15a)

where

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x-y) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} g(y_m - y) R_0 e^{-\alpha \hat{F}} \phi(x-y) \\ \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x-y) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} g(y_m - y) R_0 e^{-\alpha \hat{F}} \phi(x-y) \end{pmatrix}$$
(S12.1.15b)

is the mutant sub-matrix. Matrix (S12.1.15b) can be simplified further by substituting in the values for $\hat{F}$ and $\hat{M}$ from (S12.1.14) to give

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{1}{2} & \dfrac{g(y_m - y)}{2} \\ \dfrac{1}{2} & \dfrac{g(y_m - y)}{2} \end{pmatrix}.$$
(S12.1.15c)

Matrix (S12.1.15c) passes both checks in Recipe S12.1: $\mathbf{J}_{mut}$ does not involve the mutant variables F_m or M_m, and its leading eigenvalue is one when $y_m = y$ because $g(0) = 1$.

The eigenvalues of $\mathbf{J}_{mut}$ can be readily calculated, giving 0 and $\dfrac{1}{2}(1 + g(y_m - y))$.

☐ Consequently, the reproductive factor of a mutant allele that affects the male grasping trait is:

$$\lambda_{males}(y_m, y) = \frac{1}{2}(1 + g(y_m - y)).$$
(S12.1.16)

This completes Step 2 of Recipe 12.1, but before proceeding further we should derive the reproductive factor of a mutant allele affecting female spine length as well. In this derivation, we imagine introducing a mutant allele at the spine locus while holding the grasper locus fixed at the value y. We then construct the 2×2 mutant sub-matrix that tracks how mutant heterozygous males and females produce mutant heterozygous males and females in the next generation for the locus affecting spine length.

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All mutant females obtain at least one mate, and these mates will almost always be resident males. If the mutant female spine length is x_m, then these females will produce a total of $R_0 e^{-\alpha \hat{F}} \phi(x_m - y)$ offspring. One half of these offspring will be male and one half female, and only 1/2 of these offspring will carry the mutant allele. Thus, the number of mutant daughters and sons produced by a mutant mother both equal $R_0 e^{-\alpha \hat{F}} \phi(x_m - y)/4$.

Males will also carry unexpressed copies of the mutant allele, and these males can expect to obtain $\dfrac{\hat{F}}{\hat{M}}$ mates, almost all of which will be resident females. These females produce a total of $R_0 e^{-\alpha \hat{F}} \phi(x - y)$ offspring, and again one half of these offspring will be male and one half female. Only 1/2 of these offspring will carry the mutant allele, and thus the total number of mutant daughters and sons produced by a mutant father both equal $\dfrac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \phi(x - y)/4$.

The dynamics of a mutant allele that alters female spine length is therefore described by:

$$\begin{pmatrix} F_m(t+1) \\ M_m(t+1) \end{pmatrix} = \mathbf{J}_{mut} \begin{pmatrix} F_m(t) \\ M_m(t) \end{pmatrix},$$

(S12.1.17a)

where the mutant sub-matrix is now

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x_m - y) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \phi(x - y) \\ \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x_m - y) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \phi(x - y) \end{pmatrix}.$$

(S12.1.17b)

Matrix (S12.1.17b) can be simplified further by substituting in the values for $\hat{F}$ and $\hat{M}$ from (S12.1.14) to give

$$\mathbf{J}_{mut} = \begin{pmatrix} \dfrac{\phi(x_m - y)}{2\,\phi(x - y)} & \dfrac{1}{2} \\ \dfrac{\phi(x_m - y)}{2\,\phi(x - y)} & \dfrac{1}{2} \end{pmatrix}.$$

(S12.1.17c)

Again, you can perform the two checks in Recipe S12.1 and confirm that matrix (S12.1.17c) passes both tests.

The eigenvalues of $\mathbf{J}_{mut}$ are 0 and $\frac{1}{2}\left(1+\frac{\phi(x_m-y)}{\phi(x-y)}\right)$. Consequently, the reproductive factor of a mutant allele altering female spine length is

$$\lambda_{females}(x_m,y)=\frac{1}{2}\left(1+\frac{\phi(x_m-y)}{\phi(x-y)}\right). \tag{S12.1.18}$$

At this stage we have expressions for the reproductive factor of mutant alleles at each locus assuming that the other locus remains fixed. We can now determine the direction of evolution in both males and females. A mutant male grasper allele will invade provided that $\lambda_{males}(y_m,y)>1$. Using expression (S12.1.16) in this condition gives $\frac{1}{2}(1+g(y_m-y))>1$ or $g(y_m-y)>1$. Recalling that the function g describes the relative success of a male in gaining mates, and given the assumption that longer graspers always improve male fitness, this condition will always be satisfied for values of y_m that are larger than y. Thus, the mating advantage that males receive from longer graspers results in continual selection for longer graspers.

What about the female side of the story? A mutant female spine allele will invade provided that $\lambda_{females}(x_m,y)>1$. Using expression (S12.1.18) in this condition gives $\frac{1}{2}\left(1+\frac{\phi(x_m-y)}{\phi(x-y)}\right)>1$ or $\phi(x_m-y)>\phi(x-y)$. Thus, selection favors the female spine length, x, that maximizes the function $\phi(x-y)$. We have assumed that the female fitness is highest when her spine length exactly matches male grasper length ($x=y$), and therefore female spines will continually evolve to be longer in response to the ever-increasing male grasper length. As a result, we might expect different species to be at different stages of this evolutionary escalation in trait size, but nevertheless the male graspers and female spines of each species should approximately match.

In our description of the coevolutionary arms race hypothesis we pointed out that all genes spend just as much time in females as they do in males, and we might expect this fact to cancel out the effects of sexually antagonistic selection. Interestingly, our analysis indicates that

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this objection is logically flawed in some way, but how? This is where mathematical modeling really proves its value. In particular, we can retrace our steps in the derivation to see where this objection goes wrong.

The objection was based on the idea that, although a mutant allele increasing grasper length will be beneficial in males, it will also be found in females half the time. Moreover, it will be selected against in such females who are harmed by males with longer graspers. The mutant sub-matrix (S12.1.15b) was constructed accounting for the fact that mutant grasper alleles are also carried by females. But if we look at the first column, which specifies the reproductive output of the mutant females, it does not depend on the mutant trait at all. Because mutant females tend to mate with resident males when the mutant is rare, the mutant females experience no cost because they carry the mutant allele. Although not part of our invasion analysis, we can also reason that mutant females will begin to suffer the costs of longer male graspers when the mutant allele becomes common. But the very same reduction in fitness will be experienced by the resident females as well, and therefore a mutant allele increasing grasper length in males will not be selected against in the females, even though it is harmful to them.

Interestingly, this coevolutionary escalation can increase the extinction risk of a population. For example, equilibrium (S12.1.14) becomes unstable if a mutant allele spreads that increases grasper length so much that $R_0\phi(x - y_m)$ falls below 2. In this case, if females fail to respond fast enough by evolving longer spines, the mutant might continue to increase in frequency until the population is driven to extinction. It might well be, however, that other factors not included in the model come into play before this happens. For example, as the population declines in size, fewer males might attempt to mate with any given female, which would cause the fitness of females to depend less on the grasper length of males (causing function ϕ to flatten out). To make accurate predictions about extinction risk, we would therefore need to account explicitly for the density of males and females in the function, ϕ.

Alternatively, natural selection in males might also curb the coevolutionary escalation by directly selecting against males with very long graspers (e.g., such males might have difficulty molting; see Problem S12.7). In this case, rather than exhibiting ever-increasing evolution of the

two traits, we might expect there to be an evolutionarily stable trait value for both males and females.

Recipe S12.2: Sequential Invasion Analyses for Multiple Traits

Sequential invasions analyses can be used when there is more than one trait, and each is coded for by its own locus. This often occurs when individuals in different classes within the same population display different traits, and it also occurs when modeling the coevolution of interacting species, each with their own trait (Problem S12.9). We focus on two traits and two loci, with extensions to more traits being analogous.

Suppose that the traits of interest are denoted by x and y, with each being coded for by its own locus, and the expression of each trait being restricted to a particular class of individuals. Label the class that expresses x, as 1 and the class that expresses y, as 2. We use $\lambda_1(x_m,x,y)$ and $\lambda_2(y_m,y,x)$ to denote the reproductive factor of a mutant allele affecting each of these traits, when the other trait is held fixed. As a check, we should have $\lambda_1(x,x,y) = 1$ and $\lambda_2(y,y,x) = 1$ in discrete time or $r_1(x,x,y) = 0$ and $r_2(y,y,x) = 0$ in continuous time.

Step 4: The trait values x and y are predicted to evolve in a direction given by the signs of their fitness gradients;

$$\left. \frac{\partial \lambda_1}{\partial x_m} \right|_{x_m=x}, \qquad \left. \frac{\partial \lambda_2}{\partial y_m} \right|_{y_m=y}. \tag{S12.1.19a}$$

Directional selection ceases for trait values, $x = \hat{x}$, $y = \hat{y}$, at which both fitness gradients in (S12.1.19a) equal zero. The trait combination, $\hat{x}$ and $\hat{y}$, is convergence stable in populations that exhibit a nearby trait combination if the matrix,

$$\begin{pmatrix} \frac{\partial}{\partial x}\left(\left. \frac{\partial \lambda_1}{\partial x_m} \right|_{x_m=x} \right) & \frac{\partial}{\partial y}\left(\left. \frac{\partial \lambda_1}{\partial x_m} \right|_{x_m=x} \right) \\ \frac{\partial}{\partial x}\left(\left. \frac{\partial \lambda_2}{\partial y_m} \right|_{y_m=y} \right) & \frac{\partial}{\partial y}\left(\left. \frac{\partial \lambda_2}{\partial y_m} \right|_{y_m=y} \right) \end{pmatrix}_{\substack{x=x^* \\ y=y^*}}, \tag{S12.1.19b}$$

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has eigenvalues with negative real parts.

Step 5 – An ESS pair x^*, y^* must satisfy the following first and second derivative conditions:

$$\left.\frac{\partial \lambda_1}{\partial x_m}\right|_{\substack{x_m=x^* \\ x=x^* \\ y=y^*}} = 0, \qquad \left.\frac{\partial \lambda_2}{\partial y_m}\right|_{\substack{y_m=y^* \\ y=y^* \\ x=x^*}} = 0 \qquad\qquad\text{(S12.1.19c)}$$

and

$$\left.\frac{\partial^2 \lambda_1}{\partial x_m{}^2}\right|_{\substack{x_m=x^* \\ x=x^* \\ y=y^*}} \le 0, \qquad \left.\frac{\partial^2 \lambda_2}{\partial y_m{}^2}\right|_{\substack{y_m=y^* \\ y=y^* \\ x=x^*}} \le 0. \qquad\qquad\text{(S12.1.19d)}$$

Conditions (S12.1.19c) and (S12.1.19d) are local versions of the more general, global conditions for a joint ESS; namely, $\lambda_1(x_m,x^*,y^*) \le \lambda_1(x^*,x^*,y^*)$ and $\lambda_2(y_m,y^*,x^*) \le \lambda_2(y^*,y^*,x^*)$.

It is worth emphasizing that Recipe S12.2 defines an ESS as a point that is stable to invasion by any *single* mutant allele. For two loci within the same species, however, it remains possible that a chromosome carrying mutations at both loci might still be able to invade even though conditions (S12.1.19c) and (S12.1.19d) hold, although such double mutant chromosomes would be extremely rare. To ensure that double mutant chromosomes cannot invade, we would have to perform a simultaneous invasion analysis, obtaining the eigenvalue from a local stability matrix with mutations present at both loci.

It is also worth emphasizing that the definition of convergence stability, condition (S12.1.19b) might also be insufficient if the fate of mutations at one locus are qualitatively different when there is a polymorphism at the second locus, rather than a fixed allele (as we have assumed). For example, selection might favor certain combinations of alleles, generating linkage disequilibrium that could well alter the path of evolution. As an example, suppose that two alleles are segregating at each of the grasper and spine loci. Males carrying short grasper alleles will be unable to mate with females with long spines and so will mate predominately with females with short spines. Consequently, a positive correlation will develop such that the short grasper allele will be found more often in the same genome with the short spine allele. As a result, selection favoring longer male graspers will not only increase the frequency of the long

grasper allele, but also the long spine allele with which it is associated. This phenomenon, termed indirect selection, has played an important role in many evolutionary models (see, e.g., Kirkpatrick 1982).

The above caveats illustrate that we must take extra care when trying to understand the evolutionary dynamics of two traits. Recipe S12.2 describes the expected outcome over evolutionary time only when mutant alleles segregate at one locus at a time.

Supplementary Material 12.2: Multivariate evolutionary invasion analyses

The evolutionary invasion analyses considered in Chapter 12 focus on the evolution of single traits. Many traits of interest cannot be adequately described by a single variable. When there is more than one trait, it is often natural to model the traits using more than one locus (see section S12.1). Here, however, we suppose that a single locus codes for all traits of interest. In this case, Recipe 12.1 continues to apply, and Recipe 12.2 can be readily extended:

Recipe S12.3 – Determining the direction of evolution and ESS for multiple traits

Consider mutant alleles that differ from the resident allele by only a small amount. Suppose that there are two traits of interest, denoted by x and y, and that $\lambda(x_m, y_m, x, y)$ is the discrete-time reproductive factor of a rare mutant allele coding for trait values x_m and y_m in a population consisting of a resident allele coding for trait values x and y. Continuous-time models are handled analogously with the discrete-time reproductive factor, $\lambda(x_m, y_m, x, y)$, replaced by the continuous-time mutant growth rate, $r(x_m, y_m, x, y)$. As a check, we should have $\lambda(x, y, x, y) = 1$ in discrete time or $r(x, y, x, y) = 0$ in continuous time when the mutant allele is associated with the same trait values as the resident.

Step 4: Predicting the direction of evolution is more complicated when there are two (or more) traits because evolution can proceed in any direction in the plane describing the values of the two variables. Furthermore, the direction of evolution is sensitive to any genetic correlations between the traits. Such genetic correlations arise, for example, if mutations that increase trait x tend to have specific effects on trait y. The best solution is to construct a model that explicitly tracks the evolutionary dynamics given the genetic correlations present. As a preliminary step, however,

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researchers often ignore such correlations. In this case, we can treat each trait separately, and the direction of evolution for traits x and y are

$$\left.\frac{\partial \lambda}{\partial x_m}\right|_{\substack{x_m=x \\ y_m=y}} \quad \text{and} \quad \left.\frac{\partial \lambda}{\partial y_m}\right|_{\substack{x_m=x \\ y_m=y}}. \tag{S12.2.1a}$$

Directional selection ceases for trait values, $x = \hat{x}$, $y = \hat{y}$, at which both fitness gradients in (S12.2.1a) equal zero. Furthermore, the multivariate analogue of the convergence stability condition of Recipe 12.2 is that the matrix

$$\left(\begin{matrix} \left.\dfrac{\partial}{\partial x}\left(\left.\dfrac{\partial \lambda}{\partial x_m}\right|_{x_m=x, y_m=y}\right) & \dfrac{\partial}{\partial y}\left(\left.\dfrac{\partial \lambda}{\partial x_m}\right|_{x_m=x, y_m=y}\right) \\ \dfrac{\partial}{\partial x}\left(\left.\dfrac{\partial \lambda}{\partial y_m}\right|_{x_m=x, y_m=y}\right) & \dfrac{\partial}{\partial y}\left(\left.\dfrac{\partial \lambda}{\partial y_m}\right|_{x_m=x, y_m=y}\right)\right.\end{matrix} \right)_{x=x^*, y=y^*}, \tag{S12.2.1b}$$

has eigenvalues with negative real parts.

Step 5: An ESS pair x^*, y^* must satisfy the following first and second derivative conditions:

$$\left.\frac{\partial \lambda}{\partial x_m}\right|_{\substack{x_m=x^*, y_m=y^* \\ x=x^*, y=y^*}} = 0, \quad \left.\frac{\partial \lambda}{\partial y_m}\right|_{\substack{x_m=x^*, y_m=y^* \\ x=x^*, y=y^*}} = 0 \tag{S12.2.1c}$$

and all of the eigenvalues of the following matrix must be less than or equal to zero:

$$\mathbf{H} = \left(\begin{matrix} \dfrac{\partial^2 \lambda}{\partial x_m^2} & \dfrac{\partial^2 \lambda}{\partial y_m \partial x_m} \\ \dfrac{\partial^2 \lambda}{\partial x_m \partial y_m} & \dfrac{\partial^2 \lambda}{\partial y_m^2} \end{matrix} \right)_{\substack{x_m=x^*, y_m=y^* \\ x=x^*, y=y^*}}. \tag{S12.2.1d}$$

$\mathbf{H}$ is known as a Hessian matrix, and because it is symmetric, its eigenvalues are guaranteed to be real. Again, conditions (S12.2.1c) and (S12.2.1d) ensure that a resident population with trait value x^*, y^* cannot be invaded by mutant strategies that are similar to x^*, y^*, but they do not guarantee that large-effect mutations cannot invade.

Let's apply Recipe S12.3 to an example. We will focus here on applying condition (S12.2.1c) only, and leave the application of conditions (S12.2.1b) and (S12.2.1d) as a lab exercise.

Example: Evolution of sperm and accessory protein production in males

In many species, females mate with multiple males during a single reproductive episode. We might expect natural selection to favor adaptations in males that enhance fertilization success when sperm from multiple fathers compete for the fertilization of the same set of eggs. One way to increase fertilization success is to increase the number of sperm transferred to a female during mating. If males have a fixed resource budget for the production of sperm, however, doing so will come at the cost of reducing the number of matings in which a male can take part. There is a large body of theory that explicitly addresses how evolution proceeds under this tradeoff (reviewed by Wedell *et al.* 2002).

In recent years, it has also been documented that males of some species transfer many other so-called "accessory proteins" during mating, in addition to sperm. Many of these proteins serve to increase female ovulation and thereby increase the number of eggs to which that male's sperm has access (Chapman *et al.* 2003a, 2003b). If males must also produce these proteins from their fixed reproductive budget, then there is an additional tradeoff between sperm and protein production. In particular, if multiple males mate with a female and compete for the fertilization of her eggs, then any given male would gain the highest reproductive success if it produced only sperm, while the other males produced the accessory proteins. This is a situation in which, for Step 1 of Recipe 12.1, there are now two decision variables; (i) amount of sperm produced per mating, s, and (ii) amount of protein produced per mating, a.

We now move to Step 2 of Recipe 12.1. Suppose that the population of interest has non-overlapping generations. We use R to denote the total resources that a male devotes to reproduction over its lifetime. Within a single mating, we let s and a denote the amount of sperm and accessory protein transferred to the female, where both are measured in terms of resource units. We will suppose that the species of interest has an XY chromosomal sex determination system (i.e., males carry an X and Y chromosome whereas females carry two copies of the X chromosome) and that both traits are coded for by a single gene found on the Y chromosome. This allows us to track only the number of males within the population. If we also assume that a

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male consumes an additional c units of resource when finding and courting a potential mate, then a male with an allele coding for s and a can have a total of $R/(c + s + a)$ mates in its lifetime.

The next step is to specify the total number of male offspring that a male can expect to sire at each mating. For simplicity we assume that a female has a single mate with probability $1 - u$ and two mates with probability u. When she is singly mated, we assume that a male fertilizes all of the female's eggs. When she is doubly mated, he fertilizes only a fraction $s_1/(s_1 + s_2)$, where the male of interest transferred s_1 sperm and the competing male transferred s_2 sperm. This fraction assumes that every sperm has an equal chance of fertilizing an egg. Letting $f(x)$ denote the number of eggs produced by a female who receives a total of x accessory proteins, the number of male offspring sired per mating by a father allocating s_1 and a_1 resources to sperm and proteins is $\frac{1}{2}\left((1 - u)f(a_1) + u\frac{s_1}{s_1 + s_2}f(a_1 + a_2)\right)$, when the competing male allocates s_2 and a_2. The 1/2 reflects an assumption of a 50:50 sex ratio.

With these assumptions we can now proceed through stages (a) and (b) of Step 2 (Recipe 12.1). The number of males in the population, M, is governed by the recursion equation

$$M(t + 1) = M(t)\frac{R}{c + s + a}\frac{1}{2}\left((1 - u)f(a) + u\frac{s}{s + s}f(a + a)\right)e^{-2\alpha M(t)} \qquad \text{(S12.2.2)}$$

where the factor $e^{-2\alpha M(t)}$ accounts for density dependence (the total population size is $2M$ if the sex ratio is 50:50). The nonzero equilibrium of (S12.2.2) is

$\hat{M} = \frac{1}{2\alpha}\ln\left(\frac{R}{c + s + a}\frac{1}{2}\left((1 - u)f(a) + u\frac{1}{2}f(2a)\right)\right)$, and we will assume that this equilibrium is stable.

By working through stages (c) and (d) of Step 2 (Recipe 12.1), you can check that the reproductive factor of a rare mutant allele coding for different value of s and a is given by the eigenvalue

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$$\lambda(s_m, a_m, s, a) = \frac{c+s+a}{c+s_m+a_m} \cdot \frac{(1-u)f(a_m) + u\dfrac{s_m}{s_m+s}f(a_m+a)}{(1-u)f(a) + u\dfrac{1}{2}f(2a)}.$$ (S12.2.3)

The conditions of Recipe S12.3 above can now be used to characterize the ESS sperm and protein production strategies assuming that mutant alleles have a small effect. To simplify matters, let's use the specific function $f(x) = b\,x^k$ for the number of eggs produced by a female that receives x accessory proteins (Figure S12.2.1). In this case, conditions (S12.2.1c) of Recipe S12.3 evaluate to give a single potential ESS:

$$s^* = \frac{2^k c\, u}{(1-k)\left(4(1-u) + 2^k u\right)}, \quad a^* = \frac{c\,k}{1-k}.$$ (S12.2.4a)

This potential ESS is biologically meaningful only when $k < 1$, which implies that the number of eggs produced by a female exhibits diminishing returns as a function of the amount of accessory proteins received. (If $k > 1$, then is no internal ESS, and we must consider boundary values of the parameters, representing, for example, the allocation of all resources to a single mate, $(c + s + a) = R$.)

Figure S12.2.1: Accessory proteins and egg production. The function relating number of eggs produced to the amount of accessory protein received by a female, $f(x) = b\, x^k$, for different values of k and b = 100.
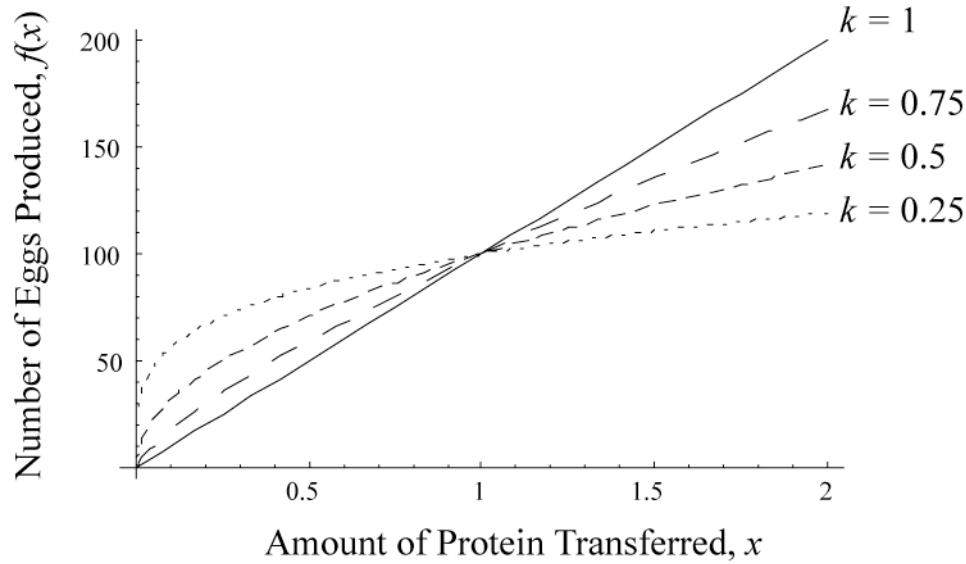

In an accompanying lab exercise, we guide you through the process of calculating and evaluating the convergence stability and second derivative conditions (S12.2.1b) and (S12.2.1d) of Recipe S12.3. Interestingly, the potential ESS is convergence stable whenever it exists ($k <$ 1), but it satisfies the second derivative condition (S12.2.1d) only when u and k are small enough. In particular, it requires that

$$u < \frac{1}{1 - \dfrac{(2 - 3k)}{2^{4-k}(1-k)}}. \tag{S12.2.4b}$$

Because the proportion of double matings, u, must be less than one, (S12.2.4b) always holds when female fecundity rises with a k less than 2/3. In Figure S12.2.2, we plot the reproductive factor of a mutant that arises within a resident population at the potential ESS. In case (a), k is sufficiently low that there is an uninvadible ESS. In case (b), k is so high that (S12.2.4b) is not satisfied, and some mutations have a reproductive factor greater than one.

Figure S12.2.2: Fate of mutant sperm and accessory protein strategies. The reproductive factor, $\lambda(s_m, a_m, s^*, a^*)$, of a mutant allocating s_m to sperm and a_m to accessory proteins that arises within a resident population at the potential ESS given by (S12.2.4a). Mutants that have the same strategy as the resident are neutral and have a reproductive factor of one (black square). (a) For low k = 0.3, the resident population cannot be invaded by any mutant. (b) For high k = 0.9, the resident population is at an evolutionary branching point and can be invaded by certain types of mutants (those above the black square on the saddle). Remaining parameters: R = 1000, c = 0.1, $\alpha = 0.1$, u = 1, $f(x) = bx^k$, b =1.
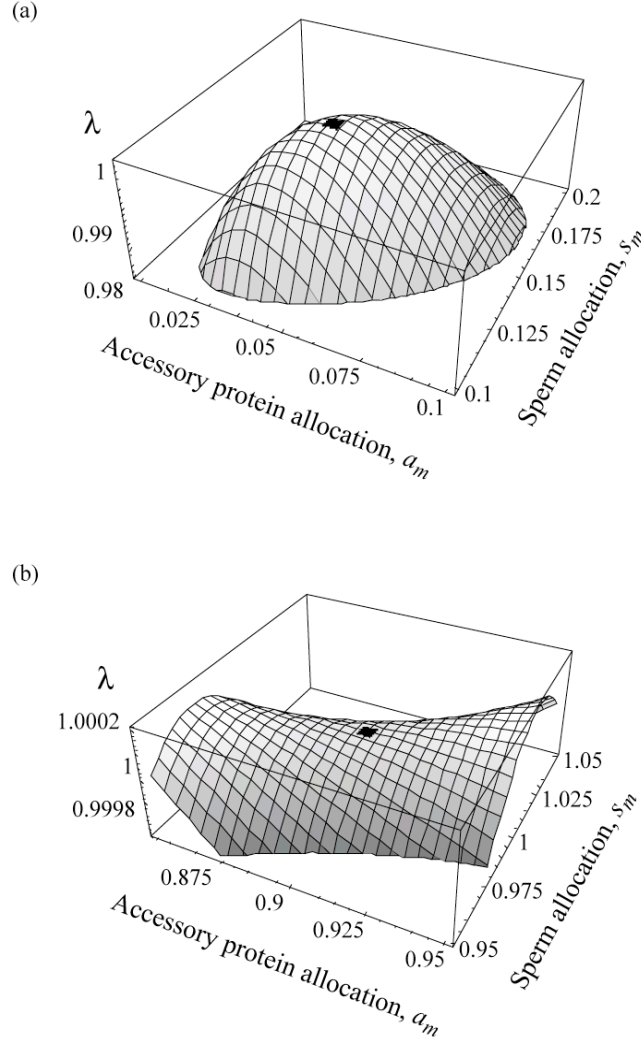
Restricting attention to parameter values for which condition (S12.2.4b) holds, the ESS values of sperm and protein allocation are plotted in Figure S12.2.3. As you can see, sperm allocation increases and protein allocation decreases as the probability of multiple mates, u, goes

up. Additionally, as the cost of finding a mate, c, increases, investment in both sperm and protein per mating increases.

Figure S12.2.3: ESS sperm and accessory protein allocation. The ESS value of (a) sperm allocation, $s*$, and (b) accessory protein allocation, $a*$, given by equations (S12.2.4a) is plotted as a function of the cost of mating, *c*, with *k* = 1/3. As the cost of finding a mate increases, males invest more in each mating. When females mate with multiple males (*u* high), males allocate more to sperm.
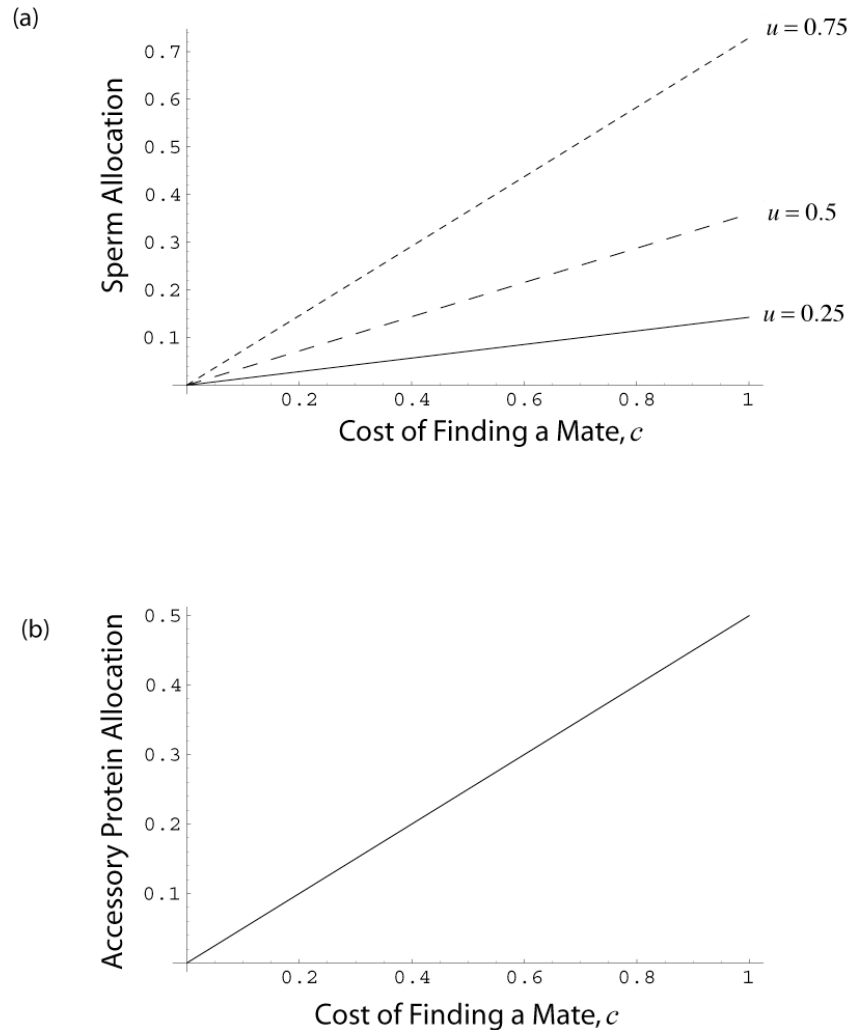
When condition (S12.2.4b) is not satisfied, a population evolves toward (S12.2.4a) because it is convergence stable. Once there, however, the population has reached a point where some mutations can spread (Figure S12.2.2b). Again, we know that these mutations will not entirely displace the resident allele, because convergence stability guarantees that the allele coding for strategy (S12.2.4a) will spread if ever it becomes rare (at least for mutations of small effect). Figure S12.2.4 presents simulation results following the spread of mutations and the appearance of a stable polymorphism. This is another example of evolutionary branching, where a population evolves toward a point at which a polymorphism arises (Geritz *et al.* 1998; Doebeli and Dieckmann 2000). When the proportion of double matings is high enough, males that invest little in accessory proteins can cheat by taking advantage of the accessory proteins provided by other males, saving their resources for additional matings.

Figure S12.2.4: Evolutionary branching in sperm and accessory protein allocation. At each time point along the horizontal axis, a mutant genotype with a randomly chosen sperm and protein allocation was introduced, and the dynamics simulated according to model (S12.2.2) until an equilibrium was reached. The equilibrium level of (a) sperm allocation and (b) accessory protein allocation was then plotted. Simulation results confirm that a polymorphic sperm and accessory protein allocation strategy can evolve, with some males producing less accessory protein and less sperm but gaining more mates over their lifetime. Parameter values are: R = 1000, c = 1, $\alpha = 0.1$, u = 1, $f(x) = bx^{k}$, b =1, k = 0.95, in which case (S12.2.4b) predicts evolutionary branching.

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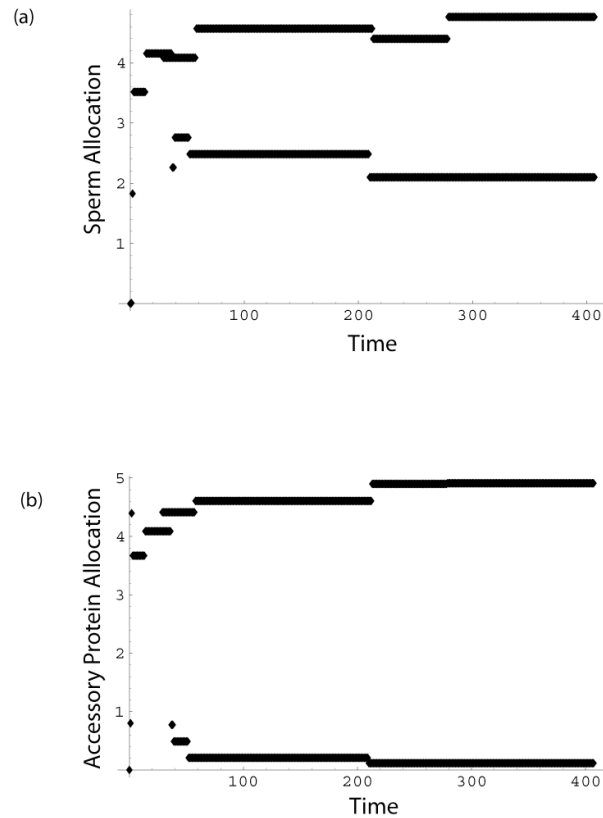
Supplementary Material 12.3: The effect of altering parameters with multiple traits

Now that we have techniques for determining ESS trait values when there are multiple traits, let's consider how altering a parameter value alter the ESS trait values, much as we did in Recipe 12.3.

We illustrate the procedure using the above model for sperm and accessory protein evolution to address the following questions. If we had chosen a different function, $f(x)$, relating protein investment to female fecundity would investment in sperm and protein still increase as the cost of mating, c, increases? Would we still see evolutionary branching? To answer these questions, we simplify the presentation by focusing only on the case where the probability of multiple matings is one (i.e., $u = 1$).

Our goal is to determine whether allocation to sperm and accessory proteins per mating always goes up as a function of the costs of finding a mate, c. To do so, we must first determine the conditions that must be met by an ESS. Using (S12.2.3) along with $u = 1$ in the first derivative conditions (S12.2.1c) of Recipe S12.3:

$$\frac{1}{2s*} - \frac{1}{c + s* + a*} = 0$$

$$\frac{\left(\dfrac{\mathrm{d}f(x)}{\mathrm{d}x}\right)_{x=2a*}}{f(2a*)} - \frac{1}{c + s* + a*} = 0. \tag{S12.3.1}$$

Because all of the parameters are positive as is the number of eggs, $f(2a*)$, produced by a female that receives $2a*$ accessory proteins, the second condition in (S12.3.1) requires that f must be an increasing function of the amount of accessory proteins she receives near any potential ESS (i.e., $\mathrm{d}f/\mathrm{d}x > 0$). Furthermore, using the accompanying lab exercise to evaluate the second derivative condition (S12.2.1d) of Recipe S12.3, we can show that any point satisfying (S12.3.1) is uninvadible if f exhibits sufficiently strong diminishing returns.

Specifically, we require that $\left.\dfrac{\mathrm{d}f^2}{\mathrm{d}^2x}\right|_{x=2a*} < -\dfrac{f(2a^*)}{8s^2}$. Nevertheless, a population converges to

(S12.3.1) whenever f exhibits any level of diminishing returns (i.e., provided that $\left.\dfrac{df^2}{d^2x}\right|_{x=2a*} < 0$;

see accompanying lab exercise). Thus, regardless of the exact form of the function describing how many eggs a female produces in response to a certain level of accessory proteins, evolutionary branching is possible as long as $f(x)$ curves down only slightly near the potential ESS.

We are now ready to determine how sperm allocation and accessory protein allocation at the ESS change as the cost of mating varies by applying a method similar to that of Recipe 12.3 in Chapter 12. Assume that an intermediate ESS occurs, and define $s*$ and $a*$ to be these ESS values. From condition (S12.2.1c) of Recipe S12.3, the ESS values of sperm and protein investment satisfy

$$\left.\frac{\partial \lambda(s_m,a_m,s,a,c)}{\partial s_m}\right|_{\substack{s_m=s*,a_m=a* \\ s=s^*,a=a*}} = 0$$

$$\left.\frac{\partial \lambda(s_m,a_m,s,a,c)}{\partial a_m}\right|_{\substack{s_m=s*,a_m=a* \\ s=s^*,a=a*}} = 0,$$
(S12.3.2)

where we have explicitly indicated that the mutant reproductive factor, λ, depends on the parameter c as well as on s_m,a_m and s,a.

Equations (S12.3.2) are functions of only $s*$, $a*$ and c because both s_m and s are evaluated at $s*$, and a_m and a are evaluated at $a*$. Therefore, to clarify the notation, let's define the functions $F(s,a,c) = \left.\partial\lambda(s_m,a_m,s,a,c)/\partial s_m\right|_{s_m=s,a_m=a}$ and $G(s,a,c) = \left.\partial\lambda(s_m,a_m,s,a,c)/\partial a_m\right|_{s_m=s,a_m=a}$. Equations (S12.3.2) can then be written as

$$F(s*,a*,c) = 0$$
$$G(s*,a*,c) = 0$$
(S12.3.3)

These equations implicitly define the ESS sperm and protein allocation as a function of the parameter c. In other words, any change in the parameter c must be met with corresponding changes in either $s*$, $a*$, or both such that the conditions (S12.3.3) continue to hold.

Our goal is to determine how the ESS sperm and accessory protein allocation change as the parameter c increases. Mathematically, these changes are given by the sign of the derivatives $\mathrm{d}s*/\mathrm{d}c$ and $\mathrm{d}a*/\mathrm{d}c$, which can be obtained by implicitly differentiating the two equations (S12.3.3) with respect to c. Although the calculations are somewhat messy, conceptually the procedure is identical to that used in section 12.4 of Chapter 12.

Implicitly differentiating (S12.3.3) with respect to c gives

$$
\begin{aligned}
&\frac{\partial F(s*,a*,c)}{\partial s*}\frac{\mathrm{d}s*}{\mathrm{d}c} + \frac{\partial F(s*,a*,c)}{\partial a*}\frac{\mathrm{d}a*}{\mathrm{d}c} + \frac{\partial F(s*,a*,c)}{\partial c} = 0 \\
&\frac{\partial G(s*,a*,c)}{\partial s*}\frac{\mathrm{d}s*}{\mathrm{d}c} + \frac{\partial G(s*,a*,c)}{\partial a*}\frac{\mathrm{d}a*}{\mathrm{d}c} + \frac{\partial G(s*,a*,c)}{\partial c} = 0
\end{aligned}
\tag{S12.3.4}
$$

which can be written in matrix notation as $\mathbf{M}\vec{v} = \vec{b}$,

$$
\mathbf{M} = \begin{pmatrix} \dfrac{\partial F(s*,a*,c)}{\partial s*} & \dfrac{\partial F(s*,a*,c)}{\partial a*} \\ \dfrac{\partial G(s*,a*,c)}{\partial s*} & \dfrac{\partial G(s*,a*,c)}{\partial a*} \end{pmatrix}, \quad \vec{v} = \begin{pmatrix} \dfrac{\mathrm{d}s*}{\mathrm{d}c} \\ \dfrac{\mathrm{d}a*}{\mathrm{d}c} \end{pmatrix}, \quad \vec{b} = -\begin{pmatrix} \dfrac{\partial F(s*,a*,c)}{\partial c} \\ \dfrac{\partial G(s*,a*,c)}{\partial c} \end{pmatrix}
\tag{S12.3.5}
$$

As discussed in Primer 2, this matrix equation can be solved for the vector, $\vec{v}$, giving $\vec{v} = \mathbf{M}^{-1}\vec{b}$. Using Definition P2.20 for the inverse of a 2×2 matrix, we get:

$$
\begin{aligned}
\frac{\mathrm{d}s*}{\mathrm{d}c} &= \left(-\frac{\partial G}{\partial a*}\frac{\partial F}{\partial c} + \frac{\partial F}{\partial a*}\frac{\partial G}{\partial c} \right)\Big/ \mathrm{Det}(\mathbf{M}) \\
\frac{\mathrm{d}a*}{\mathrm{d}c} &= \left(\frac{\partial G}{\partial s*}\frac{\partial F}{\partial c} - \frac{\partial F}{\partial s*}\frac{\partial G}{\partial c} \right)\Big/ \mathrm{Det}(\mathbf{M})
\end{aligned}
\tag{S12.3.6}
$$

Equation (S12.3.6) is an explicit expression for how the ESS allocation to sperm, $s*$, and accessory proteins, $a*$, vary as a function of the parameter, c.

Let's now rewrite (S12.3.6) using the definitions for $F(s,a,c)$ and $G(s,a,c)$. It clarifies the procedure if we first rewrite the partial derivatives, e.g., $\partial G(s*,a*,c*)/\partial a*$ as $\partial G(s,a,c)/\partial a\big|_{s=s*,\,a=a*}$; this makes it more obvious that we don't have to know the ESS values of $s*$ and $a*$ to evaluate the derivatives. Equation (S12.3.6) then becomes:

$$\frac{\mathrm{d}s*}{\mathrm{d}c} = \left\{ \left(\left(-\frac{\partial}{\partial a}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) + \frac{\partial}{\partial a}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \right) \middle/ \mathrm{Det}(\mathbf{M}) \right\} \Bigg|_{\substack{s=s*\\a=a*}}$$

$$\frac{\mathrm{d}a*}{\mathrm{d}c} = \left\{ \left(\left(\frac{\partial}{\partial s}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) - \frac{\partial}{\partial s}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \right) \middle/ \mathrm{Det}(\mathbf{M}) \right\} \Bigg|_{\substack{s=s*\\a=a*}}$$ (S12.3.7)

The matrix $\mathbf{M}$ is the same as matrix (S12.2.1b) in Recipe S12.3 used to evaluate convergence stability. Therefore, provided that we are dealing with an ESS that is convergence stable, the eigenvalues of this matrix will have negative real parts. Because the determinant of a 2 x 2 matrix equals the product of its eigenvalues (see section P2.8 in Primer 2), $\mathrm{Det}(\mathbf{M})$ will be positive near a convergence stable equilibrium. Therefore, when the cost of mating, c, is increased, the ESS value of both traits changes in a direction given by the sign of

$$\frac{\mathrm{d}s*}{\mathrm{d}p} \propto \left\{ -\frac{\partial}{\partial a}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) + \frac{\partial}{\partial a}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \right\} \Bigg|_{\substack{s=s*\\a=a*}}$$

$$\frac{\mathrm{d}a*}{\mathrm{d}p} \propto \left\{ \frac{\partial}{\partial s}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) - \frac{\partial}{\partial s}\left(\frac{\partial \lambda}{\partial s_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \frac{\partial}{\partial c}\left(\frac{\partial \lambda}{\partial a_m} \bigg|_{\substack{s_m=s\\a_m=a}} \right) \right\} \Bigg|_{\substack{s=s*\\a=a*}}$$ (S12.3.8)

Evaluating equations (S12.3.8) using the mutant reproductive factor, $\lambda(s_m,a_m,s,a,c)$, in the sperm/accessory protein model given by equation (S12.2.3), we get:

$$\frac{\mathrm{d}s*}{\mathrm{d}c} \propto 2\left(\left(\frac{\mathrm{d}f(x)}{\mathrm{d}x} \right)^2_{x=2a*} - f(2a*)\left(\frac{\mathrm{d}^2 f(x)}{\mathrm{d}x^2} \right)_{x=2a*} \right) \frac{1}{(c+s*+a*)^2 (f(2a))^2}$$

$$\frac{\mathrm{d}a*}{\mathrm{d}c} \propto \frac{1}{2(s*)^2 (c+s*+a*)^2}.$$ (S12.3.9)

Using the requirements that must be met by the function, f, near an ESS (i.e., $\mathrm{d}f/\mathrm{d}x > 0$ and $\mathrm{d}^2 f/\mathrm{d}x^2 < 0$), the first expression in (S12.3.9) is positive, as is the second expression. Consequently, the ESS investment in sperm and accessory proteins per mating is always predicted to increase as the cost of mating increases. Thus our previous qualitative prediction

holds regardless of the unknown function relating female egg production to the amount of accessory proteins she receives.

Recipe S12.4: The effect of parameters on multivariate ESS

Suppose that p is the parameter of interest. The following result is presented for a discrete-time model but analogous results hold for continuous-time models.

<u>Step 1</u>: Determine any restrictions that must be met in order for an intermediate ESS to exist by evaluating conditions (S12.2.1b), (S12.2.1c), and (S12.2.1d) of Recipe S12.3. Attempt to simplify conditions (S12.2.1b) and (S12.2.1d) by using the relationship that must hold among the functions and parameters given by condition (S12.2.1c).

<u>Step 2</u>: Assuming that the ESS is convergence stable, the ESS trait values x^* and y^* will change with a small increase in the parameter p in a direction given by the sign of

$$
\frac{\mathrm{d}x^*}{\mathrm{d}p} \propto \left\{ -\frac{\partial}{\partial y}\left(\frac{\partial\lambda}{\partial y_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\frac{\partial}{\partial p}\left(\frac{\partial\lambda}{\partial x_m}\Big|_{\substack{x_m=x\\y_m=y}}\right) + \frac{\partial}{\partial y}\left(\frac{\partial\lambda}{\partial x_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\frac{\partial}{\partial p}\left(\frac{\partial\lambda}{\partial y_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\right\}_{\substack{x=x^*\\y=y^*}}.
$$

$$
\frac{\mathrm{d}y^*}{\mathrm{d}p} \propto \left\{ \frac{\partial}{\partial x}\left(\frac{\partial\lambda}{\partial y_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\frac{\partial}{\partial p}\left(\frac{\partial\lambda}{\partial x_m}\Big|_{\substack{x_m=x\\y_m=y}}\right) - \frac{\partial}{\partial x}\left(\frac{\partial\lambda}{\partial x_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\frac{\partial}{\partial p}\left(\frac{\partial\lambda}{\partial y_m}\Big|_{\substack{x_m=x\\y_m=y}}\right)\right\}_{\substack{x=x^*\\y=y^*}}
$$

Supplementary Problems

Problem S12.1: Consider a female bird that produces a single clutch of offspring during her life and then gathers food at some rate to provision the nestlings before they fledge. We want to predict the offspring size and level of provisioning that we expect to evolve. Thus, we have two decision variables: offspring size, s, and level of care through food gathering, f, and both are allowed to take any positive value. Let's suppose that, as with the model of reproductive effort in section 12.2, the ESS trait values s^* and f^* maximize the female's lifetime reproductive output. Females survive to reproduce with probability q, at which point they have a fixed amount of

resources, R, at their disposal for the production of offspring. Given that a female produces offspring of size s, she will produce a total number of offspring, R/s.

Producing small offspring has the benefit that a large number of them can be produced, but suppose that offspring survival to fledging, V, is an increasing function of their size. Thus the female faces a trade-off between producing lots of offspring that survive poorly or producing fewer offspring that survive well. Further, suppose that offspring survival to fledging is an increasing function of the amount of food they receive from their mother, but that high levels of such care result in a cost in terms of brood survival because large periods of time spent gathering food expose the nest to a higher risk of predation. Thus the probability of a brood surviving predation, p, is a decreasing function of the level of care. With these definitions and assumptions, the female's lifetime reproductive output is

$$W(s,f) = q\frac{R}{s}V(s,f)p(f).$$

(a) Derive a pair of equations that any potential ESS, s^* and f^*, must satisfy using condition (S12.2.1c) in Recipe S12.3. **(b)** Assume that offspring survival in the absence of predation is given by $V(s,f) = s^2 f / (a + s^2 f)$ and that the probability that the offspring survive predation is $p(f) = 1/(1 + bf)$, where a and b are positive parameters. Plot these functions for various values of the parameters, and describe the underlying biological assumptions that are implicit in the shape of these functions. **(c)** Evaluate the potential ESS, s^* and f^*, identified in (a) using the functions in (b). **(d)** Demonstrate that the point, s^* and f^*, in part (c) represents an ESS that maximizes, rather than minimizes, W.

Problem S12.2: (a) Calculate the leading eigenvalue of the mutant sub-matrix (S12.1.5c) for the model of sex ratio evolution. **(b)** Use the eigenvalue found in part (a) to demonstrate that $s = 1/2$ is the only possible ESS (equation 12.1c in Recipe 12.2). **(c)** Demonstrate that the second derivative condition (12.1d) in Recipe 12.2 is zero for the sex ratio model. What does this mean? **(d)** Demonstrate that the convergence stability condition (12.1c) in Recipe 12.2 is satisfied for the sex ratio model.

Problem S12.3: In Box 12.4 of Chapter 12 we discussed pairwise invasibility plots. **(a)** Construct a pairwise invasibility plot for the sex ratio model. [Hint: You will first need to calculate the leading eigenvalue of the mutant sub-matrix (S12.1.5c).] **(b)** Use the plot from part (a) to demonstrate that any mutant allele that increases the proportion of males ($s_m > s$) can invade when the sex ratio is female-biased ($s < 1/2$), while any mutant allele that decreases the proportion of males ($s_m < s$) can invade when the sex ratio is male-biased ($s > 1/2$). **(c)** It can be shown that the second derivative condition (12.1d) in Recipe 12.1 evaluates to zero for the sex ratio model (Problem S12.3). How is this reflected in the pairwise invasibility plot?

Problem S12.4: (a) Calculate the leading eigenvalue of the mutant sub-matrix (S12.1.3) for the model of intralocus sexual conflict. **(b)** Using your answer to part (a), demonstrate that the general condition for a mutant allele to invade is given by

$$\frac{p_f(x_{het})}{p_f(x)} \frac{(p_m(x_m) + p_m(x))/2}{p_m(x)} > 1.$$

(c) Provide a biologically motivated explanation of this condition.

Problem S12.5: The sex ratio model in the text assumed that the locus affecting sex ratio evolution was located on an autosome and that females expressed this locus. Here we consider a species that has an XY sex determination system (females are XX and males are XY), and we suppose that the locus affecting the sex ratio is located on the X chromosome and is expressed in males only. Specifically, the allele found on the X chromosome in males determines the proportion of its sperm that carry the X chromosome (and thus give rise to daughters) versus the proportion that carry the Y chromosome (and thus give rise to sons). **(a)** Demonstrate that the final entries in Table S12.1.1 for this model now become

	# Mutant Daughters Produced	# Mutant Sons Produced
Mutant Mothers	Total $= R_0\, e^{-\alpha \hat{F}}\dfrac{(1-s)}{2}$	Total $= R_0\, e^{-\alpha \hat{F}}\dfrac{s}{2}$
Mutant Fathers	Total $= \dfrac{\hat{F}}{\hat{M}} R_0\, e^{-\alpha \hat{F}}(1-s_m)$	Total $= 0$

(b) Demonstrate that the mutant sub-matrix for this model is given by

$$\mathbf{M} = \frac{R_0\, e^{-\alpha \hat{F}}}{2}\begin{pmatrix} 1-s & 2\dfrac{\hat{F}}{\hat{M}}(1-s_m) \\ s & 0 \end{pmatrix}, \text{ where } \hat{F} \text{ is the same as equilibrium (S12.1.2). } \textbf{(c)} \text{ Prove that,}$$

for this model, the sex ratio is predicted to evolve to ever smaller values until only females are produced (also see Hamilton 1967). **(d)** Provide a biological explanation for the predictions of this model.

Problem S12.6: In the model of intra-locus sexually antagonistic selection (S12.1.7) consider two alleles: one that benefits females with $\left(\dfrac{1}{p_f}\dfrac{dp_f}{dx}\right) = -\left(\dfrac{1}{p_m}\dfrac{dp_m}{dx}\right) > 0$ and one that benefits

males with $-\left(\dfrac{1}{p_f}\dfrac{dp_f}{dx}\right) = \left(\dfrac{1}{p_m}\dfrac{dp_m}{dx}\right) > 0.$ Use (S12.1.12) to determine when the first allele can invade a population fixed for the second allele and vice versa. Use these results to determine whether a polymorphism is possible. [Caution: Remember that h is the dominance of the rare mutant allele in (S12.1.12).]

Problem S12.7: The model of interlocus sexual conflict in section S12.1 predicted a never-ending evolutionary escalation in female spine length and male grasper length. Such an evolutionary arms race must eventually come to a halt, and one reason for this might be that natural selection opposes the indefinite increase in these morphological traits. Here we revise the model to incorporate natural selection. Suppose that the population of males and females is censused just prior to mating, and suppose that males with grasper length y survive to adulthood with probability $p_m(y)$ and that females with spine length x survive with probability $p_f(x)$. **(a)** Demonstrate that the mutant sub-matrices (S12.1.15b) and (S12.1.17b) are then given by

$$\begin{pmatrix} \dfrac{1}{4}R_0\, e^{-\alpha \hat{F}}\phi(x-y)p_f(x) & \dfrac{1}{4}\dfrac{\hat{F}}{\hat{M}}g(y_m-y)R_0\, e^{-\alpha \hat{F}}\phi(x-y)p_f(x) \\ \dfrac{1}{4}R_0\, e^{-\alpha \hat{F}}\phi(x-y)p_m(y_m) & \dfrac{1}{4}\dfrac{\hat{F}}{\hat{M}}g(y_m-y)R_0\, e^{-\alpha \hat{F}}\phi(x-y)p_m(y_m) \end{pmatrix}$$

and

$$\left(\begin{array}{cc} \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x_m - y) p_f(x_m) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \phi(x - y) p_f(x_m) \\[3mm] \dfrac{1}{4} R_0 e^{-\alpha \hat{F}} \phi(x_m - y) p_m(y) & \dfrac{1}{4} \dfrac{\hat{F}}{\hat{M}} R_0 e^{-\alpha \hat{F}} \phi(x - y) p_m(y) \end{array} \right).$$

(b) Calculate the leading eigenvalue for each of these mutant sub-matrices. **(c)** Suppose that female survival is unaffected by spine length (i.e., $p_f(x) \equiv 1$), that male survival is given by the linearly decreasing function $p_m(y) = 1 - c_m y$ (with $0 < c_m < 1$), that $g(y_m - y) = e^{y_m - y}$, and that $\phi(d) = e^{-d^2}$. Calculate the ESS values of grasper and spine length using conditions (S12.1.19c) and (S12.1.19d) in Recipe S12.1. **(d)** What is the predicted probability of male survival at this ESS?

Problem S12.8: The sex ratio model (S12.1.1) assumed that the production of a male requires the same amount of resources as the production of a female. Suppose that producing a son requires c_m resource units, whereas producing a daughter requires c_f units. In this case, if a mother devotes $R_0 \exp(-\alpha \hat{F}) s$ resource units to sons and $R_0 \exp(-\alpha \hat{F})(1 - s)$ resource units to daughters, then she will produce a total of $R_0 \exp(-\alpha \hat{F}) s / c_m$ sons and $R_0 \exp(-\alpha \hat{F})(1 - s)/c_f$ daughters. **(a)** Derive the corresponding mutant sub-matrix (S12.1.5c) for this modified model. **(b)** Calculate the ESS allocation strategy, s^*. **(c)** What is the predicted ratio of males to females in the population if this ESS allocation strategy is being used?

Problem S12.9: Although all of the models of Chapter 12 and Sup. Mat. 12 have focused on the evolution of one of more traits within a single species, an invasion analysis for the coevolution of two species is a natural extension. Here, we consider the simplest case, where each species has a single locus coding for the trait of interest, and we perform a sequential analysis (as in Recipe S12.2). The analysis begins with each species fixed for a resident allele. We then introduce a rare mutant allele in one species, while holding the other fixed and derive an expression for the growth rate of this mutant allele. This procedure is then repeated for the second species, resulting in a growth rate expression for each species. Conditions analogous to (S12.1.19c) and (S12.1.19d) in Recipe S12.4 are then used to characterize the joint ESS of the two species. As an example, consider the coevolution of a host-parasite system, in which the host trait of interest is the rate at which it clears the infection, c, and the parasite trait of interest is the mortality rate that

it induces on its host (i.e., virulence, v). Here, we suppose that host survival increases with investment in parasite clearance according to the rational function $c/(c+v)$, but that host fertility declines as resources are diverted away from reproduction according to $\alpha/(\beta+c)$, where α and β are positive parameters (see Figure P1.5). Under these assumptions, the reproductive factor of a mutant host allele coding for a different clearance level is given by

$$\lambda_{host} = \frac{\left(c_m/(c_m+v)\right)\left(\alpha/(\beta+c_m)\right)}{\left(c/(c+v)\right)\left(\alpha/(\beta+c)\right)}.$$ We also suppose that parasite fitness equals the probability

that the host is killed $v/(c+v)$, divided by v^{γ} to account for the possibility that killing the host early reduces the chance of transmission, where $0 < \gamma < 1$. Accordingly, the reproductive factor of a mutant parasite allele coding for a different virulence level is given by

$$\lambda_{parasite} = \frac{v_m^{1-\gamma}/(v_m+c)}{v^{1-\gamma}/(v+c)}.$$ **(a)** Apply the first derivative conditions (S12.1.19c) in Recipe S12.2

to demonstrate that a potential joint ESS is $v* = \beta\,(1-\gamma)^2/\gamma^2$ and $c* = \beta\,(1-\gamma)/\gamma$. **(b)** Demonstrate that this potential ESS satisfies the second derivative condition (S12.1.19d) in Recipe S12.2. **(c)** How do the ESS virulence and clearance levels depend on the extent to which parasite virulence reduces transmissibility, γ? **(d)** At this joint ESS, what is the probability of host death, $v*/(c*+v*)$?

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