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**Article** *in* The American Naturalist · November 2018

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# Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes

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*Submitted February 12, 2018; Accepted August 21, 2018; Electronically published November 15, 2018*

*Online enhancements:* appendixes.

ABSTRACT: The evolutionary trajectories of species with separate sexes depend on the effects of genetic variation on female and male traits as well as the direction and alignment of selection between the sexes. Classical theory has shown that evolution is equally responsive to selection on females and males, with natural selection increasing the product of the average relative fitness of each sex over time. This simple rule underlies several important predictions regarding the maintenance of genetic variation, the genetic basis of adaptation, and the dynamics of "sexually antagonistic" alleles. Nevertheless, theories of sex-specific selection overwhelmingly focus on evolution in constant environments, and it remains unclear whether they apply under changing conditions. We derived four simple models of sex-specific selection in variable environments and explored how conditions of population subdivision, the timing of dispersal, sex differences in dispersal, and the nature of environmental change mediate the evolutionary dynamics of sex-specific adaptation. We find that these dynamics are acutely sensitive to ecological, demographic, and life-history attributes that vary widely among species, with classical predictions breaking down in contexts of environmental heterogeneity. The evolutionary rules governing sex-specific adaptation may therefore differ between species, suggesting new avenues for research on the evolution of sexual dimorphism.

*Keywords:* sexual antagonism, hard selection, soft selection, balancing selection, polymorphism, sexual dimorphism.

> Natural selection will maximize the product of the average fitness of the two sexes. (Campos Rosado and Robertson 1966, p. 328)
>
> An autosomal [allele] that produces a sexually-antagonistic phenotype will only increase in frequency when rare if the advantage to one sex is larger than the disadvantage to the other sex. (Rice 1984, p. 736)

## Introduction

Females and males make roughly equal genetic contributions to offspring, and this symmetry in genetic transmission is expected to equalize the relative contributions of selection within each sex to the evolutionary dynamics of a population. Although natural selection does not necessarily increase adaptation of both sexes, it does simultaneously favor evolutionary changes that balance gains in adaptation for one sex against reductions in adaptation for the other (Lande 1980; Rice 1984). For example, an allele that improves adaptation for one sex may spread within a population despite harming the other sex as long as the fitness gain to the benefitting sex outweighs the fitness cost to the harmed sex (Mandel 1971; Kidwell et al. 1977; Rice 1984). Such "sexually antagonistic" selection has been documented in a range of animal and plant populations (e.g., Bonduriansky and Chenoweth 2009; Cox and Calsbeek 2009; Delph et al. 2011; Morrissey 2016) and contributes to the maintenance of genetic variation (Chippindale et al. 2001; Mank 2017) and the manifestation of evolutionary constraints to adaptation (Lewis et al. 2011; Gosden et al. 2012; Stearns et al. 2012).

In cases where natural selection cannot simultaneously improve adaptation in both sexes, what exactly will selection increase? Classical population genetics theory long ago identified a simple maximization rule for evolution in populations with separate sexes. In a population where selection is frequency and density independent and breeding adults mate randomly with respect to selected loci, natural selection increases the product of female and male mean relative fitness (Wright 1942; Campos Rosado and Robertson 1966; Turner 1968). To illustrate the simplest form of the theory, consider the case of selection at a haploid-expressed gene with two alleles (e.g., alleles *A* and *B*; Gregorius 1982; Immler

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Am. Nat. 2019. Vol. 193, pp. 000–000. 
0003-0147/2019/19301-58259$15.00.
DOI: 10.1086/700720

et al. 2012; see below). The expected change in frequency of the *A* allele per generation is

$$\Delta p = \frac{p(1-p)}{2}\frac{d\ln(\overline{W}_f\overline{W}_m)}{dp}, \quad (1)$$

where $p$ is the frequency of the *A* allele, $\overline{W}_f$ and $\overline{W}_m$ are the mean relative fitnesses of females and males (respectively), and $d\ln(\overline{W}_f\overline{W}_m)/dp$ is a selection gradient on the *A* allele that describes how the product of the mean fitnesses, $\overline{W}_f\overline{W}_m$, varies with changes in the allele frequencies. The gradient is positive when selection favors the *A* allele ($p$ increases), negative when selection favors the *B* allele ($p$ decreases), and zero at a polymorphic equilibrium ($p$ remains constant). Equation (1) implies that natural selection will increase and ultimately maximize $\overline{W}_f\overline{W}_m$. Moreover, the contribution of selection within each sex to evolutionary change is symmetrical, as evident from the decomposition of the gradient into equal parts female and male selection: $d\ln(\overline{W}_f\overline{W}_m)/dp = d\ln(\overline{W}_f)/dp + d\ln(\overline{W}_m)/dp$.

Remarkably, the general form of equation (1) and the simple maximization rule that it implies consistently emerge from a broad array of theoretical contexts, from population genetic models of sex-linked and autosomal loci (Wright 1942; Campos Rosado and Robertson 1966; Turner 1968; Hartl 1972) to polygenic models for the evolution of quantitative traits (Lande 1980). Moreover, equation (1) and its extensions provide the theoretical foundation for research on sex-specific adaptation. For example, analyses of equation (1) and its diploid and sex-linked equivalents yield influential predictions about the dynamics of alleles with sex-specific fitness effects (see below), including (i) conditions for evolutionary spread of sexually antagonistic alleles, (ii) conditions for maintaining genetic polymorphisms, and (iii) equilibrium genetic variance and allele frequencies under sex-specific selection (e.g., Mandel 1971; Kidwell et al. 1977; Patten and Haig 2009; Jordan and Charlesworth 2012; Connallon and Jordan 2016). The theory also provides a basis for interpreting empirical patterns of sex-specific selection (Cox and Calsbeek 2009; De Lisle et al. 2018), genetic variation (Gibson et al. 2002; Fry 2010; Poissant et al. 2010; Griffin et al. 2013), evolutionary constraints (Lewis et al. 2011; Gosden et al. 2012; Stearns et al. 2012), and molecular genetic diversity (Cheng and Kirkpatrick 2016; Mank 2017).

On the other hand, current theories of sex-specific selection overwhelmingly focus on evolution in constant environments—a condition that is certain to be violated in many species, including taxa where sex differences in selection are well documented (e.g., Delph et al. 2011; De Lisle et al. 2018). Here we show that the evolutionary dynamics predicted by classical theories of sex-specific selection—as captured in equation (1) and reviewed below—often break down in heterogeneous environments. We illustrate this point by presenting four models of sex-specific selection in spatially and temporally variable environments. These models build on the classical population genetic framework of selection in changing environments (e.g., Dempster 1955; Christiansen 1975; Felsenstein 1976).

Our models show that species-specific features of dispersal and environmental heterogeneity can have profound consequences for the evolutionary dynamics of alleles that differentially affect female and male fitness. Opportunities for adaptation in females and males are mediated by life-history and demographic characteristics that widely vary among species. In some contexts, evolutionary trajectories become biased toward the interests of one sex at the expense of the other. These new results strengthen the theoretical foundations of emerging empirical research on the interaction between environmental variation and sex-specific selection (see Delph et al. 2011; Long et al. 2012; Berger et al. 2014; Miller and Svensson 2014; De Lisle and Rowe 2017; Connallon et al. 2018; De Lisle et al. 2018; Olito et al. 2018) and provide new impetus for diversifying the range of species in which sexual conflict is studied.

## Sex-Specific Adaptation in Constant Environments

The theoretical dynamics of sex-specific adaptation in constant environments represent a natural point of contrast for our extended models. According to this theory, which we briefly review, selection favors genetic variation that increases the product of the average relative fitness of females and males ($\overline{W}_f\overline{W}_m$). The example of haploid selection, as outlined in the introduction, is a special case that illustrates the point. As we outline below, the general prediction applies much more broadly and underlies predictions about the evolutionary dynamics of sex-specific adaptation and the maintenance of sexually antagonistic genetic variation.

### *Models of Sex-Specific Selection and Evolution*

Most population genetic models of sex-dependent selection (e.g., Wright 1942; Owen 1953; Haldane 1962; Kidwell et al. 1977; Gregorius 1982; Immler et al. 2012; Connallon and Jordan 2016) consider evolution at single, biallelic loci with frequency- and density-independent effects on the relative fitnesses of females and males. Following this tradition, consider a locus with two alleles, *A* and *B*, that affect fitness in either the haploid or the diploid state (table 1). As in prior theory, assume that generations are discrete, and the life cycle follows the following order of events: (i) birth, (ii) sex-dependent natural selection, (iii) production of offspring via random mating of females and males from the postselection pool of individuals, and (iv) death. Population size is sufficiently large that genetic drift can be ignored.

Evolutionary dynamics under haploid selection are exactly described by equation (1) above. With minor adjustments, results of the haploid model can be extended to cases

**Table 1:** Relative fitness per sex and genotype in a constant environment

| | Genotype | | |
|---|---|---|---|
| | *AA*, *A* | *AB* | *BB*, *B* |
| Haploid selection model: | | | |
| Female fitness | $w_A$ | ... | $w_B$ |
| Male fitness | $v_A$ | ... | $v_B$ |
| Diploid selection model: | | | |
| Female fitness | $w_{AA}$ | $w_{AB}$ | $w_{BB}$ |
| Male fitness | $v_{AA}$ | $v_{AB}$ | $v_{BB}$ |

of diploid or haplodiploid (e.g., x-linked) inheritance (see app. A; apps. A–E are available online). Several studies have derived approximations for the diploid selection case, assuming modest to weak selection and arbitrary dominance relations between alleles (e.g., Campos Rosado and Robertson 1966; Turner 1968; Connallon and Hall 2018). For these cases, the change in frequency of the *A* allele is well approximated by

$$\begin{aligned}\Delta p &\approx \frac{p(1-p)}{4}\left(\frac{d\ln(\overline{W}_{\mathrm{f}})}{dp}+\frac{d\ln(\overline{W}_{\mathrm{m}})}{dp}\right)\\ &=\frac{p(1-p)}{4}\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp},\end{aligned} \tag{2}$$

where $d\ln(\overline{W}_{\mathrm{f}})/dp$ represents the selection gradient for females and $d\ln(\overline{W}_{\mathrm{m}})/dp$ represents the selection gradient for males. Equation (2) reveals that, as with haploid selection, natural selection in diploids increases $\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}}$. Wright (1942) and Campos Rosado and Robertson (1966) have derived analogous results for x-linked loci (see app. A). Lande (1980) has shown that the maximization principle implied by equations (1) and (2) also emerges from scenarios of quantitative trait evolution (see Lande 1980, p. 295).

With no dominance interactions between alleles (i.e., $w_{AB} = (w_{AA} + w_{BB})/2$ and $v_{AB} = (v_{AA} + v_{BB})/2$; see table 1), the exact evolutionary dynamics under arbitrarily strong selection in diploids are described by

$$\Delta p = \left(\frac{p(1-p)}{4}-\frac{\delta^2}{16}\right)\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}, \tag{3}$$

where $\overline{W}_{\mathrm{f}} = w_{BB} + p(w_{AA} - w_{BB})$, $\overline{W}_{\mathrm{m}} = v_{BB} + p(v_{AA} - v_{BB})$, and $\delta^2 = (p_{\mathrm{m}} - p_{\mathrm{f}})^2$ represents the square of the allele frequency difference between breeding females and males (see Connallon and Hall 2018). Equation (3) shows once again that natural selection increases $\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}}$.

### *Evolutionary Dynamics of Sexually Antagonistic Alleles*

Equations (1)–(3) are quite general and can be used to retrieve specific results from previous theory, including influential predictions about the evolutionary potential for the spread of sexually antagonistic alleles and the maintenance of polymorphism (Mandel 1971; Kidwell et al. 1977; Immler et al. 2012). Previous studies typically delineate conditions for invasion and "protected" polymorphism (sensu Prout 1968) by way of a linear stability analysis of the boundary equilibria ($p = 0$ and $p = 1$) of generalized recursions under sex-specific selection (e.g., Kidwell et al. 1977; Patten and Haig 2009; Immler et al. 2012). Results equivalent to these earlier studies can also be obtained by direct analysis of the selection gradient, $d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})/dp$, for a haploid, diploid, or x-linked locus (see app. A).

Consider, for example, the evolution of sexually antagonistic alleles under haploid or diploid selection with no dominance (for additional results, see app. A). Assume that the *A* allele is beneficial to females and the *B* allele is beneficial to males, with $s_{\mathrm{f}}$ and $s_{\mathrm{m}}$ representing the female and male selection coefficients ($0 < s_{\mathrm{f}}, s_{\mathrm{m}} < 1$; see Kidwell et al. 1977; Fry 2010). The condition for invasion of the female-beneficial allele is $s_{\mathrm{f}} > s_{\mathrm{m}}/(1 + s_{\mathrm{m}})$ and for invasion of the male-beneficial allele is $s_{\mathrm{m}} > s_{\mathrm{f}}/(1 + s_{\mathrm{f}})$. Selection maintains both alleles when

$$\frac{s_{\mathrm{m}}}{1+s_{\mathrm{m}}} < s_{\mathrm{f}} < \frac{s_{\mathrm{m}}}{1-s_{\mathrm{m}}} \tag{4}$$

(Kidwell et al. 1977; Pamilo 1979; Immler et al. 2012; see app. A). These inequalities can be used to calculate proportions of parameter space (within the range $0 < s_{\mathrm{f}}, s_{\mathrm{m}} < 1$) that lead to fixation or polymorphism of sexually antagonistic alleles. As originally shown by Pamilo (1979), the parameter space divides symmetrically into three parts, with (i) $1 - \ln(2) \approx 0.31$ representing the fraction of parameter space where female-beneficial alleles are fixed, (ii) $1 - \ln(2) \approx 0.31$ representing the fraction of parameter space where male-beneficial alleles are fixed, and (iii) $\ln(4) - 1 \approx 0.38$ representing the fraction of parameter space where polymorphisms are maintained. Thus, the majority of parameter space results in fixation of one allele or the other, and opportunities for adaptation are symmetric between the sexes (see Kidwell et al. 1977; Pamilo 1979; Prout 2000; fig. 1).

## Models of Sex-Specific Adaptation in Heterogeneous Environments

Sex-specific selection that varies over space or time can lead to evolutionary predictions quantitatively and qualitatively different than those of the standard theory, reviewed above. Moreover, when selection varies over space, details of a species' life history and demography can influence evolutionary dynamics in ways that are not possible in models with constant selection. To illustrate this point, we present four models that vary in the life stage or sex that disperses and in the form of environmental heterogeneity that impacts selection (i.e., spatial vs. temporal heterogeneity; Levene 1953; Demp-

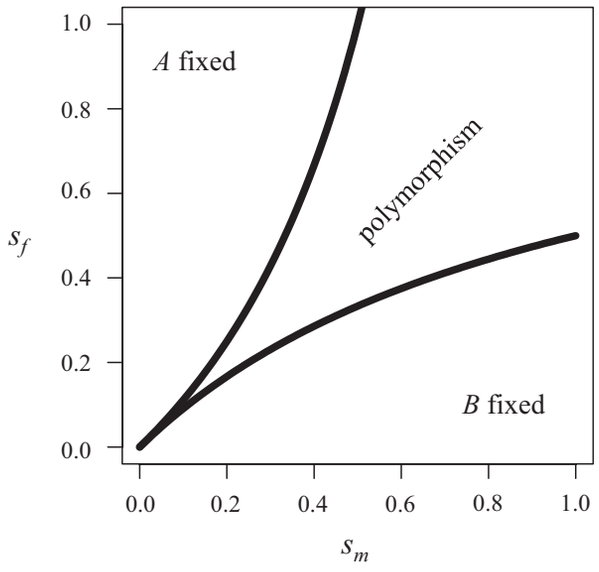


**Figure 1:** Evolutionary fates of sexually antagonistic alleles in a constant environment. Curves are based on the inequalities below equation (4), where allele *A* is the female-beneficial allele and *B* is the male-beneficial allele. These results apply to haploid models of sexual conflict (Gregorius 1982; Immler et al. 2012) and diploid models of sexual conflict with codominant (additive) fitness effects of alleles within each sex (e.g., Kidwell et al. 1977). Note two specific patterns in the figure. First, the figure documents the entire plausible parameter space of sexually antagonistic selection in constant environment ($0 < s_f, s_m < 1$), in which ~38% of parameter combinations give rise to a polymorphic equilibrium (see Pamilo 1979 and the main text). Second, the parameter space delineates sexually symmetric evolutionary outcomes: the proportion of parameter space leading to fixation of the female-beneficial allele (*A* fixed) is equal to the proportion leading to fixation of the male-beneficial allele (*B* fixed).

ster 1955; Felsenstein 1976; Huang et al. 2015). Apart from the assumption of environmental constancy, all other major assumptions of our new models match those of the theory outlined above. The new models span only a subset of conceivable contexts of sex-specific adaptation in changing environments (see "Discussion"), but they nevertheless cover a broad range of conditions of biological interest. They therefore provide a solid foundation for more complex models and a clear basis for future empirical tests of population genetic theory of heterogeneous environments (e.g., along the lines of Gallet et al. 2018).

### *Overview and Assumptions of the Models*

Following classical scenarios of population structure with high gene flow (e.g., Levene 1953; Dempster 1955; Christiansen 1975), we developed three models of spatial heterogeneity in which dispersing individuals move randomly among an arbitrary number of habitat patches. Our models assume that population density regulation occurs after dispersal, which conforms to the notion of "hard selection" (Christiansen 1975; DéBarre and Gandon 2011; elaborated in "Discussion"). Hard selection implies a direct demographic feedback between the genetic composition of each subpopulation and its relative contribution to the population dynamics of the species. In contrast, "soft selection" implies a lack of such feedbacks: the contribution of each subpopulation to population dynamics is independent of its genetic composition. Previous theory has shown that scenarios of hard and soft selection have different consequences for the maintenance of genetic variation, with soft selection more conducive to maintaining polymorphism than hard selection (Levene 1953; Dempster 1955; Li 1955; Christiansen 1975; DéBarre and Gandon 2011). Our assumption of hard selection will therefore render our predictions about polymorphism conservative.

In model 1 (the adult dispersal model), local selection occurs before individuals of both sexes disperse from their natal habitats, and mating and reproduction occurs afterward. This scenario applies to species with relatively immobile early-life stages, including many vertebrate and insect taxa (Burgess et al. 2016). In model 2 (the juvenile dispersal model), juveniles disperse from their natal habitats prior to local selection, mating, and reproduction. This scenario mimics dispersal in species where individuals are highly mobile during early-life stages (e.g., seed dispersal in plants, larval dispersal in many aquatic organisms; Burgess et al. 2016). In model 3 (the sex-limited dispersal model), adults from only one sex disperse from their natal habitats prior to mating and reproduction. Sex-biased migration is common in animals, ranging from strongly female biased in some species to male biased in others (Trochet et al. 2016). Finally, following classical theory of selection in temporally varying environments (Dempster 1955; Felsenstein 1976), we develop a model of sex-specific selection that changes over time but is otherwise uniform across space (model 4, the temporal heterogeneity model). In keeping with the assumptions of models 1–3, we assume in model 4 that population density is externally regulated and constant over time and note that different results may emerge in models that include demographic fluctuations (see Lande 2007, 2008).

We focus on haploid models of sex-specific selection, which allow for straightforward comparisons between the new models and established theories of sex-specific adaptation in constant environments (as reviewed above). By focusing on haploid selection, we neglect the potentially facilitating effects of genetic dominance on the maintenance of polymorphism (e.g., Gillespie and Langley 1974; Kidwell et al. 1977; Fry 2010; Sellis et al. 2011; Connallon and Clark 2014; Spencer and Priest 2016), which should once again render our predictions about polymorphism conservative.

Each model follows the evolution of a single locus with two alleles, *A* and *B*, that segregate at population frequencies of $p$ and $q = 1 - p$, respectively. The relative fitness of each

**Table 2:** Sex-specific relative fitnesses for genotypes *A* and *B* in the *i*th environment type

| | Genotype *A* | Genotype *B* |
|---|---|---|
| Female fitness | $w_{Ai}$ | $w_{Bi}$ |
| Male fitness | $v_{Ai}$ | $v_{Bi}$ |

genotype depends on the sex and environmental context in which it is expressed (table 2). As in prior theory, with which we draw comparisons, fitness is frequency and density independent (e.g., Levene 1953; Dempster 1955; Li 1955; Christiansen 1975; Felsenstein 1976; Kidwell 1977; Gregorius 1982; Immler et al. 2012). All models further assume that (1) generations are discrete; (2) each population is sufficiently large and demographically stable that we can ignore genetic drift, sperm limitation, and the possibility of local extinctions; and (3) males do not contribute to parental investment in offspring, so that the number of new births in a (sub)population is independent of the degree to which males are locally adapted (i.e., females are "demographically dominant"; Harts et al. 2014). Following previous population genetic theories of sex-specific selection (e.g., Kidwell et al. 1977; Gregorius 1982; Immler et al. 2012), the life cycle in our models assumes random mating among the postselection pool of adults in each patch. All four models will therefore capture effects of viability selection, whereas only a subset apply to cases of fecundity or fertility selection (e.g., models 2 and 4).

Below we present an overview of the major results of each model, with most of the mathematical details provided in appendixes. For analyses of sexually antagonistic alleles and sex-limited polymorphisms, we focus on the simplest context of environmental heterogeneity, in which there are two equally common environment types (or "habitats") in time or space. Interested readers can obtain results for various other scenarios of environmental heterogeneity by evaluating the general equations provided in the appendixes.

### *Model 1: Spatially Heterogeneous Selection with Adult Dispersal*

When sex-specific selection occurs during early life, followed by dispersal and then mating among adults, the expected change in frequency of the *A* allele per generation becomes

$$\begin{aligned}\Delta p &= \frac{p(1-p)}{2}\left(\frac{d\ln(\overline{\mathbb{W}}_{\mathrm{f}})}{dp}+\frac{d\ln(\overline{\mathbb{W}}_{\mathrm{m}})}{dp}\right)\\ &= \frac{p(1-p)}{2}\frac{d\ln(\overline{\mathbb{W}}_{\mathrm{f}}\overline{\mathbb{W}}_{\mathrm{m}})}{dp}\end{aligned} \tag{5}$$

(see app. B), where $\overline{\mathbb{W}}_{\mathrm{f}}$ and $\overline{\mathbb{W}}_{\mathrm{m}}$ represent the weighted arithmetic mean fitness of each sex. These are defined as $\overline{\mathbb{W}}_{\mathrm{f}} = \sum_{i=1}^{n} c_i(w_{Bi} + p(w_{Ai} - w_{Bi}))$ and $\overline{\mathbb{W}}_{\mathrm{m}} = \sum_{i=1}^{n} c_i(v_{Bi} + p(v_{Ai} - v_{Bi}))$, where $c_i$ represents the fraction of $n$ habitat types that are of the $i$th type ($\sum c_i = 1$). Equation (5) shows that the selection gradient of each sex favors an increase in arithmetic mean fitness of that sex (i.e., selection in males favors the increase of $\overline{\mathbb{W}}_{\mathrm{m}}$, selection in females favors the increase of $\overline{\mathbb{W}}_{\mathrm{f}}$). Selection acting jointly in both sexes maximizes the product $\overline{\mathbb{W}}_{\mathrm{f}}\overline{\mathbb{W}}_{\mathrm{m}}$. The evolutionary dynamics therefore remain structurally similar to those of standard models of sex-specific selection (see eqq. [1]–[3]). Effects of environmental heterogeneity among habitat patches are subsumed into the expressions for arithmetic mean fitness of each sex.

Interestingly, the mode of sex-specific selection captured by equation (5) bears a striking resemblance to classical hard selection scenarios of adaptation in subdivided populations. Under hard selection (and ignoring sex differences), evolution favors alleles that increase arithmetic mean population fitness (see Dempster 1955; Christiansen 1975; Connallon and Hall 2018, app. 3). In equation (5), evolution increases the product of male and female arithmetic mean fitness ($\overline{\mathbb{W}}_{\mathrm{f}}\overline{\mathbb{W}}_{\mathrm{m}}$), yet the nature of selection within each sex—as captured by the sex-specific selection gradients, $d\ln(\overline{\mathbb{W}}_{\mathrm{f}})/dp$ and $d\ln(\overline{\mathbb{W}}_{\mathrm{m}})/dp$—conforms exactly to hard selection scenarios noted in previous theory.

*Sexually Antagonistic Alleles.* Under sexually antagonistic selection, the *A* allele is favored in females and the *B* allele is favored in males. Using notation analogous to figure 1 (the constant environment model; see Kidwell et al. 1977), let $s_{\mathrm{f}i}$ and $s_{\mathrm{m}i}$ represent the female and male selection coefficients in the $i$th habitat type (i.e., $w_{Ai} = 1$, $w_{Bi} = 1 - s_{\mathrm{f}i}$, $v_{Ai} = 1 - s_{\mathrm{m}i}$, and $v_{Bi} = 1$; table 2). This fitness scheme results in sexual antagonism at both local and global scales (i.e., $d\ln(\overline{\mathbb{W}}_{\mathrm{f}})/dp$ and $d\ln(\overline{\mathbb{W}}_{\mathrm{m}})/dp$ have opposite signs).

As implied by the structure of equation (5), the evolutionary consequences of sexual antagonism remain symmetric between the sexes, with equal fractions of parameter space resulting in the fixation of female- and male-beneficial alleles (overlapping red and blue curves in fig. 2). Environmental variability promotes the maintenance of genetic variation beyond the comparatively restrictive conditions for polymorphism in a constant environment (cf. solid black and dotted curves in fig. 2). Whereas previous theory predicts that ~38% of random parameter combinations will maintain polymorphism (Pamilo 1979), a majority of parameter combinations (~55%) maintain polymorphism in the case of two equally abundant habitat patch types ($n = 1/c_1 = 1/c_2 = 2$). The discrepancy between results for constant and changing environments is most pronounced when selection coefficients are large (i.e., $\max(s_{\mathrm{f}}, s_{\mathrm{m}}) = 1$ in fig. 2).

*Sex-Limited Polymorphisms.* Although environmental heterogeneity expands conditions for maintaining sexually antagonistic polymorphisms, it cannot maintain variation under sex-limited selection (as shown in app. B), at least in the

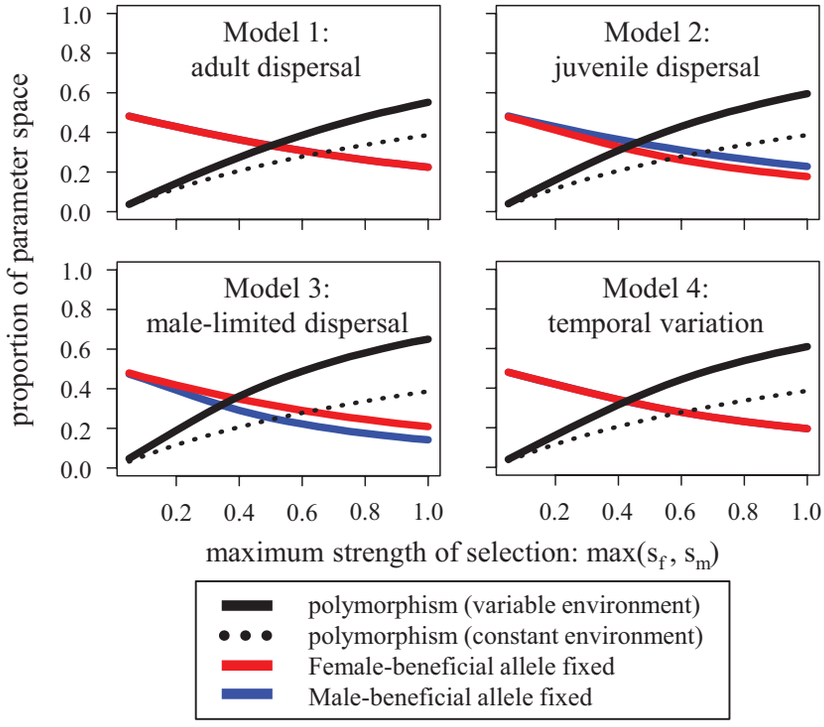


**Figure 2:** Evolutionary fates of sexually antagonistic alleles in models of spatially and temporally varying selection. Results are based on numerical evaluation of eigenvalues of boundary equilibria ($p = 0$ and $p = 1$) for scenarios involving two equally abundant habitat patches in space or two equally common environment types over time. Model 3 shows the case of male-limited dispersal; equivalent results with sexes reversed arise when females are the dispersing sex. Note that the red curves overlie the blue curves in models 1 and 4. The full parameter space for each model was explored by simulating $20 \times 10^6$ random parameter sets, with female and male selection coefficients for each patch ($s_{fi}$ and $s_{mi}$) drawn independently from uniform distributions with maximum defined by $\max(s_f, s_m)$. Parameter conditions for sexually antagonistic polymorphism in a constant environment are plotted for contrast and conform to previous theory (see Kidwell et al. 1977; Pamilo 1979; Gregorius 1982).

haploid selection context that we explored here. This conforms with previous models of hard selection without sex differences (Dempster 1955; Christiansen 1975; DéBarre and Gandon 2011).

### *Model 2: Spatially Heterogeneous Selection with Juvenile Dispersal*

When dispersal occurs during early life (prior to selection), the expected change in frequency of the *A* allele per generation becomes

$$\Delta p = \frac{p(1-p)}{2}\left(\frac{d\ln(\overline{\mathbb{W}}_f)}{dp} + \sum_{i=1}^{n}\frac{c_i\overline{w}_i}{\overline{\mathbb{W}}_f}\frac{d\ln(\overline{v}_i)}{dp}\right) \quad (6a)$$

(see app. C), where $c_i$ and $\overline{\mathbb{W}}_f$ are defined as above and $\overline{w}_i$ and $\overline{v}_i$ represent the mean relative fitness of females and males (respectively) that randomly disperse to habitat type *i*. Equation (6a) differs in structure from each of the models that we have seen so far (see eqq. [1]–[5]): the form of selection on each sex is no longer symmetric.

As in model 1, selection in females favors an increase in the allele that elevates their arithmetic mean fitness ($\overline{\mathbb{W}}_f$, as in hard selection models). In contrast, selection in males maximizes a quantity that is similar to the weighted geometric mean of male fitness. This becomes apparent if we focus on the special case of male-limited selection ($d\ln(\overline{\mathbb{W}}_f)/dp = 0$). In this case, equation (6a) simplifies to

$$\Delta p = \frac{p(1-p)}{2}\sum_{i=1}^{n}c_i\frac{d\ln(\overline{v}_i)}{dp} = \frac{p(1-p)}{2}\frac{d\ln(\overline{\mathbb{V}}_G)}{dp}, \quad (6b)$$

where $\overline{\mathbb{V}}_G = \prod_{i=1}^{n}\overline{v}_i^{c_i}$ is the weighted geometric mean fitness of males across the set of habitats. With male-limited selection, evolution maximizes the weighted geometric mean of fitness of males, as in classical models of soft selection (see eqq. [11]–[14] of Li 1955). When selection occurs in both sexes ($d\ln(\overline{\mathbb{W}}_f)/dp, d\ln(\overline{\mathbb{W}}_m)/dp \neq 0$), selection in males favors the maximization of a function that is similar to $\overline{\mathbb{V}}_G$ but that is also sensitive to selection on females (note the $\overline{w}_i/\overline{\mathbb{W}}_f$ terms within the summation of eq. [6a]). The end re-

sult is selection through males that is intermediate between the classical domains of hard and soft selection.

Asymmetric evolutionary responses to selection on each sex are consequences of localized mating within each habitat patch, which partially buffers subpopulations with weakly adapted males from having to compete against superior males from other subpopulations. This effectively softens selection on males. In contrast, selection remains hard for females because habitat patches with well-adapted females produce more offspring than do patches with poorly adapted females, leading to interpopulation competition for productivity.

*Sexually Antagonistic Alleles.* Using the same notation as before ($s_{fi}$ and $s_{mi}$ per environment $i$), we explored the parameter space of sexually antagonistic selection for the case of two equally abundant habitat patches ($c_1 = c_2 = 1/2$). Conditions for polymorphism were once again greater in a changing environment, with roughly 60% of parameter values resulting in polymorphism (fig. 2). Interestingly, male-beneficial alleles are fixed more readily than female-beneficial alleles. The sexual asymmetry in selection, which is inherent in equation (6a), therefore results in a sexual asymmetry in the fates of sexually antagonistic alleles and an evolutionary bias toward male adaptations.

*Sex-Limited Polymorphisms.* The asymmetry of selection in equation (6a) also impacts opportunities for maintaining sex-limited polymorphisms at loci responding to opposing selection between habitats (i.e., allele $A$ favored in some habitats and disfavored in others). Selection cannot maintain female-limited polymorphisms (see app. C), yet it can maintain male-limited polymorphisms. With two equally abundant habitat types ($c_1 = c_2 = 1/2$), conditions for polymorphism reduce to

$$\frac{t_2}{1 + t_2} < t_1 < \frac{t_2}{1 - t_2}, \tag{7}$$

where $t_1$ is the strength of selection in the first habitat and $t_2$ is the strength of opposing selection in the second ($0 < t_1$, $t_2 < 1$). This criterion is mathematically identical to the condition for maintaining codominant sexually antagonistic alleles in a constant environment (eq. [4]; Kidwell et al. 1977; Pamilo 1979) and for maintaining polymorphisms in Levene's (1953) model of soft selection with two patches and no dominance. As in these earlier models, roughly 38% of random parameter values maintain polymorphism (fig. 3).

### *Model 3: Spatially Heterogeneous Selection with Sex-Limited Dispersal*

With sex-limited dispersal, allele frequency differences between habitat patches can persist throughout the life cycle, and this precludes a simple analytical formula for allele frequency change (for exact results, see app. D). However, the exact result simplifies greatly as allele frequencies approach equality between habitat patches—a condition that may apply under weak selection or immediately following a species' invasion of the set of habitats. For example, when dispersal is female limited, the allele frequency change for the population is

$$\begin{aligned}\Delta p &= \frac{p(1-p)}{2}\left(\frac{d\ln(\overline{\mathbb{V}}_G)}{dp} + \frac{d\ln(\overline{\mathbb{W}}_f)}{dp}\right) + O(\delta_{pi}) \\ &\approx \frac{p(1-p)}{2}\frac{d\ln(\overline{\mathbb{V}}_G\overline{\mathbb{W}}_f)}{dp}\end{aligned} \tag{8}$$

(app. D), where $\overline{\mathbb{W}}_f = \sum_{i=1}^{n} c_i\overline{w}_i$ is the weighted arithmetic mean fitness of females, $\overline{\mathbb{V}}_G = \prod_{i=1}^{n}\overline{v}_i^{c_i}$ is the weighted geometric mean fitness of males, and $\delta_{pi}$ is the deviation in allele frequency between the $i$th patch and the global population

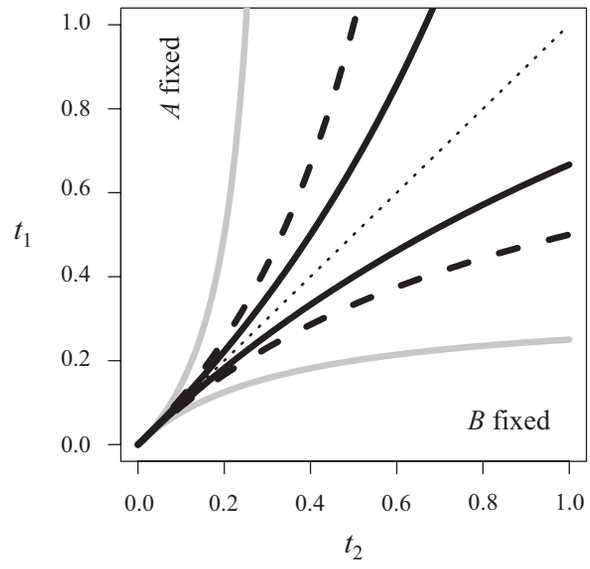


**Figure 3:** Conditions for maintaining female- or male-limited polymorphisms. All curves show the specific case of two equally abundant habitat patches in space or two equally common environment types over time. Allele $A$ is favored in one of the environments or patches, and allele $B$ is favored in the other, with selection coefficients $t_1$ and $t_2$ representing the fitness cost of carrying the disfavored allele within each of the two environment or patch types. Selection can maintain a sex-limited polymorphism that is expressed in (1) males in the juvenile dispersal model (model 2 between the dashed curves, based on eq. [7]), (2) the nondispersing sex in the sex-limited dispersal model (model 3 between the solid gray curves, based on eq. [9]), and (3) both sexes in the temporal variation model (model 4 between the solid black curves, based on eq. [13]). Selection cannot maintain a sex-limited polymorphism that is expressed in (1) females in the juvenile dispersal model and (2) the dispersing sex in the sex-limited dispersal model. In the latter cases, the dotted diagonal line delineates two possible outcomes of selection: $A$ is fixed above the line, and $B$ is fixed below the line. See the main text for further details.

frequency $p$. The element $O(\delta_{pi})$ refers to the terms of the exact equation of order $\delta_{pi}$, $\delta_{pi}^2$, $\delta_{pi}^3$, and so on. A similar result applies under male-limited dispersal but with the sexes reversed (see app. D).

The final approximation in equation (8), which ignores terms involving $\delta_{pi}$, is not particularly accurate unless the allele frequency differences among populations are small, yet it provides insight into the qualitative dynamics of sex-specific selection in species with strong sexual dimorphism in dispersal. As in the juvenile dispersal model (model 2), sex-limited dispersal buffers members of one sex—the nondispersing sex—from competition against members of the same sex that were born in a different habitat type. The buffering effect softens selection for the nondispersing sex, though selection remains hard for the dispersing sex, resulting in sexually asymmetric effects of selection on evolutionary dynamics (similar to the asymmetry in model 2).

*Sexually Antagonistic Alleles.* With two equally abundant habitat types ($c_1 = c_2 = 1/2$), the parameter space of sexually antagonistic alleles once again fundamentally differs from predictions in constant environments. As in models 1 and 2, conditions for polymorphism in model 3 were permissive compared to prior theory (eq. [4] and surrounding text), with ~65% of the total parameter space maintaining polymorphism (fig. 2). The sexual asymmetry in fixation of sexually antagonistic alleles is even more pronounced than in model 2, with alleles benefitting the nondispersing sex more likely to fix than alleles benefitting the dispersing sex (see fig. 2, which shows results for the male-limited dispersal scenario).

*Sex-Limited Polymorphisms.* Opportunities for maintaining sex-limited polymorphisms are extremely sex biased. Polymorphisms expressed in the dispersing sex cannot be maintained (see app. D), consistent with results from models 1 and 2 and standard theory invoking hard selection. In contrast, polymorphism is readily maintained when expressed in the nondispersing sex. With two equally abundant habitat types ($c_1 = c_2 = 1/2$), polymorphism is maintained under the following condition:

$$\frac{t_2}{1+3t_2} < t_1 < \frac{t_2}{1-3t_2} \tag{9}$$

(see app. D), where $t_1$ and $t_2$ represent the strengths of opposing selection between the patches (as in model 2). Across the entire parameter space ($0 < t_1,\ t_2 < 1$), the fraction of parameter combinations maintaining polymorphism is $1/3+4\ln(2)/9 \sim 0.64$ (i.e., nearly two-thirds of the parameter space; fig. 3).

### *Model 4: Temporally Heterogeneous Selection*

Under temporally variable selection, the expected change in allele frequency in a single generation depends on the distribution of fitness values per genotype over time. Equation (1) describes the change in frequency of the $A$ allele across a single generation and for a given set of genotypic fitness values. When the fitness values vary over time, the expected change for a given allele frequency $p$ is

$$\mathrm{E}[\Delta p] = \frac{p(1-p)}{2}\left[\mathrm{E}\left(\frac{d\ln(\overline{w}_\mathrm{f})}{dp}\right) + \mathrm{E}\left(\frac{d\ln(\overline{w}_\mathrm{m})}{dp}\right)\right], \tag{10}$$

which depends on the average strength and direction of selection in each sex: $\mathrm{E}(d\ln(\overline{w}_\mathrm{f})/dp)$ and $\mathrm{E}(d\ln(\overline{w}_\mathrm{m})/dp)$. The dynamics are sexually symmetric, as was also the case for model 1 and classical theory in stable environments.

The exact evolutionary trajectories of the $A$ and $B$ genotypes depend on both the distribution of relative fitnesses over time and the order of changes in the environment (Felsenstein 1976; Cvijović et al. 2015). While it does not appear possible to obtain general predictions for the allele frequencies over time, we can characterize general conditions for evolutionary invasion of each genotype and conditions for maintaining polymorphism. Following prior population genetic models of temporally fluctuating selection in large, demographically stable populations (Dempster 1955; Felsenstein 1976; Charlesworth and Charlesworth 2010, pp. 73–75; see app. E), the condition for deterministic evolutionary invasion of the $B$ genotype in a population initially fixed for $A$ is

$$1 < \prod_{t=0}^{t-1}\frac{1}{2}\left(\frac{w_{B,i}}{w_{A,i}} + \frac{v_{B,i}}{v_{A,i}}\right) \Leftrightarrow 0 < \mathrm{E}\left[\ln\left(\frac{w_{B,i}}{2w_{A,i}} + \frac{v_{B,i}}{2v_{A,i}}\right)\right], \tag{11}$$

and the condition for invasion of a rare $A$ genotype is

$$1 < \prod_{i=0}^{t-1}\frac{1}{2}\left(\frac{w_{A,i}}{w_{B,i}} + \frac{v_{A,i}}{v_{B,i}}\right) \Leftrightarrow 0 < \mathrm{E}\left[\ln\left(\frac{w_{A,i}}{2w_{B,i}} + \frac{v_{A,i}}{2v_{B,i}}\right)\right]. \tag{12}$$

Conditions (11) and (12) apply in the limit of large $t$ (i.e., over many generations). Polymorphism is maintained when conditions (11) and (12) are both true.

*Sexually Antagonistic Alleles.* With two equally common environment types over time, the parameter space for maintaining sexually antagonistic alleles is sexually symmetric. Conditions for polymorphism are substantially higher than those in constant environments (fig. 2), with roughly 60% of the total parameter space maintaining polymorphism.

*Sex-Limited Polymorphisms.* Opportunities for maintaining sex-limited polymorphisms are narrow and symmetric between the sexes (for similar results, see Reinhold 2000). With

two equally common environment types, a sex-limited polymorphism is maintained when

$$\frac{t_2}{1 + t_2/2} < t_1 < \frac{t_2}{1 - t_2/2} \tag{13}$$

(see app. E), where $t_1$ and $t_2$ represent the strengths of opposing selection between environment types (as in models 2 and 3). Across the entire parameter space ($0 < t_1, t_2 < 1$), the fraction of parameter combinations maintaining polymorphism is $8\ln(3/2) - 3 \sim 0.24$ (i.e., about three-quarters of the parameter space will not maintain polymorphism; fig. 3).

## Discussion

The classical theory of sex-specific selection makes two general predictions about the evolutionary dynamics of alleles that differentially affect female and male fitness. First, evolution should be as responsive to selection in females as it is to selection in males, leading to symmetrical opportunities for the adaptation of each sex to its environment (Wright 1942; Mandel 1971; Lande 1980). Second, sexually antagonistic selection is more likely to erode genetic variation than maintain it (Pamilo 1979; Prout 2000; Turelli and Barton 2004; Patten and Haig 2009; Jordan and Charlesworth 2012). These theoretical predictions are foundational to research on the evolution of sex differences, with associated models often guiding interpretation of empirical patterns of sex-specific selection and genetic variation (e.g., Fry 2010; Lewis et al. 2011; Gosden et al. 2012; Stearns et al. 2012; Cheng and Kirkpatrick 2016).

We have shown that both of the key predictions of prior theory can break down in changing environments. First, environmental variation across space can generate asymmetry in the evolutionary consequences of selection in females versus selection in males, leading to sex-biased opportunities for adaptation and sexually asymmetric conditions for maintaining polymorphisms (models 2 and 3). Second, environmental variation promotes the maintenance of genetic variation beyond the parameter-restrictive conditions for polymorphism in constant environments (cf. models 1–4; Pamilo 1979; Prout 2000). Below we discuss the scope and implications of these results for the study of adaptation in species with separate sexes. In light of this theory, we emphasize new empirical opportunities for exploring the effects of life history and demography on the evolutionary dynamics of female and male adaptations.

### *Sex-Biased Evolutionary Dynamics in Subdivided Populations*

Most genes are symmetrically transmitted from parents to same-sex and opposite-sex offspring. In the absence of environmental heterogeneity, the equal genetic contributions of females and males to reproduction equalize the impact of selection through females and males on the evolutionary dynamics of the population (Kidwell et al. 1977; Lande 1980; Wyman et al. 2013; eqq. [1]–[3]). Yet, in subdivided populations where the environment varies across space, the timing and sex specificity of dispersal can generate sex asymmetries in the nature of selection in each sex and the evolutionary response to natural selection.

Sex-biased outcomes of natural selection can arise in species where individuals disperse early during their life cycles (model 2) and those where one sex disperses readily while the other is strongly philopatric (model 3), leading to the preferential accumulation of male- or female-beneficial alleles and greater potential for maintaining sex-limited polymorphisms in one sex compared to the other. In contrast, opportunities for adaptation and polymorphism are sexually symmetric in species with late juvenile or adult-stage dispersal, provided both sexes disperse at similar rates (model 1). Insects conform to the idealized conditions of our model 1, as they have sedentary larval and highly dispersive adult stages and typically show similar patterns of male and female dispersal (see Markow and Castrezana 2000; Trochet et al. 2016; though dispersal data for insects are sparse). Coincidentally, insects are also the dominant study systems in sexual conflict research (e.g., Bonduriansky and Chenoweth 2009; Berger et al. 2014). Many other species have life-history attributes akin to those in our models 2 and 3, including plants and aquatic animals where early dispersal is the norm (i.e., seed and larval dispersal, as in model 2; Burgess et al. 2016) and terrestrial vertebrates with pronounced sex biases in dispersal (e.g., many bird and mammal taxa; Trochet et al. 2016). In light of the new theoretical predictions, dioecious plants and marine invertebrates with sessile adult stages may represent particularly useful taxa for future studies of sex-specific adaptation and sexual conflict, given the feasibility of estimating quantitative genetic parameters and fitness components within these systems (e.g., Delph et al. 2011; Chirgwin et al. 2017).

Why exactly do subdivided populations respond differently to selection in females versus selection in males? Concepts of hard and soft selection from classical theories of population subdivision (see Christiansen 1975; DéBarre and Gandon 2011) provide some insight into the underlying causes of evolutionary asymmetries between the sexes. Hard and soft selection represent two idealized extremes for the ways in which individuals across subdivided populations leave descendants. Under soft selection (Levene 1953; Christiansen 1975), the relative contribution of each subpopulation to reproduction remains fixed and independent of the genetic quality of individuals that inhabit them. Consequently, each individual's fitness is determined by their performance in competition against other individuals within their local habitat and not by individuals in other habitats in the species'

range. The absence of differential selection and competition among subpopulations ensures that individuals that perform well relative to their local competitors have high fitness, even if they carry low-quality genotypes at a global scale. In contrast, under hard selection (Dempster 1955; Christiansen 1975), individuals are fully exposed to selection and competition with members of their own and other subpopulations, and this full exposure to competition ensures that globally superior genotypes win the evolutionary contest. These two idealized forms of evolutionary competition lead to fundamentally different evolutionary responses to selection. Whereas soft selection increases the geometric mean fitness of individuals of the metapopulation and readily maintains genetic polymorphism, hard selection increases arithmetic mean fitness and erodes genetic variation.

Our models consistently invoke hard selection in the sense that density regulation is global and occurs after dispersal of individuals among subpopulations (i.e., density is controlled after adults disperse in models 1 and 3 and after juveniles disperse in model 2). Nevertheless, models 2 and 3 illustrate that juvenile or sex-biased dispersal can effectively soften selection for one sex while leaving it hard for the other. When juveniles disperse, male fitness is largely determined locally rather than globally, leading to a relatively soft form of selection in males. Yet, because offspring production of each population is tied to the survival and reproduction of its females, individual female fitness is determined relative to the fitness of other females in the entire metapopulation, and selection on females remains hard. A similar dynamic occurs under sex-limited dispersal, where local selection is all-important for the nondispersing sex (softening selection in that sex), whereas global selection applies to the dispersing sex (hardening selection in that sex). In both cases (models 2 and 3), maintaining sex-limited polymorphism is much more feasible for the sex under soft selection, and sexually antagonistic alleles are more likely to invade and fix when they benefit the sex under soft selection.

The sex asymmetries that emerge by way of demography in our models have some parallels in previous studies. First, sex-biased modes of inheritance, including sex linkage, mitochondrial inheritance, and evolution in self-fertilizing populations, can bias evolution toward the evolutionary interests of one sex over the other (Jordan and Charlesworth 2012; Jordan and Connallon 2014; Kuijper et al. 2015; Olito 2017; Smith and Connallon 2017; Olito et al. 2018). Such inheritance asymmetries appear to contribute, for example, to the accumulation of male-beneficial alleles on y chromosomes (e.g., in guppies; Charlesworth 2018) and male-harming alleles in the mitochondrial genome (e.g., in humans; Milot et al. 2017) and to the degeneration of male sex functions in self-fertilizing hermaphrodites (i.e., "selfing syndromes"; Sicard and Lenhard 2011). Second, a recent simulation study by Harts et al. (2014) demonstrated that female-beneficial sexually antagonistic alleles can, in at least some cases, preferentially spread in subdivided populations undergoing local adaptation, local extinction, and recolonization. This finding reinforces one of the major messages of this article: that the evolutionary dynamics of sex-specific adaptation critically hinge on the details of a species' life history and demography.

### *Sex-Specific Selection and the Maintenance of Genetic Variation*

The pervasiveness of balancing selection is a long-standing and unresolved question in evolutionary biology. Although enthusiasm for balancing selection peaked and subsequently declined more than half a century ago (e.g., Dobzhansky 1955), recent analyses of quantitative genetic data (Charlesworth 2015; Sharp and Agrawal 2018), identification of candidate genes under balancing selection (e.g., Leffler et al. 2013; Chakraborty and Fry 2016; Unckless et al. 2016; despite low statistical power [Fijarczyk and Babik 2015]), and new theory (Sellis et al. 2011; Connallon and Clark 2014) all suggest that balancing selection is likely to play an important role in maintaining genetic variation for fitness.

Several mechanisms of balancing selection can potentially maintain variation. The question is which ones are most common. Our results show that interactive effects of sex-specific selection and environmental heterogeneity substantially expand the range of conditions leading to balancing selection, compared to predictions based on either mechanism in isolation (e.g., environmental heterogeneity [Levene 1953; Dempster 1955; Felsenstein 1976]; sexual antagonism [Kidwell et al. 1977; Fry 2010; Connallon and Clark 2014]). For example, we find that a majority of the parameter space in our models leads to balancing selection—a substantial increase of parameter space relative to prior theory (e.g., at least 50% of parameter combinations in our models vs. ~38% in haploid and codominant diploid models of sexual antagonism in constant environments; Pamilo 1979). While conditions for balancing selection remain restrictive when selection is weak (see fig. 2; Prout 2000), empirical examples of candidate sexually antagonistic polymorphisms suggest that strong selection is plausible in specific cases (Forsman 1995; Forsman and Shine 1995; Barson et al. 2015). Whether or not selection is strong, interactions between sex and environmental heterogeneity expand criteria for maintaining polymorphism. Moreover, the new theoretical conclusions regarding balanced polymorphisms are potentially conservative, as they neglect the potentially facilitating effects of dominance on the maintenance of genetic variation (see Fry 2010; Connallon and Clark 2014; Barson et al. 2015; Spencer and Priest 2016).

Our results also place a spotlight on sex-limited polymorphisms as a potential reflection of sex-biased evolutionary

dynamics in structured populations (models 2 and 3). Conspicuous sex-limited color polymorphisms have been documented in a wide range of animal species, including mammals, birds, reptiles, fish, and insects (Galeotti et al. 2003; Van Gossum et al. 2008; Wellenreuther et al. 2014). The dynamics of these polymorphisms are potentially influenced by environmental heterogeneity, frequency-dependent selection, and sexual conflict (Calsbeek et al. 2010; Wellenreuther et al. 2014; Svensson 2017). Our models predict that such polymorphisms should be particularly common in species with strong sex-biased dispersal and species where dispersal occurs early during the life cycle. These predictions should be testable using modern comparative phylogenetic approaches to formally, statistically assess associations between dispersal traits and the presence or absence of sex-limited polymorphisms.

## Conclusion

Interactions between sex differences and environmental variation can yield surprising evolutionary outcomes that are not always obvious from theories dealing with each factor in isolation (Harts et al. 2014; Connallon et al. 2018). Although population genetic theories of environmental variation and sex-specific selection are individually well developed (Felsenstein 1976; Kidwell et al. 1977; DéBarre and Gandon 2011; Jordan and Charlesworth 2012), rarely are the two contexts of selection merged and analyzed together (e.g., Reinhold 2000). As a launching point, we have analyzed four simple models of sex-specific adaptation in changing environments. These simple models often produce surprising results when measured against predictions of classical population genetics models of sex-specific selection in stable environments.

The four models reveal diverse evolutionary consequences of sex-specific selection in heterogeneous environments. The theory broadly implies that the evolutionary "rules" for adaptation in species with separate sexes may vary from species to species. The predictions of this theory provide motivation for diversifying the range of species that serve as models for studying sex-specific selection and the evolutionary genetic consequences of sexual conflict. The theory also stands to enrich our interpretation of documented patterns of sexual dimorphism and sex-specific selection across species with different life-history attributes (e.g., Cox and Calsbeek 2009; Morrissey 2016; De Lisle et al. 2018) and among populations that evolved in simple versus complex habitats (Yun et al. 2017, 2018). Finally, our results point to future opportunities for theoretical work that expands on our framework by considering effects of frequency- and density-dependent selection (Mokkonen et al. 2011; Olito et al. 2017), assortative mating (Seger and Trivers 1986; Albert and Otto 2005; Arnqvist 2011), habitat choice (Ravigné et al. 2004), different forms of sex-dependent population dynamics (see Lande 2007, 2008; Rankin and Kokko 2007), and genetic drift (Connallon and Clark 2012, 2014; Mullen et al. 2012) on the evolution of female and male adaptations.

## Acknowledgments

We are grateful for comments by Clémentine Lasne, Sandra Hangartner, Genevieve Matthews, Crystal Dao, Daniel Bolnick, Locke Rowe, and two anonymous reviewers. This work was funded by the Australian Research Council and the School of Biological Sciences at Monash University. C.O. was supported by a Dean's International Graduate Student Scholarship and a Dean's Graduate Student Scholarship.

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Associate Editor: Locke Rowe
Editor: Daniel I. Bolnick

 DOI: 10.1086/700720

# Appendix A from T. Connallon et al., "Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes" (Am. Nat., vol. 193, no. 1, p. 000)

## Invasion and Polymorphism Conditions for the x and Autosomes

### Diploid Loci

With small allele frequency differences between breeding females and males—applicable when one of the two alleles is rare or when selection is weak—the mean fitness of each sex can be approximated as

$$\overline{W}_{\mathrm{f}} \approx p^2 w_{AA} + 2p(1-p)w_{AB} + (1-p)^2 w_{BB}$$

and

$$\overline{W}_{\mathrm{m}} \approx p^2 v_{AA} + 2p(1-p)v_{AB} + (1-p)^2 v_{BB}.$$

Conditions for invasion of rare alleles (i.e., invasion of $A$ when $p \approx 0$ and $B$ when $p \approx 1$) may be obtained through analysis of the selection gradient for each of the boundary equilibria. A rare $A$ allele should invade when

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=0} > 0.$$

A rare $B$ allele should invade when

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=1} < 0.$$

Using the above approximations for mean fitness, we get

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=0} = \frac{2(w_{AB}-w_{BB})}{w_{BB}} + \frac{2(v_{AB}-v_{BB})}{v_{BB}}$$

and

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=1} = \frac{2(w_{AA}-w_{AB})}{w_{AA}} + \frac{2(v_{AA}-v_{AB})}{v_{AA}}.$$

Though they are based on approximations of mean fitness per sex, the above analysis nevertheless arrives at the standard exact results for invasion and polymorphism at diploid loci. For example, the $A$ allele invades when

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=0} > 0 \Leftrightarrow 1 < \frac{w_{AB}}{2w_{BB}} + \frac{v_{AB}}{2v_{BB}}.$$

The $B$ allele will invade when

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=1} < 0 \Leftrightarrow 1 < \frac{w_{AB}}{2w_{AA}} + \frac{v_{AB}}{2v_{AA}}.$$

Balancing selection (protected polymorphism) occurs when both conditions are true. These results are identical to those obtained by evaluating the leading eigenvalues of the exact set of recursion equations (see Kidwell et al. 1977, p. 177).

## X-Linked Loci

Following Campos Rosado and Robertson (1966), with weak selection at an x-linked locus so that allele frequency differences between sexes are negligible, mean fitness per sex is

$$\overline{W}_{\mathrm{f}} = p^2 w_{AA} + 2p(1-p)w_{AB} + (1-p)^2 w_{BB},$$
$$\overline{W}_{\mathrm{m}} = p v_A + (1-p)v_B,$$

where $p$ is the frequency of the $A$ allele at birth. The within-generation changes in allele frequency for females and males, respectfully, are

$$\Delta p_{\mathrm{f}} \approx \frac{p(1-p)}{2}\frac{d\ln(\overline{W}_{\mathrm{f}})}{dp},$$
$$\Delta p_{\mathrm{m}} \approx p(1-p)\frac{d\ln(\overline{W}_{\mathrm{m}})}{dp}.$$

The change in frequency across a single generation is

$$\Delta p = \frac{2\Delta p_{\mathrm{f}} + \Delta p_{\mathrm{m}}}{3} \approx \frac{p(1-p)}{3}\left(\frac{d\ln(\overline{W}_{\mathrm{f}})}{dp} + \frac{d\ln(\overline{W}_{\mathrm{m}})}{dp}\right) = \frac{p(1-p)}{3}\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp},$$

as first presented by Wright (1942) and later derived by Campos Rosado and Robertson (1966). The approximation is fairly accurate as long as allele frequency differences between sexes at birth are small, which requires that selection is weak.

Assuming weak selection, the final approximation can be used to obtain approximate conditions for invasion from each of the two boundary equilibria. These conditions are

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=0} > 0 \Leftrightarrow \frac{2(w_{BB}-w_{AB})}{w_{BB}} < \frac{v_A - v_B}{v_B}$$

and

$$\left.\frac{d\ln(\overline{W}_{\mathrm{f}}\overline{W}_{\mathrm{m}})}{dp}\right|_{p=1} < 0 \Leftrightarrow \frac{2(w_{AA}-w_{AB})}{w_{AA}} < \frac{v_B - v_A}{v_A}.$$

Under weak sexually antagonistic selection, with $w_{AA} = v_B = 1$, $w_{AB} = 1 - s_{\mathrm{f}}h$, $w_{BB} = 1 - s_{\mathrm{f}}$, and $v_A = 1 - s_{\mathrm{m}}$, the condition for maintaining polymorphism becomes

$$2s_{\mathrm{f}}h < s_{\mathrm{m}} < 2s_{\mathrm{f}}(1-h),$$

which ignores terms of $O(s_{\mathrm{f}}s_{\mathrm{m}}, s_{\mathrm{f}}^2, s_{\mathrm{m}}^2)$. When selection is weak and/or male-beneficial alleles are recessive in females, this approximation compares well to exact results derived in previous studies, for example, equation (7) of Patten and Haigh (2009):

$$\frac{2s_{\mathrm{f}}h}{1+hs_{\mathrm{f}}} < s_{\mathrm{m}} < \frac{2s_{\mathrm{f}}(1-h)}{1-hs_{\mathrm{f}}}.$$

As selection or dominance increases in strength, the approximation increasingly underestimates the parameter conditions for polymorphism. When $h > 0.5$ (male-beneficial alleles are dominant to female-beneficial alleles), the approximation predicts that polymorphism cannot be maintained. Although this prediction holds true under weak selection, it breaks down when selection is strong. Figure A1 presents representative contrasts between approximate and exact invasion and polymorphism results for sexual antagonism at an x-linked locus.

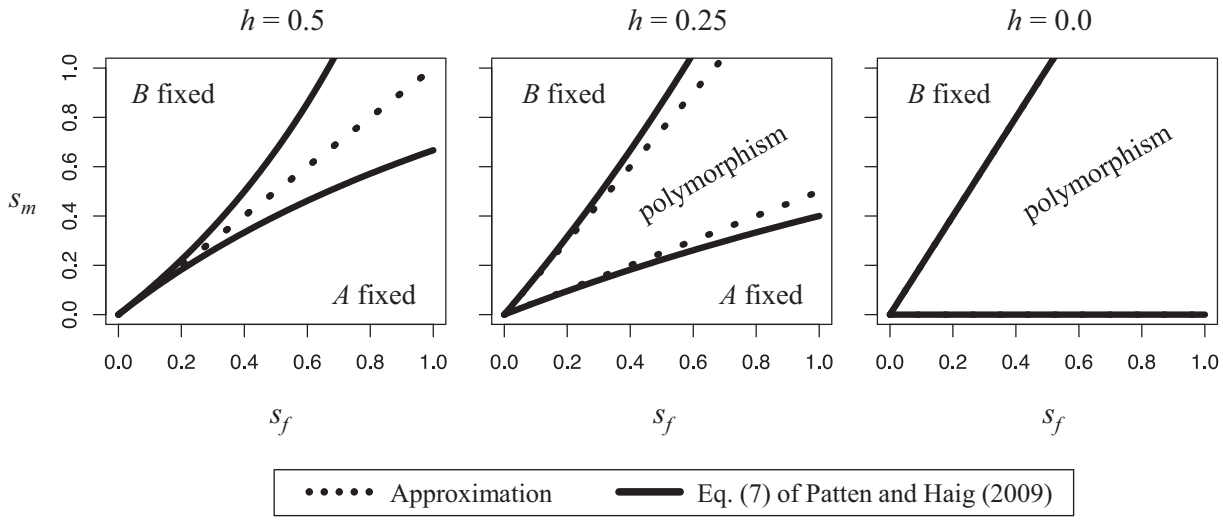


**Figure A1:** Evolutionary fates of x-linked sexually antagonistic alleles in a constant environment. Curves are based on the inequalities in appendix A, where allele *A* is the female-beneficial allele and *B* is the male-beneficial allele.

 DOI: 10.1086/700720

# Appendix B from T. Connallon et al., "Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes" (Am. Nat., vol. 193, no. 1, p. 000)

## Model 1: Adult Dispersal

### Evolutionary Dynamics

The life cycle is birth, selection, and dispersal, followed by random mating, reproduction, and death. Random dispersal among the habitats equalizes allele frequencies between patches at the beginning of each generation. The frequency of the $i$th habitat type is $c_i$, where $\Sigma c_i = 1$. We assume that the number of adults of each sex that emerge from a given patch is proportional to the degree of local adaption of members of that sex that were born in the patch (this follows standard assumptions of hard selection; see Christiansen 1975). Under these conditions, the allele frequency recursion for a single generation is

$$p' = \frac{1}{2}\sum_{i=1}^{n}\frac{c_i\overline{w}_i}{\overline{\mathbb{W}}_{\mathrm{f}}}p_{i\mathrm{f}} + \frac{1}{2}\sum_{i=1}^{n}\frac{c_i\overline{v}_i}{\overline{\mathbb{W}}_{\mathrm{m}}}p_{i\mathrm{m}} = \frac{1}{2}\sum_{i=1}^{n}\frac{c_i\overline{w}_i}{\overline{\mathbb{W}}_{\mathrm{f}}}\frac{pw_{Ai}}{\overline{w}_i} + \frac{1}{2}\sum_{i=1}^{n}\frac{c_i\overline{v}_i}{\overline{\mathbb{W}}_{\mathrm{m}}}\frac{pv_{Ai}}{\overline{v}_i},$$

where $n$ is the total number of habitat patch types, $\overline{w}_i$ and $\overline{v}_i$ are the sex-specific mean fitnesses in the $i$th patch type, and $\overline{\mathbb{W}}_{\mathrm{f}}$ and $\overline{\mathbb{W}}_{\mathrm{m}}$ are weighted arithmetic mean fitnesses for the sexes:

$$\overline{\mathbb{W}}_{\mathrm{f}} = \sum_{i=1}^{n} c_i\overline{w}_i = \sum_{i=1}^{n} c_i(w_{Bi} + p(w_{Ai} - w_{Bi}))$$

and

$$\overline{\mathbb{W}}_{\mathrm{m}} = \sum_{i=1}^{n} c_i\overline{v}_i = \sum_{i=1}^{n} c_i(v_{Bi} + p(v_{Ai} - v_{Bi})).$$

Using the expressions immediately above and following some algebra, the change in allele frequency across a generation eventually reduces to the expression in equation (5):

$$\begin{aligned}
\Delta p &= \frac{p}{2\overline{\mathbb{W}}_{\mathrm{f}}}\sum_{i=1}^{n} c_i(w_{Ai} - \overline{w}_i) + \frac{p}{2\overline{\mathbb{W}}_{\mathrm{m}}}\sum_{i=1}^{n} c_i(v_{Ai} - \overline{v}_i)\\
&= \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{f}}}\sum_{i=1}^{n} c_i\frac{d\overline{w}_i}{dp} + \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{m}}}\sum_{i=1}^{n} c_i\frac{d\overline{v}_i}{dp}\\
&= \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{f}}}\frac{d}{dp}\sum_{i=1}^{n} c_i\overline{w}_i + \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{m}}}\frac{d}{dp}\sum_{i=1}^{n} c_i\overline{v}_i\\
&= \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{f}}}\frac{d\overline{\mathbb{W}}_{\mathrm{f}}}{dp} + \frac{p(1-p)}{2\overline{\mathbb{W}}_{\mathrm{m}}}\frac{d\overline{\mathbb{W}}_{\mathrm{m}}}{dp} = \frac{p(1-p)}{2}\frac{d\ln(\overline{\mathbb{W}}_{\mathrm{f}}\overline{\mathbb{W}}_{\mathrm{m}})}{dp}.
\end{aligned}$$

The final result is presented as equation (5) in the main text.

### Evolutionary Invasion and Polymorphism in the Two-Patch Version of the Model

When there are two equally abundant habitat patch types ($c_1 = c_2$), the recursion equation simplifies to

$$p' = \frac{p(w_{A1} + w_{A2})}{4\overline{\mathbb{W}}_{\mathrm{f}}} + \frac{p(v_{A1} + v_{A2})}{4\overline{\mathbb{W}}_{\mathrm{m}}},$$

where

$$\overline{\mathbb{W}}_{\mathrm{f}} = \frac{1}{2}(w_{B1} + p(w_{A1} - w_{B1})) + \frac{1}{2}(w_{B2} + p(w_{A2} - w_{B2}))$$

and

$$\overline{\mathbb{W}}_{\mathrm{m}} = \frac{1}{2}(v_{B1} + p(v_{A1} - v_{B1})) + \frac{1}{2}(v_{B2} + p(v_{A2} - v_{B2})).$$

The recursion can be used to calculate eigenvalues for the two boundary equilibria (i.e., for $p = 0$ and $p = 1$, respectively):

$$\left.\frac{dp'}{dp}\right|_{p=0} = \frac{w_{A1} + w_{A2}}{2(w_{B1} + w_{B2})} + \frac{v_{A1} + v_{A2}}{2(v_{B1} + v_{B2})}$$

and

$$\left.\frac{dp'}{dp}\right|_{p=1} = \frac{w_{B1} + w_{B2}}{2(w_{A1} + w_{A2})} + \frac{v_{B1} + v_{B2}}{2(v_{A1} + v_{A2})}.$$

Note that each eigenvalue depends on the sex-specific arithmetic mean fitness of each allele, averaged across the two environments.

*Conditions for Sex-Limited Polymorphism*

When selection acts only in females ($v_{ij} = 1$), balancing selection requires that both of the following conditions are met:

$$1 < \frac{w_{A1} + w_{A2}}{w_{B1} + w_{B2}}$$

and

$$1 < \frac{w_{B1} + w_{B2}}{w_{A1} + w_{A2}}.$$

Since the right-hand side of the first condition is the reciprocal of the right-hand side of the second, it is clear that only one condition can be true. Consequently, balancing selection is impossible when selection is female limited. Under male-limited evolution ($w_{ij} = 1$), results are effectively the same as in the female-limited case.

 DOI: 10.1086/700720

# Appendix C from T. Connallon et al., "Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes" (Am. Nat., vol. 193, no. 1, p. 000)

## Model 2: Juvenile Dispersal

### Evolutionary Dynamics

The life cycle is birth, dispersal, and selection, followed by random mating, reproduction, and death. Random dispersal among the habitats equalizes allele frequencies between patches at the beginning of each generation. Once again, the frequency of the $i$th habitat type is $c_i$, where $\Sigma c_i = 1$. We census the population immediately after dispersal, at which time the frequency of $A$ is equalized across patches ($=p$). After local selection, the frequency in the postselection pool of breeding adults within the $i$th patch is

$$p_i = \frac{pw_{Ai}}{2\overline{w}_i} + \frac{pv_{Ai}}{2\overline{v}_i},$$

where $\overline{w}_i$ and $\overline{v}_i$ represent mean relative fitness of females and males that dispersed to the $i$th habitat type (i.e., because census occurs after dispersal in model 2, rather than at birth as in model 1).

With new births being female dependent, the fraction of offspring in the next generation that are attributable to the $i$th patch is

$$\frac{c_i\overline{w}_i}{\overline{\overline{\mathbb{W}}}_{\mathrm{f}}},$$

where $\overline{\overline{\mathbb{W}}}_{\mathrm{f}}$ is the weighted arithmetic mean fitness of females for the entire set of $n$ habitat patches: $\overline{\overline{\mathbb{W}}}_{\mathrm{f}} = \sum_{i=1}^{n} c_i\overline{w}_i = \sum_{i=1}^{n} c_i(w_{Bi} + p(w_{Ai} - w_{Bi}))$. Taking both patch-specific allele frequencies and patch-specific productivity into account, the allele frequency in the next generation will be

$$p' = \sum_{i=1}^{n} \frac{c_i\overline{w}_i}{\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} p_i = \frac{p}{2\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \sum_{i=1}^{n} c_i\overline{w}_i \left(\frac{w_{Ai}}{\overline{w}_i} + \frac{v_{Ai}}{\overline{v}_i}\right).$$

The allele frequency change per generation is

$$\Delta p = \frac{p}{2\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \sum_{i=1}^{n} c_i\overline{w}_i \left(\frac{w_{Ai} - \overline{w}_i}{\overline{w}_i} + \frac{v_{Ai} - \overline{v}_i}{\overline{v}_i}\right) = \frac{p(1-p)}{2\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \sum_{i=1}^{n} \left(c_i \frac{d\overline{w}_i}{dp} + c_i \frac{\overline{w}_i}{\overline{v}_i} \frac{d\overline{v}_i}{dp}\right)$$

$$= \frac{p(1-p)}{2} \left(\frac{d\ln(\overline{\overline{\mathbb{W}}}_{\mathrm{f}})}{dp} + \sum_{i=1}^{n} \frac{c_i\overline{w}_i}{\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \frac{d\ln(\overline{v}_i)}{dp}\right),$$

with the final result presented as equation (6) in the main text.

### Evolutionary Invasion and Polymorphism in the Two-Patch Version of the Model

When there are two equally abundant habitat patch types ($c_1 = c_2$), the recursion equation simplifies to

$$p' = \frac{p}{4\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \overline{w}_1 \left(\frac{w_{A1}}{\overline{w}_1} + \frac{v_{A1}}{\overline{v}_1}\right) + \frac{p}{4\overline{\overline{\mathbb{W}}}_{\mathrm{f}}} \overline{w}_2 \left(\frac{w_{A2}}{\overline{w}_2} + \frac{v_{A2}}{\overline{v}_2}\right),$$

where

$$\overline{\mathbb{W}}_{\mathrm{f}} = \frac{1}{2}(w_{B1} + p(w_{A1} - w_{B1})) + \frac{1}{2}(w_{B2} + p(w_{A2} - w_{B2})).$$

The recursion can be used to calculate eigenvalues for the two boundary equilibria (i.e., for $p = 0$ and $p = 1$, respectively):

$$\left.\frac{dp'}{dp}\right|_{p=0} = \frac{w_{B1}}{2(w_{B1} + w_{B2})}\left(\frac{w_{A1}}{w_{B1}} + \frac{v_{A1}}{v_{B1}}\right) + \frac{w_{B2}}{2(w_{B1} + w_{B2})}\left(\frac{w_{A2}}{w_{B2}} + \frac{v_{A2}}{v_{B2}}\right)$$

and

$$\left.\frac{dp'}{dp}\right|_{p=1} = \frac{w_{A1}}{2(w_{A1} + w_{A2})}\left(\frac{w_{B1}}{w_{A1}} + \frac{v_{B1}}{v_{A1}}\right) + \frac{w_{A2}}{2(w_{A1} + w_{A2})}\left(\frac{w_{B2}}{w_{A2}} + \frac{v_{B2}}{v_{A2}}\right).$$

*Conditions for Sex-Limited Polymorphism*

When selection acts only in females ($v_{ij} = 1$), balancing selection requires that both of the following conditions are met:

$$1 < \frac{w_{B1}}{2(w_{B1} + w_{B2})}\left(\frac{w_{A1}}{w_{B1}} + 1\right) + \frac{w_{B2}}{2(w_{B1} + w_{B2})}\left(\frac{w_{A2}}{w_{B2}} + 1\right)$$

and

$$1 < \frac{w_{A1}}{2(w_{A1} + w_{A2})}\left(\frac{v_{B1}}{v_{A1}} + \frac{w_{B1}}{w_{A1}}\right) + \frac{w_{A2}}{2(w_{A1} + w_{A2})}\left(\frac{w_{B2}}{w_{A2}} + \frac{v_{B2}}{v_{A2}}\right).$$

As in model 1 (adult dispersal), both conditions cannot simultaneously be true.

When selection acts only in males ($w_{ij} = 1$), polymorphism can be maintained when both of the following conditions are true:

$$1 < \frac{v_{A1}}{2v_{B1}} + \frac{v_{A2}}{2v_{B2}}$$

and

$$1 < \frac{v_{B1}}{2v_{A1}} + \frac{v_{B2}}{2v_{A2}}.$$

Interestingly, these conditions correspond to criteria for maintaining polymorphism in Levene's (1953) model of soft selection (i.e., accounting for haploid selection, male-limited expression, and equal patch abundances, $c_1 = c_2 = 1/2$), as well as conditions for maintaining polymorphism in a single population with separate sexes (if we let females correspond to environment 1 and males correspond to environment 2).

These parallels with previous models inform us about the opportunity for balancing selection of alleles subject to male-limited selection. If there is a fitness trade-off between two environments (such that $v_{B1}/v_{A1} > 1$ and $v_{B2}/v_{A2} < 1$), then we can reparameterize the model so that the $A$ allele is favored in one environment and $B$ is favored in the other. Let $t_1$ and $t_2$ represent selection coefficients for the maladapted allele in each of the two environments (1 and 2, respectively; table C1). The condition for balancing selection of a male-limited polymorphism becomes

$$\frac{t_2}{1 + t_2} < t_1 < \frac{t_2}{1 - t_2},$$

which is identical to the condition for maintaining a sexually antagonistic polymorphism in a haploid or codominant diploid model of selection (see above; Kidwell et al. 1977). Roughly 38% of the parameter range ($0 < t_1, t_2 < 1$) will result in balancing selection on male-limited polymorphisms, which is substantially higher than conditions for balancing selection of female-limited polymorphisms (0% for females).

**Table C1:** Sex-specific relative fitnesses for genotypes $A$ and $B$ in an arbitrary environment $i$

| | Genotype $A$ | Genotype $B$ |
|---|---|---|
| Male fitness (environment 1) | 1 | $1 - t_1$ |
| Male fitness (environment 2) | $1 - t_2$ | 1 |

 DOI: 10.1086/700720

# Appendix D from T. Connallon et al., "Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes" (Am. Nat., vol. 193, no. 1, p. 000)

## Model 3: Sex-Limited Dispersal

### Evolutionary Dynamics

With sex-biased migration, allele frequencies per patch can remain differentiated even after dispersal. In such cases, we allow for allele frequency at census (birth), with a census frequency of $p_i$ for the $i$th patch. We first consider the scenario where females disperse and males remain within the habitats in which they were born. We then consider the opposite case, where males disperse and females remain in their natal patch.

*Model 3A: Female-Limited Dispersal*

At birth, the initial frequency in a given patch (patch $i$) is $p_i$. Following selection in the $i$th patch, the frequency of the $A$ allele in the postselection population of females and males will be

$$p_{i\mathrm{f}} = \frac{p_i w_{Ai}}{\overline{w}_i}$$

and

$$p_{i\mathrm{m}} = \frac{p_i v_{Ai}}{\overline{v}_i},$$

where $\overline{w}_i = w_{Bi} + p_i(w_{Ai} - w_{Bi})$ and $\overline{v}_i = v_{Bi} + p_i(v_{Ai} - v_{Bi})$.

Males do not disperse, whereas females disperse randomly among patches. We assume that the number of females emerging from a given patch is proportional to the degree of local adaption of females that were born in that patch (the standard hard selection assumption; see Christiansen 1975). Thus, the overall allele frequency among dispersing females will be

$$p_{\mathrm{f}} = \sum_{i=1}^{n} \frac{c_i \overline{w}_i}{\overline{\mathbb{W}}_{\mathrm{f}}} \frac{p_i w_{Ai}}{\overline{w}_i},$$

with $\overline{\mathbb{W}}_{\mathrm{f}}$ defined as before. Dispersal of females equalizes allele frequencies of breeding females in each of the habitats. Following random mating within patches, the frequency of the $A$ allele in offspring of the next generation will be

$$p_i' = \frac{1}{2}(p_{i\mathrm{m}} + p_{\mathrm{f}}) = \frac{1}{2}\left(\frac{p_i v_{Ai}}{\overline{v}_i} + \sum_{j=1}^{n} \frac{c_j \overline{w}_j}{\overline{\mathbb{W}}_{\mathrm{f}}} \frac{p_j w_{Aj}}{\overline{w}_j}\right),$$

where we use $j$ to distinguish between $i$ of the focal patch. The allele frequency change in the $i$th patch is

$$\begin{aligned}\Delta p_i &= \frac{1}{2}\left(\frac{p_i(v_{Ai} - \overline{v}_i)}{\overline{v}_i} + \frac{1}{\overline{\mathbb{W}}_{\mathrm{f}}}\left[\left(\sum_{j=1}^{n} c_j p_j w_{Aj}\right) - p_i \overline{\mathbb{W}}_{\mathrm{f}}\right]\right) \\ &= \frac{p_i(1 - p_i)}{2}\left(\frac{d \ln(\overline{v}_i)}{dp} + \sum_{j=1}^{n} \frac{c_j(p_j w_{Aj} - p_i \overline{w}_j)}{\overline{\mathbb{W}}_{\mathrm{f}} p_i(1 - p_i)}\right).\end{aligned}$$

In the limit of small differences between habitats, we can obtain an approximation for change in the $i$th patch:

$$\lim_{p_i \to p} \Delta p_i = \frac{p(1-p)}{2}\left(\frac{d \ln(\overline{v}_i)}{dp} + \frac{1}{\overline{\mathbb{W}}_{\mathrm{f}}} \sum_{j=1}^{n} c_j \frac{d\overline{w}_j}{dp}\right) = \frac{p(1-p)}{2}\left(\frac{d \ln(\overline{v}_i)}{dp} + \frac{d \ln(\overline{\mathbb{W}}_{\mathrm{f}})}{dp}\right),$$

where $p = \Sigma c_i p_i$. In the same limit, the overall change in allele frequency will be

$$\lim_{p_i \to p} \Delta p = \lim_{p_i \to p} \sum_{i=1}^{n} c_i \Delta p_i = \frac{p(1-p)}{2} \sum_{i=1}^{n} \left( c_i \frac{d \ln(\overline{v}_i)}{dp} + c_i \frac{d \ln(\overline{\mathbb{W}}_{\mathrm{f}})}{dp} \right)$$

$$= \frac{p(1-p)}{2} \left( \frac{d \ln(\overline{\mathbb{V}}_G)}{dp} + \frac{d \ln(\overline{\mathbb{W}}_{\mathrm{f}})}{dp} \right),$$

where $\overline{\mathbb{V}}_G$ is the weighted geometric mean of male fitness across the set of patches: $\overline{\mathbb{V}}_G = \prod_{i=1}^{n} \overline{v}_i^{c_i}$. The final expression is used in equation (7) in the main text.

*Model 3B: Male-Limited Dispersal*

Under male-limited dispersal, we again allow for arbitrary initial frequencies of the alleles in each patch. Let $p_i$ be the frequency of allele $A$ in patch $i$. After selection in the patch, the frequencies in females and males (respectively) will be

$$p_{i\mathrm{f}} = \frac{p_i w_{Ai}}{\overline{w}_i}$$

and

$$p_{i\mathrm{m}} = \frac{p_i v_{Ai}}{\overline{w}_i}$$

(as before), where $\overline{w}_i = w_{Bi} + p_i(w_{Ai} - w_{Bi})$ and $\overline{v}_i = v_{Bi} + p_i(v_{Ai} - v_{Bi})$.

During migration, the number of males emerging from a given patch is proportional to the degree of local adaption of males that were born in that patch (the hard selection assumption). The overall allele frequency among dispersing females will be

$$p_{\mathrm{m}} = \sum_{i=1}^{n} \frac{c_i \overline{v}_i}{\overline{\mathbb{W}}_{\mathrm{m}}} \frac{p_i v_{Ai}}{\overline{v}_i}.$$

This formulation of the model assumes that the number of offspring produced by each patch is limited by density regulation set by the local environment. Each subpopulation will then reach a stable size at the beginning of each generation as long as the subpopulations are self-sustaining (i.e., the absolute fitness of each is greater than one).

After random mating between females and males of a patch, the frequency in offspring of the next generation will be

$$p_i' = \frac{1}{2}(p_{i\mathrm{f}} + p_{\mathrm{m}}) = \frac{1}{2} \left( \frac{p_i w_{Ai}}{\overline{w}_i} + \sum_{j=1}^{n} \frac{c_j \overline{v}_j}{\overline{\mathbb{W}}_{\mathrm{m}}} \frac{p_j v_{Aj}}{\overline{v}_j} \right).$$

The allele frequency change in the $i$th patch is

$$p_i' = \frac{1}{2} \left( \frac{p_i(w_{Ai} - \overline{w}_i)}{\overline{w}_i} + \frac{1}{\overline{\mathbb{W}}_{\mathrm{m}}} \left[ \left( \sum_{i=1}^{n} c_j \overline{v}_j \frac{p_j v_{Aj}}{\overline{v}_j} \right) - p_i \overline{\mathbb{W}}_{\mathrm{m}} \right] \right)$$

$$= \frac{p_i(1-p_i)}{2} \left( \frac{d \ln(\overline{w}_i)}{dp} + \sum_{i=1}^{n} \frac{c_j(p_j v_{Aj} - p_i \overline{v}_j)}{\overline{\mathbb{W}}_{\mathrm{m}} p_i(1-p_i)} \right),$$

which is a mirror image (with sexes reversed) of the general equation for the female-limited dispersal model.

## Evolutionary Invasion and Polymorphism in the Two-Patch Version of the Model

We focus on the case of female-biased dispersal (males do not disperse) here, which can be extrapolated to the case of male-biased dispersal (with reversed predictions for the two sexes). When there are two equally abundant habitat patch types ($c_1 = c_2$), the recursion equations simplify to

$$p_1' = \frac{1}{2}\left(\frac{p_1 v_{A1}}{v_{B1} + p_1(v_{A1} - v_{B1})} + \frac{p_1 w_{A1} + p_2 w_{A2}}{2\overline{\mathbb{W}}_f}\right),$$

$$p_2' = \frac{1}{2}\left(\frac{p_2 v_{A2}}{v_{B2} + p_2(v_{A2} - v_{B2})} + \frac{p_1 w_{A1} + p_2 w_{A2}}{2\overline{\mathbb{W}}_f}\right),$$

where

$$\overline{\mathbb{W}}_f = \frac{1}{2}[w_{B1} + p_1(w_{A1} - w_{B1}) + w_{B2} + p_2(w_{A2} - w_{B2})].$$

The recursion can be used to calculate a Jacobian matrix for each boundary equilibrium ($p = 0$ and $p = 1$, respectively):

$$J_0 = \begin{pmatrix} \left.\frac{\partial p_1'}{\partial p_1}\right|_{p_i=0} & \left.\frac{\partial p_1'}{\partial p_2}\right|_{p_i=0} \\ \left.\frac{\partial p_2'}{\partial p_1}\right|_{p_i=0} & \left.\frac{\partial p_2'}{\partial p_1}\right|_{p_i=0} \end{pmatrix} = \begin{pmatrix} \frac{1}{2}\left(\frac{v_{A1}}{v_{B1}} + \frac{w_{A1}}{w_{B1} + w_{B2}}\right) & \frac{1}{2}\left(\frac{w_{A2}}{w_{B1} + w_{B2}}\right) \\ \frac{1}{2}\left(\frac{w_{A1}}{w_{B1} + w_{B2}}\right) & \frac{1}{2}\left(\frac{v_{A2}}{v_{B2}} + \frac{w_{A2}}{w_{B1} + w_{B2}}\right) \end{pmatrix}$$

and

$$J_1 = \begin{pmatrix} \left.\frac{\partial p_1'}{\partial p_1}\right|_{p_i=1} & \left.\frac{\partial p_1'}{\partial p_2}\right|_{p_i=1} \\ \left.\frac{\partial p_2'}{\partial p_1}\right|_{p_i=1} & \left.\frac{\partial p_2'}{\partial p_1}\right|_{p_i=1} \end{pmatrix} = \begin{pmatrix} \frac{1}{2}\left(\frac{v_{B1}}{v_{A1}} + \frac{w_{B1}}{w_{A1} + w_{A2}}\right) & \frac{1}{2}\left(\frac{w_{B2}}{w_{A1} + w_{A2}}\right) \\ \frac{1}{2}\left(\frac{w_{B1}}{w_{A1} + w_{A2}}\right) & \frac{1}{2}\left(\frac{v_{B2}}{v_{A2}} + \frac{w_{B2}}{w_{A1} + w_{A2}}\right) \end{pmatrix}.$$

Leading eigenvalues can be obtained for each by solving for the largest $\lambda$ in

$$\det(J - \lambda I) = \det\begin{pmatrix} a - \lambda & b \\ c & d - \lambda \end{pmatrix} = 0,$$

where $a$, $b$, $c$, and $d$ are appropriate expressions from $J_0$ or $J_1$. Since $a$, $b$, $c$, and $d$ are positive, the leading eigenvalue will take the form

$$\lambda_L = \frac{(a + d) + \sqrt{(a + d)^2 - 4(ad - bc)}}{2}.$$

*Conditions for Sex-Limited Polymorphism*

When selection acts only in females ($v_{ij} = 1$), balancing selection requires that both of the following conditions are met:

$$1 < \frac{w_{A1} + w_{A2}}{w_{B1} + w_{B2}}$$

and

$$1 < \frac{w_{B1} + w_{B2}}{w_{A1} + w_{A2}},$$

which cannot simultaneously be true. Thus, female-limited selection cannot maintain polymorphism.

When selection is male limited ($v_{ij} = 1$), balancing selection requires that both of the following conditions are met:

$$\lambda_0 = \frac{1 + (v_{A1}/v_{B1}) + (v_{A2}/v_{B2}) + \sqrt{1 + [(v_{A1}/v_{B1}) - (v_{A2}/v_{B2})]^2}}{4} > 1$$

and

$$\lambda_1 = \frac{1 + (v_{B1}/v_{A1}) + (v_{B2}/v_{A2}) + \sqrt{1 + [(v_{B1}/v_{A1}) - (v_{B2}/v_{A2})]^2}}{4} > 1.$$

Substituting the parameterization from table C1, the condition for polymorphism simplifies to

$$\frac{t_2}{1 + 3t_2} < t_1 < \frac{t_2}{1 - 3t_2}.$$

With this inequality, we can calculate the fraction of parameter space leading to sex-limited polymorphism in the nondispersing sex as a function of the upper limit of intensity of selection ($a = \max(t_1, t_2)$):

$$F_{\text{poly}} = 1 - \frac{2}{a^2}\int_0^a \frac{t\,dt}{1 + 3t} = 1 - \frac{6a - 2\ln(1 + 3a)}{9a^2}.$$

For the entire parameter space ($a = 1$), this reduces to

$$F_{\text{poly}} = \frac{1}{3} + \frac{4\ln(2)}{9} \approx 0.64.$$

 DOI: 10.1086/700720

# Appendix E from T. Connallon et al., "Evolutionary Consequences of Sex-Specific Selection in Variable Environments: Four Simple Models Reveal Diverse Evolutionary Outcomes" (Am. Nat., vol. 193, no. 1, p. 000)

## Model 4: Temporally Heterogeneous Selection

### Evolutionary Dynamics

In each generation, the life cycle is birth and selection, followed by random mating, reproduction, and death in a single homogenous population (i.e., there is no population subdivision). We assume that the population size is temporally stable and sufficiently large that genetic drift can be ignored. As mentioned in the main text, some of our predictions may not hold in alternative scenarios in which population demography also fluctuates over time (see Lande 2007, 2008). Future models could focus on scenarios of fluctuating sex-specific selection with strong demographic feedbacks.

Genotypic fitness values within an arbitrary generation $t$ are denoted $w_{A,t}$ and $w_{B,t}$ for females and $v_{A,t}$ and $v_{B,t}$ for males. With $p_t$ representing the frequency of the $A$ allele at birth in generation $t$, the frequency in adult females and males (respectively) will be

$$p_{\mathrm{f},t} = \frac{p_t w_{A,t}}{p_t w_{A,t} + q_t w_{B,t}}$$

and

$$p_{\mathrm{m},t} = \frac{p_t v_{A,t}}{p_t v_{A,t} + q_t v_{B,t}}.$$

The frequency at birth in generation $t + 1$ becomes

$$p_{t+1} = \frac{p_{\mathrm{f},t} + p_{\mathrm{m},t}}{2} = p_t\left(\frac{w_{A,t}(p_t v_{A,t} + q_t v_{B,t}) + v_{A,t}(p_t w_{A,t} + q_t w_{B,t})}{2(p_t w_{A,t} + q_t w_{B,t})(p_t v_{A,t} + q_t v_{B,t})}\right).$$

### Stability of Boundary Equilbria

To evaluate conditions for invasion from each of the boundary conditions ($p = 0$ or $p = 1$), we follow the approach of prior models of adaptation in temporally variable environments (Dempster 1955; Felsenstein 1976; Charlesworth and Charlesworth 2010, pp. 72–76). To evaluate conditions for invasion of $B$ in a population nearly fixed for $A$ ($p \to 1$), we first reexpress allele frequency recursions as the ratio of $A$ to $B$ frequencies. Letting $u_t = q_t/p_t$, we have

$$u_{t+1} = \frac{q_{t+1}}{p_{t+1}} = u_t \frac{p_t(v_{A,t}w_{B,t} + w_{A,t}v_{B,t}) + q_t(v_{B,t}w_{B,t} + w_{B,t}v_{B,t})}{p_t(v_{A,t}w_{A,t} + w_{A,t}v_{A,t}) + q_t(w_{A,t}v_{B,t} + w_{B,t}v_{A,t})}.$$

With $B$ rare in the population ($q \approx 0$), the recursion simplifies to

$$\frac{u_{t+1}}{u_t} \approx \frac{1}{2}\left(\frac{w_{B,t}}{w_{A,t}} + \frac{v_{B,t}}{v_{A,t}}\right),$$

and it follows that

$$\frac{u_t}{u_0} \approx \prod_{t=0}^{t-1} \frac{1}{2}\left(\frac{w_{B,t}}{w_{A,t}} + \frac{v_{B,t}}{v_{A,t}}\right).$$

In the limit of large $t$, the condition for evolutionary invasion of a rare $B$ allele is

$$1 < \prod_{t=0}^{t-1} \frac{1}{2}\left(\frac{w_{B,i}}{w_{A,i}} + \frac{v_{B,i}}{v_{A,i}}\right) \Leftrightarrow 0 < \mathrm{E}\left[\ln\left(\frac{w_{B,i}}{2w_{A,i}} + \frac{v_{B,i}}{2v_{A,i}}\right)\right].$$

To evaluate the other boundary ($p \to 0$), we redefine the ratio as $u_t = p_t/q_t$, which leads to the recursion

$$u_{t+1} = \frac{p_{t+1}}{q_{t+1}} = u_t \frac{p_t(v_{A,t}w_{A,t} + w_{A,t}v_{A,t}) + q_t(w_{A,t}v_{B,t} + w_{B,t}v_{A,t})}{p_t(v_{A,t}w_{B,t} + w_{A,t}v_{B,t}) + q_t(v_{B,t}w_{B,t} + w_{B,t}v_{B,t})}.$$

With $A$ rare in the population ($p \approx 0$), the recursion simplifies to

$$\frac{u_{t+1}}{u_t} \approx \frac{1}{2}\left(\frac{w_{A,t}}{w_{B,t}} + \frac{v_{A,t}}{v_{B,t}}\right),$$

and it follows that

$$\frac{u_t}{u_0} \approx \prod_{t=0}^{t-1} \frac{1}{2}\left(\frac{w_{A,t}}{w_{B,t}} + \frac{v_{A,t}}{v_{B,t}}\right).$$

In the limit of large $t$, the condition for evolutionary invasion of a rare $A$ allele is

$$1 < \prod_{t=0}^{t-1} \frac{1}{2}\left(\frac{w_{A,i}}{w_{B,i}} + \frac{v_{A,i}}{v_{B,i}}\right) \Leftrightarrow 0 < \mathrm{E}\left[\ln\left(\frac{w_{A,i}}{2w_{B,i}} + \frac{v_{A,i}}{2v_{B,i}}\right)\right].$$

## Sexually Antagonistic Alleles

With two major and equally common environment types, the condition for invasion of the $A$ allele becomes

$$1 < \left(\frac{1}{2(1 - s_{\mathrm{f}1})} + \frac{1 - s_{\mathrm{m}1}}{2}\right)\left(\frac{1}{2(1 - s_{\mathrm{f}2})} + \frac{1 - s_{\mathrm{m}2}}{2}\right).$$

The condition for invasion of $B$ is

$$1 < \left(\frac{1 - s_{\mathrm{f}1}}{2} + \frac{1}{2(1 - s_{\mathrm{m}1})}\right)\left(\frac{1 - s_{\mathrm{f}2}}{2} + \frac{1}{2(1 - s_{\mathrm{m}2})}\right).$$

Polymorphism is maintained when both conditions are true.

## Sex-Limited Polymorphism

The condition for sex-limited polymorphism (in either sex) is

$$\frac{t_2}{1 + t_2/2} < t_1 < \frac{t_2}{1 - t_2/2}.$$

Therefore, the fraction of parameter space that maintains polymorphism is

$$F_{\mathrm{poly}} = 1 - \frac{4}{a^2}\int_0^a \frac{t\,dt}{2 + t} = \frac{8\ln[1 + (a/2)] - a(4 - a)}{a^2},$$

which for the full parameter space ($a = 1$) evaluates to $8\ln(3/2) - 3 \sim 0.24$.