Effects of selfing on the evolution of sexual and asexual reproduction

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Abstract

Selfing has multiple effects on the evolution of sex through both direct and indirect selection. Using population genetic models, this study investigates the effects of sprorophytic and gametophytic selfing on the evolution of a modifier of sex. Results show that although indirect selection due to segregation and selection against genome-wide deleterious mutations can favor sexual reproduction under gametophytic selfing, it greatly inhibits the evolution of both asexual and sexual reproduction under spropophytic selfing. Both sporophytic and gametophytic selfing also reduce the fitness of sexually reproduced offspring relative to asexually reproduced offspring, which can cause direct selection against sexual reproduction. Selfing also weakens selection against sex caused by the transmission disadvantage and mate limitation. However, under sporophytic selfing, indirect selection due to segregation is much stronger than other types of selection, but is comparable to other types of selection under gametophytic selection. The results offer novel insights on the evolution of diverse reproductive modes in plant populations, and suggest that the evolution of the rate of sexual reproduction may precede the evolution of selfing in angiosperms and gymnosperms.

Introduction

The evolution of sexual reproduction has attracted long-lasting interest in evolutionary biology because sexual reproduction, although prevalent, bear several costs compared to asexual reproduction (reviewed in Lehtonen et al. 2012). One of the major costs of sex is the “cost of males”. In separate-sex populations, females allocate resources to form males (or the male function in hermaphrodites) which contribute few resources to the offspring zygote, and searching for and choosing mates require extra resources and energy of females (Smith and Maynard-Smith 1978, Lehtonen et al. 2012, Meirmans et al. 2012). Such costs of sex have been observed in various taxa (Michaels and Bazzaz 1986, Innes et al. 2000, Kumpulainen et al. 2004, Wolinska and Lively 2008). Another cost of sex is the “cost of meiosis” (Williams 1975), which happens because asexual reproduction is uniparental, so that an allele that increases asexual reproduction but keeps the male function can enjoy a transmission advantage by

fertilizing female gametes of sexual individuals. It should be noted that the cost of males can apply to both individual and group selection and may occur in both hermaphroditic and separate-sex populations. In contrast, the cost of meiosis only applies to individual selection and occurs in hermaphrodites because in separate-sex populations, asexual females do not have the male function to fertilize sexual females.

Despite the many-fold costs of sex, sexual reproduction is common in eukaryotes (Bell 1982). To explain the paradox, many studies have tried to identify the indirect selective advantages of sex. Sexual reproduction differs from asexual reproduction by two genetic characteristics. First, sexual reproduction allows the possibility of recombination, and previous studies show that modifiers that increase the recombination rate can evolve under the background of deleterious mutations in the genome (Charlesworth et al. 1979, Roze and Lenormand 2005, Keightley and Otto 2006). Second, sexual reproduction causes segregation, which changes genotype frequencies at a locus. Specifically, when deleterious mutations are not too recessive, the mean fitness mainly depends on the frequency of heterozygotes. If homozygotes are averagely less fit than heterozygotes, under asexual reproduction, there will be more heterozygotes than that expected under Hardy-Weinberg equilibrium (Chasnov 2000, Agrawal and Chasnov 2001). As a result, sex can be indirectly favored through segregation, which breaks this association and increases the mean fitness (Otto 2003).

Nevertheless, most of these studies on the evolution of sex assume random mating, while the costs and benefits of sex can be greatly influenced by mating systems. This is particularly pertinent to plant populations, which often adopt a mixed strategy of outcrossing, selfing and asexual reproduction (vegetative reproduction or apomixis). In fact, apomictic plants are usually related to self-incompatible and obligate outcrossers (Asker and Jerling 1992, Hörandl 2010, Hojsgaard and Hörandl 2019), suggesting that selfing may inhibit the evolution of asexual reproduction. However, before discussing the multiple effects of selfing on the evolution of sex, it is helpful to review two types of selfing. As the life cycle of plants consists of an alternation of generations between haploid gametophytes and diploid

sporophytes, correspondingly, there are two types of selfing: gametophytic selfing and sporophytic selfing. Gametophytic selfing is that two gametes from the same hermaphroditic gametophyte form a sporophyte, which mainly happens in homosporous mosses and ferns (Soltis and Soltis 1992). Sporophytic selfing is that two gametes from different gametophytes produced from the same sporophyte form a sporophyte, which happens in most gymnosperms and angiosperms, as well as some groups of animals (Jarne and Charlesworth 1993). Sporophytes from gametophytic selfing are always homozygous, but this is not true for sporophytic selfing.

Selfing can have multiple effects on the evolution of sexual reproduction. Selfing, as uniparental reproduction, can reduce the transmission advantage of asexual reproduction (Charlesworth 1980). Also, selfing can favor the evolution of recombination under certain types of epistasis between deleterious mutations (Charlesworth et al. 1979, Roze and Lenormand 2005), which may lead to the evolution of sex, given that an increased level of sex entails an increased level of recombination. However, the effects of selfing at other aspects are less clear. First, selfing may affect the direction and strength of indirect selection on modifiers of sex caused by selection on deleterious or adaptive alleles. Otto (2003) shows that gametophytic selfing tend to promote the invasion of modifiers that increase sexual reproduction through segregation. However, the effects of sporophytic selfing are less understood, although Uyenoyama and Bengtsson (1989) find that sporophytic selfing can promote the evolution of sex when mutations are completely lethal. Second, deleterious mutations in the genome can create differences in of asexually reproduced and sexually reproduced offspring. Selfing, by increasing homozygosity, affects the relative fitness of asexually and sexually reproduced offspring (Muirhead and Lande 1997), which will affect the direct selection on sexual reproduction, but this effect remains unclear.

In this study, I investigate the effects of selfing on evolution of sex through: 1) indirect selection caused by segregation and selection on deleterious mutations or adaptive allele, 2) direct selection caused by deleterious mutations due to fitness differences between asexually reproduced, selfed and outcrossed

offspring, 3) selection caused by the transmission disadvantage and mate limitation. The results show that unlike gametophytic selfing, sporophytic selfing greatly inhibits the evolution of both sexual and asexual reproduction through indirect selection. Both sporophytic and gametophytic selfing tend to impose direct selection against sex since they lower the relative fitness of sexually reproduced. However, under sporophytic selfing, indirect selection is much stronger than direct selection, while is of comparable strength to direct selection under gametophytic selfing. The results suggest that selfing may be crucial for maintaining mixed reproductive strategies in plants. Given the transition from outcrossing to selfing being frequent in angiosperms, most evolutionary changes of the rate of sexual reproduction may happen when populations are outcrossing.

Model

I consider a diploid population with an infinitely large population size so that genetic drift is ignored. Biological meanings of key symbols used in the model are summarized in Table 1. I consider a locus that modifies the proportion of sexual reproduction **M**, and a fitness locus **A**, with a recombination rate r between them. The modifier locus has two alleles M and m, and the rate of sexual reproduction of the genotype MM, Mm and mm is σ_0, $\sigma_1 = \sigma_0 + h_\sigma \Delta\sigma$, $\sigma_2 = \sigma_0 + \Delta\sigma$, respectively. Within sexual reproduction, the selfing rates for sporophytic and gametophytic selfing are denoted by α and β, respectively. The fitness locus has two alleles A and a, where A is the wild-type allele. The selection coefficient and dominance of allele a are denoted by s and h, and I consider both cases when allele a is a deleterious allele or an adaptive allele. When a is deleterious, the mutation rate from A to a is μ, and I assume there is no backward mutation from a to A since a is rare. I denote the genotype frequency by x_{ij}, where $i \leq j$ and $i, j = 1,2,3,4$ (1 for MA, 2 for Ma, 3 for mA, and 4 for ma).

Recursion equations under gametophytic selfing are described by the previous study (Otto 2003), so here I present recursion equations under sporophytic selfing. Let x'_{ij} be the genotype frequency after selection

at the fitness locus as $x'_{11} = x_{11}/\overline{W}$, where $\overline{W}$ is the mean fitness. The genotype frequencies after mutation and selection, denoted by x''_{ij}, are

$$x''_{11} = (1-\mu^2)x'_{11}, \tag{1a}$$

$$x''_{12} = 2\mu(1-\mu)x'_{11} + (1-\mu)x'_{12}, \tag{1b}$$

and the frequency of other genotypes can be expressed in a similar way.

Reproduction happens after selection and mutation at the fitness locus, and I denote the genotype frequency among asexually reproduced, selfed and outcrossed offspring by $x_{ij}^{(a)}$, $x_{ij}^{(s)}$ and $x_{ij}^{(t)}$, respectively. The genotype frequency among asexually reproduced offspring is merely $x_{ij}^{(a)} = x''_{ij}$. The genotype frequency among selfed offspring can be calculated as

$$\overline{\sigma}x_{11}^{(s)} = \sigma_0\left(x''_{11} + \frac{x''_{12}}{4}\right) + \sigma_1\left(\frac{x''_{13}}{4} + \frac{x''_{14}(1-r)^2}{4} + \frac{x''_{23}r^2}{4}\right) + \sigma_2\frac{x''_{34}}{4}, \tag{2a}$$

$$\overline{\sigma}x_{12}^{(s)} = \sigma_0\frac{x''_{12}}{2} + \sigma_1\left(\frac{x''_{14}r(1-r)}{2} + \frac{x''_{23}r(1-r)}{2}\right), \tag{2b}$$

where $\overline{\sigma}$ is the average rate of sexual reproduction, given by

$$\overline{\sigma} = \sigma_0(x''_{11} + x''_{12} + x''_{22}) + \sigma_1(x''_{13} + x''_{14} + x''_{23} + x''_{24}) + \sigma_2(x''_{33} + x''_{34} + x''_{44}). \tag{3}$$

The frequency of other genotypes $x_{ij}^{(s)}$ can be obtained in a similar way.

For outcrossed offspring, I denote the haplotype frequency among female gametes (i.e., ovules) and male gametes (i.e., pollen) by y_i and z_i, respectively. Under gametophytic selfing, y_i and z_i are the same because both female and male gametes are produced from the same hermaphroditic gametophyte. In contrast, under sporophytic selfing, female gametes and male gametes are respectively produced from female and male gametophytes, so y_i and z_i may differ. Specifically, y_i and z_i are respectively given by

$$y_1 = \frac{1}{\overline{\sigma}}\left[\sigma_0\left(x''_{11} + \frac{x''_{12}}{2}\right) + \sigma_1\left(\frac{x''_{13}}{2} + \frac{x''_{14}(1-r)}{2} + \frac{x''_{23}r}{2}\right)\right], \tag{4a}$$

$$z_1 = \frac{1}{1-c(1-\overline{\sigma})}\left[\left(1-c(1-\sigma_0)\right)\left(x''_{11}+\frac{x''_{12}}{2}\right)+\left(1-c(1-\sigma_1)\right)\left(\frac{x''_{13}}{2}+\frac{x''_{14}(1-r)}{2}+\frac{x''_{23}r}{2}\right)\right]. \quad (4b)$$

In equation (4b), the parameter c is the relative reduction of the male gamete production due to asexual reproduction. When $c < 1$, asexual reproduction enjoys a transmission advantage because it can produce extra male gametes. When $c = 1$, asexual reproduction will reduce both the female and male gamete production, so the sex modifier is neutral when there is no fitness locus. The frequency of other haplotypes can be obtained similarly. The genotype frequency among outcrossed offspring is

$$x_{ij}^{(t)} = \begin{cases} y_i z_j, i = j \\ y_i z_j + y_j z_i, i < j \end{cases}. \quad (5)$$

Due to deleterious mutations in the genome, the fitness of asexually reproduced, outcrossed and selfed offspring will differ. I denote the fitness of asexually reproduced offspring and selfed offspring relative to outcrossed offspring by $w_a = 1 - \delta_a$ and $w_s = 1 - \delta_s$, respectively, and δ_a and δ_s are the fitness reduction relative to outcrossed offspring. The genotype frequency in the next generation after viability selection is

$$\tilde{x}_{ij} = \frac{(1-\delta_a)x_{ij}^{(a)} + (1-\delta_s)\alpha x_{ij}^{(s)} + (1-\alpha)x_{ij}^{(t)}}{1-\delta_a(1-\overline{\sigma}) - \delta_s\alpha\overline{\sigma}}, \quad (6)$$

where the denominator is the mean fitness.

Results

The result section consists of four subsections. The first subsection investigates the effects of selfing on the invasibility of a modifier that either increases or decreases sexual reproduction through indirect selection caused by selection at the fitness locus. This is done by assuming that the fitness of asexually reproduced offspring, outcrossed and selfed offspring to be the same. The second subsection investigates the effects of selfing on direct selection on sexual reproduction due to deleterious mutations, by analyzing how the rate of sexual reproduction and the sporophytic or gametophytic selfing rate affect the relative fitness of asexually reproduced and selfed offspring. The third subsection compares the strength of

indirect and direct selection on a modifier of sex caused by deleterious mutations under different rates of sexual reproduction and selfing. The fourth section investigates the effects of selfing on the strength of selection on a modifier of sex caused by the transmission disadvantage and mate limitation.

Indirect selection

For indirect selection on a modifier of sex caused by selection at the fitness locus, I consider two cases. In the first case, the allele a at the fitness locus is a deleterious mutation at mutation-selection balance, and the modifier locus is fixed with allele M. I investigate whether a modifier allele m can invade or not by calculating the leading eigenvalue λ of the Jacobian matrix of equation (6), and invasion is possible when $|\lambda| > 1$. In the second case, the allele a is an adaptive allele which sweeps from a low initial frequency to high frequency, and I investigate whether the frequency of the modifier m will increase or not.

Sporophytic selfing

When allele a is deleterious, results show that sporophytic selfing can greatly inhibit invasion of both sexual and asexual reproduction. When the modifier increases sexual reproduction, as Fig. 1(a) illustrates, a slight amount of sporophytic selfing ($\alpha = 0.001$) can greatly increase the parameter space where sex is favored compared to that under completely outcrossing. However, as the selfing rate becomes higher, although still at a low level ($\alpha = 0.05, 0.08$), the parameter space where sex is favored quickly shrinks. In fact, for the parameter values used in Fig. 1(a), the modifier cannot invade in the whole parameter space when $\alpha \geq 0.1$. A similar pattern is found for a modifier that decreases sexual reproduction, as Fig. 1(b) shows. Therefore, under sporophytic selfing, there is a large parameter space where neither sexual nor asexual reproduction can invade.

Further investigations for the effects of other key factors show that under sporophytic selfing, a lower recombination rate slightly between the modifier locus and the fitness locus increases the parameter space

for sexual reproduction to invade, while decreases the parameter space for asexual reproduction to invade (Fig. S1). Invasion of both sexual and asexual reproduction generally becomes less likely when the background rate of sexual reproduction σ_0 is higher (Fig. S2), or when the invading modifier is recessive (Fig. S3), unless the selfing rate is extremely low (e.g., $\alpha = 0.001$).

When allele a is a sweeping adaptive allele, sporophytic selfing tends to inhibit the evolution of sex but promote the evolution of asexual reproduction. When the modifier increases sexual reproduction, as Fig. 1(c) illustrates, the parameter space where the allele frequency of the modifier increases shrinks as the sporophytic selfing rate becomes higher. In contrast, a higher sporophytic selfing rate enlarges the parameter space where a modifier promoting asexual reproduction increases in frequency, as shown in Fig. 1(d). However, in Figs. 1(c) and 1(d), there exists small parameter space where neither sexual nor asexual reproduction is favored, which mainly occurs when the selection coefficient is small.

Gametophytic selfing

Otto (2003) found that gametophytic selfing can greatly increase the parameter space for sex to be favored when the allele a is a deleterious allele, but tends to decrease the parameter space for a modifier of sex to increase in frequency when allele a is adaptive allele. Further investigations in this study find that the parameter space where sexual reproduction is favored is nearly complementary to the parameter space where asexual reproduction is favored. Therefore, unlike results under sporophytic selfing that there is parameter space where neither sexual nor asexual reproduction is favored, under gametophytic selfing, if sexual reproduction is favored, it means asexual reproductive is selected against, and *vice versa*.

Direct selection caused by deleterious mutations

Deleterious mutations can cause offspring produced by different ways to have different fitness, which imposes direct selection on the modifier of sex. In the previous subsection, to eliminate the effects of selfing on direct selection of a modifier of sex, it is assumed that asexually and sexually reproduced

offspring have identical fitness. To see how selfing can affect direct selection on sex, here I derive the relative fitness of asexually reproduced and selfed offspring compared to outcrossed offspring. To do this, I first obtain the genotypic frequencies at the fitness locus under mutation-selection balance based on equation (6). The average fitness of asexually reproduced offspring w_a is equal to the average fitness of the population $\overline{W}$. The average fitness of selfed offspring is

$$w_s = x_{11}^{(s)} + x_{13}^{(s)} + x_{33}^{(s)} + (1-hs)\left(x_{12}^{(s)} + x_{14}^{(s)} + x_{23}^{(s)} + x_{34}^{(s)}\right) + (1-s)\left(x_{22}^{(s)} + x_{44}^{(s)}\right). \qquad (7)$$

The average fitness of outcrossed offspring w_t can be calculated in a similar way.

Here I only present results under sprorophytic selfing, but the results also qualitatively hold under gametophytic selfing (see Appendix). Under sporophytic selfing, the relative fitness reductions of asexually reproduced offspring and selfed offspring caused by deleterious mutations at a single locus are respectively

$$d_a = \frac{F(1-2h)\sigma}{(1+F-\sigma)hs + (F+h-hF)\sigma}\mu + o(\mu), \qquad (8a)$$

$$d_s = \frac{1}{2}\frac{(1-2h)\big(s(1+F-\sigma) + \sigma(1+F)\big)}{(1+F-\sigma)hs + (F+h-hF)\sigma}\mu + o(\mu), \qquad (8b)$$

where $F = \frac{\alpha}{2-\alpha}$ is the inbreeding coefficient when the sporophytic selfing rate is α. For genome-wide deleterious mutations, when there are L fitness loci with identical effects, by ignoring linkage between loci, the relative fitness reduction of asexually reproduced and selfed offspring is approximately $\delta_a = 1 - e^{-d_a L}$ and $\delta_s = 1 - e^{-d_s L}$, respectively (see equations (A3a) and (A3b) in Appendix). Equations (8a) and (8b) show that when deleterious mutations are recessive ($h < 0.5$), both asexually reproduced and selfed offspring are less fit than outcrossed offspring. Appendix further shows that when deleterious mutations are recessive, as the rate of sexual reproduction and the selfing rate increase, the relative fitness of asexually reproduced offspring is lower, while the relative fitness of selfed offspring is higher. The latter effect is consistent with previous findings from simulations (Muirhead and Lande 1997). However, it can be shown that the fitness reduction of selfed offspring relative to asexually reproduced offspring is

$$\frac{d_s}{d_a} = \frac{1}{\alpha} + s\left(\frac{1-\sigma}{\alpha\sigma} + \frac{1}{2}\right) \geq 1. \tag{9}$$

Therefore, selfed offspring are always averagely less fit than asexually reproduced offspring.

To see how the selfing rate affects the relative fitness of asexually and sexually reproduced offspring, note that the mean fitness of sexually reproduced offspring is $w_{sex} = \alpha(1-d_s) + 1 - \alpha = 1 - \alpha d_s$. Therefore, the fitness reduction of sexually reproduced offspring relative to asexually reproduced offspring is

$$\frac{1-w_{sex}}{1-w_{asex}} = \begin{cases} 1, & \alpha = 0 \\ 1 + s\left(\frac{\alpha}{2} + \frac{1}{\sigma} - 1\right) \geq 1, & \alpha > 0 \end{cases} \tag{10}$$

Equation (10) show that sporophytic selfing causes direct selection against sexual reproduction, and when the mating system changes from completely outcrossing to sporophytic selfing, the change of the relative fitness of sexually reproduced offspring is discrete. Also, a higher sporophytic selfing rate lowers the relative fitness of sexually reproduced offspring, and thus increases direct selection against sex. However, under sporophytic selfing, the fitness of sexually reproduced offspring relative to asexually reproduced offspring does not depend on the gametophytic selfing rate (see equation (A8)).

Comparison of the strength of direct and indirect selection on a modifier of sex

Genetic analyses show that there are two major categories of deleterious mutations: small-effect, partially dominant mutations and large-effect, highly recessive lethal mutations (Mukai et al. 1972, Eyre-Walker and Keightley 2007, Charlesworth and Willis 2009). Based on previous estimations (Simmons and Crow 1979), I set $s = 0.05, h = 0.2$ for small-effect deleterious mutations, and $s = 0.8, h = 0.02$ for large-effect lethal mutations. By locating the parameter combinations in the parameter space of Figs. 1(a) and 1(b), it can be seen that for both categories of mutations, unless the selfing rate is low, sporophytic selfing tends to inhibit the evolution of both sexual and asexual reproduction. Therefore, to determine whether sexual reproduction is relatively favored over asexual reproduction or not, it is necessary to compare the

selective strength against sexual and asexual reproduction under sporophytic selfing. It is also interesting to compare the relative strength of indirect and direct selection. This may be especially important for evolution of sex under gametophytic selfing, since gametophytic selfing tends to promote the evolution of sex through indirect selection but inhibit the evolution of sex through direct selection.

Following Otto (2003), I define the strength of selection on a modifier allele as $\lambda - 1$, where λ is the leading eigenvalue of the Jacobian matrix. When the allele is rare and selection is weak, $\lambda - 1$ measures the scaled rate of allele frequency change as

$$\lambda - 1 \approx \frac{p_m' - p_m}{p_m(1 - p_m)}. \tag{11}$$

For strength of indirect selection, since selection caused by a single fitness locus is weak, when there are L identical fitness loci, the genome-wide indirect selection is approximately $L(\lambda - 1)$. The strength of direct selection can be obtained by setting $s = 0$, and letting the fitness of asexually reproduced and selfed offspring to be $W_a = 1 - \delta_a$ and $W_s = 1 - \delta_s$ (see equation (8) and the text below).

Sporophytic selfing

Under sporophytic selfing, sexual reproduction is generally disfavored by both indirect and direct selection, but results show that indirect selection is much stronger than direct selection. For indirect selection, Fig. 2(a) shows that indirect selection caused by small-effect deleterious mutations is often on the order of 10^{-2}, and becomes the strongest when the rate of sexual reproduction is high and the sporophytic selfing rate is intermediate ($\alpha \approx 0.75$). In contrast, Fig. 2(d) shows that the strength of indirect selection caused by large-effect mutations is usually on the order of 10^{-3}, and increases as the selfing rate becomes higher. Compared with Figs. 2(a) and 2(d), Figs. 2(b) and 2(e) show that the strength of direct selection on sex is very weak relative to indirect selection. Although sex is favored by direct selection when the rate of sexual reproduction is high and the sporophytic selfing rate is low, the strength is on the order of 10^{-5}, which is too weak to outcompete indirect selection against sex.

Since sporophytic selfing also inhibits the invasion of asexual reproduction, I compare the strength of selection on sexual and asexual reproduction. Generally, indirect selection against asexual reproduction is slightly weaker than indirect selection against sexual reproduction. Specifically, for indirect selection caused by small-effect mutations, Fig. 2(c) shows that asexual reproduction is more strongly selected than sexual reproduction when either the rate of sexual reproduction or the selfing rate is low (grey area in Fig. 2(c)). In contrast, for indirect selection caused by large-effect mutations, Fig. 2(f) shows that asexual reproduction is favored when the selfing rate is low (refer to the isoline 0 in Fig. 2(f)). However, since indirect caused by small-effect mutations are stronger, we expect overall, asexual reproduction is more strongly selected against than sexual reproduction when the sporophytic selfing rate is low. Nevertheless, as long as the rate of sexual reproduction and the selfing rate are not low, selection against asexual reproduction is slightly weaker than that for sexual reproduction.

Gametophytic selfing

Under gametophytic selfing, as Fig. 3 shows, the strength of indirect and direct selection is generally comparable, and the strength of indirect selection is much smaller than that under sporophytic selfing. Fig. 3(a) shows the strength of indirect selection caused by small-effect deleterious mutations, unlike results under sporophytic selfing, there are two regions where sex is favored, either when the rate of sexual reproduction is not low and the gametophytic selfing rate is not high (region I), or when the rate of sexual reproduction is low and the gametophytic selfing rate is high (region II). Moreover, in region I, sex is also favored by direct selection, as Fig. 3(b) shows. However, in region II, sex is disfavored by direct selection (see Fig. 3(b)), but overall, sex is still favored because indirect selection is stronger than direct selection in region II.

For selection caused by large-effect, highly recessive mutations, sex is unfavored through both indirect and direct selection in a large parameter space. Although Fig. 3(c) shows that when the gametophytic

selfing rate is high, sex is favored by indirect selection, overall, sex is still disfavored because the strength of direct selection against sex is strong when the gametophytic selfing rate becomes high (see Fig. 3(d)). Therefore, under gametophytic selfing, sex is favored by selection of small-effect deleterious mutations, but unfavored by selection of large-effect lethal mutations, and the selective strength of the two opposing effects are comparable. Therefore, whether gametophytic selfing promotes the evolution of sex should depend on the relative genomic mutation rate of the two categories of mutations. Moreover, Fig. 3 does not show the strength of selection on modifiers that increase asexual reproduction, because under gametophytic selfing, when sexual reproduction is disfavored, asexual reproduction is always favored with a comparable selective strength.

Effects of selfing on selection due to the transmission disadvantage and mate limitation

Although results in the previous subsection suggest that sporophytic selfing tends to inhibit the evolution of sexual reproduction through indirect selection, selfing can reduce the strength of selection against sexual reproduction by two ways. First, selfing lowers the transmission advantage of asexual reproduction. Second, selfing is an alternative method (as opposed to asexual reproduction) of providing reproductive assurance when mates are limited. The strength of selection on a modifier of sex caused by the two effects can be obtained by setting $s = 0$ and $w_a = w_s = \frac{1}{1-m}$ in the recursion equation, where m is the strength of mate limitation. The strength of selection of a small-effect modifier allele ($\Delta\sigma \ll 1$) is $s_e(h_\sigma + F - h_\sigma F)$, where F is the inbreeding coefficient ($F = \frac{\alpha}{2-\alpha}$ under sporophytic selfing, and $F = \beta$ under gametophytic selfing), and s_e is the effective selection coefficient, given by

$$s_e = -\frac{(1-\alpha)\big(1 - c + m - mc(1-2\sigma_0)\big)}{2\big(1 - c(1-\sigma_0)\big)(1 - m(1-\alpha)\sigma_0)}\Delta\sigma. \tag{12}$$

Equation (12) holds for both sporophytic and gametophytic selfing, but under gametophytic selfing, asexual reproduction may not have a transmission advantage (i.e., $c = 1$). In the denominator of equation (12), the first term captures selection due to the transmission disadvantage, and the second term accounts

for selection caused by reproductive assurance. In the nominator, the term $mc(1-2\sigma_0)$ captures the interaction between mate limitation and transmission disadvantage.

Based on equation (12), Fig. 4 shows that generally, the strength of selection against a modifier of sex with $\Delta\sigma = 0.01$ is on the order of 10^{-3}, even when both the transmission disadvantage and mate limitation are strong (see Fig. 4(b)). A higher selfing rate generally reduces the strength of selection, but under strong mate limitation ($m = 0.8$ in Fig. 4), selection against sexual reproduction is strongest under a low selfing rate rather than under completely outcrossing. This is because although selfing reduces the effective selection coefficient, it increases the strength of selection by increasing homozygosity (thus a higher inbreeding coefficient F).

Therefore, under sporophytic selfing, indirect selection on a modifier of sex caused by genome-wide deleterious mutations, which is on the order of 10^{-2}, is much stronger than selection caused by the transmission disadvantage and mate limitation. In contrast, under gametophytic selfing, selection caused by the transmission disadvantage and mate limitation is stronger than indirect and direct selection caused by genome-wide deleterious mutations, which is on the order of 10^{-4}.

Discussion

Many plants perform mixed reproductive strategies of selfing, outcrossing and asexual reproduction. This study investigates the effects of sporophytic and gametophytic selfing on the evolution of sex through three types of selection: 1) indirect selection caused by segregation due to selection on deleterious mutations or adaptive allele, 2) direct selection caused by deleterious mutations due to the fitness difference between sexually and asexually reproduced offspring, 3) selection caused by the transmission disadvantage and mate limitation. Key results are summarized in Table 2.

Sporophytic selfing (the union of two gametes from the same diploid sporophyte), which mainly happens in gymnosperm and angiosperms, greatly decreases the parameter space for either sexual or asexual reproduction to invade through indirect selection, although indirect selection against asexual reproduction is slightly weaker than that against sexual reproduction. Sporophytic selfing also causes direct selection against sexual reproduction by lowering the fitness of sexually reproduced offspring relative to asexually reproduced offspring, but it can weaken selection against sex caused by the transmission disadvantage and mate limitation. Generally, the strength of indirect selection caused by genome-wide deleterious mutations is much stronger than the other two types of selection. Nevertheless, the difference in the strength of indirect selection against asexual versus sexual reproduction under sporophytic selfing is comparable to the reduced strength of selection against sex caused by the transmission disadvantage and mate limitation. Therefore, although sporophytic selfing imposes strong selection against both sexual and asexual reproduction, it may lower the selective disadvantage of sexual reproduction relative to asexual reproduction.

Gametophytic selfing (the union of gametes from the same haploid gametophytic), which happens in homosporous ferns, generally promotes indirect selection favoring sexual reproduction (Otto 2003). The current study further confirms that unlike that under sporophytic selfing, when sexual reproduction is favored, asexual reproduction is always disfavored, and *vice versa*. Also, the strength of indirect selection under gametophytic selfing is much weaker than that under sporophytic selfing. Gametophytic selfing can cause direct selection against sex, with a strength comparable to indirect selection. However, overall, gametophytic selfing tends to favor the evolution of sexual reproduction.

Previous studies show that selfing may promote indirect selection for a modifier that increases the recombination rate under some conditions of epistasis (Roze and Lenormand 2005). Therefore, selfing may also affect the indirect selection on sex, and here I give an estimation of the effects of selfing on the strength of indirect selection on sex due to changes of the recombination rate. A recent study shows that

the strength of indirect selection on a recombination modifier increasing map length by ΔR is approximately $1.8U^2\Delta R/R^3N_e$, where U is the genomic mutation rate of deleterious mutations, R is the map length, and N_e is the effective population size (Roze 2021). Since a modifier of sex increasing the rate of sexual reproduction by $\Delta\sigma$ increases the map length by $R\Delta\sigma$, the strength of indirect selection on it is $1.8U^2\Delta\sigma/R^2N_e$. Since the map length in most organisms is on the order of $1 \sim 10$ (Otto and Payseur 2019), and the genomic mutation rate is usually on the order of $0.1 \sim 1$, the maximum strength of indirect selection is about $0.018/N_e$, given $\Delta\sigma = 0.01$. Therefore, unless the effective population size is very small, we should expect that indirect selection on sex through recombination is much weaker than indirect section caused by segregation (refer to the strength of indirect selection in Fig. 2).

The inhibition of the evolution of either sexual or asexual reproduction by sporophytic selfing may explain why reproductive modes are diverse in plants. In completely outcrossing populations, indirect selection due to segregation is quite weak, so that the evolution of sexual or asexual reproduction are relatively easy, and completely sexual or asexual reproduction is possible. However, evolution from outcrossing to selfing can be frequent (Barret 2008, Goldberg et al. 2010). Therefore, once populations evolve from outcrossing to selfing, evolutionary changes of the rate of sex are greatly inhibited, so that the populations are likely to maintain mixed-mating (i.e., asexual reproduction, selfing and outcrossing). This indicates most of evolutionary changes of the rate of sex should be completed when populations are still outcrossing. This prediction can be tested by carrying out phylogenetic estimation and comparison of the transition rates between asexual and sexual reproduction in self-incompatible and self-compatible lineages.

Inhibition of the evolution of sex by sporophytic selfing may also provide an alternative explanation for the pattern that evolution of apomixis is often from predominantly outcrossing populations (Horandl 2010). This pattern is often explained by that selfing is an alternative method (as opposed to asexual

reproduction) of providing reproductive assurance under mate limitation, but the current results suggest that it may be due to inhibition of the evolution of asexual reproduction through indirect selection under sporophytic selfing. Moreover, pseudogamous apomictic populations are often associated with a breakdown of self-incompatiblity (Horandl 2010, 2019), and the current results suggest the evolution of apomixis may precede the breakdown of self-incompatiblity.

The current finding that sporophytic selfing greatly inhibits the evolution of sexual reproduction is contrary to the previous results in Uyenoyama and Bengtsson (1989), which assumes a completely lethal allele ($s = 1$) and shows that a higher selfing rate can increase the parameter space for sexual reproduction to invade. This difference is mainly because a lethal deleterious mutation is a special case, and in the two models, the invasion of a modifier of sex happens at different stages of the life cycle. In Uyenomaya and Bengtsson (1989), mutation of a modifier of sex is assumed to happen after viability selection, at which stage there are only two genotypes at the fitness locus, since homozygotes of the lethal allele are eliminated. In contrast, in the current model, the mutation of a modifier of sex happens after the mutation at the fitness locus, so all three genotypes at the fitness locus are present. This difference can qualitatively affect the invasibility of a modifier of sex. However, when the deleterious mutation is not completely lethal, the two assumptions do not make much difference.

In all, this study shows that indirect selection due to segregation and selection against genome-wide deleterious mutations tends to be a dominant selective force that inhibits evolution of both asexual and sexual reproduction under spropophytic selfing, but it will favor sexual reproduction under gametophytic selfing. This suggests that evolution of the rate of sexual reproduction in saprophytic selfing populations may be due to group selection instead of individual selection.

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Tables

Table 1. The biological meaning of key symbols.

Symbol	Biological meaning
σ	Rate of sexual reproduction
α, β	Sporophytic and gametophytic selfing rate within sexual reproduction
σ_0	Background rate of sexual reproduction
h_σ	Dominance of a sex modifier
$\Delta\sigma$	Effects of a sex modifier on changing the rate of sexual reproduction
s, h	Selection coefficient and dominance of the allele at a fitness locus
μ	Mutation rate at a fitness locus
r	Recombination rate between the sex modifier and fitness loci
U	Genomic mutation rate of deleterious mutations
m	Strength of mate limitation
d_a, δ_a	Fitness reduction of asexually reproduced offspring relative to outcrossed offspring caused by a single locus, and all deleterious mutations in the genome
d_s, δ_s	Fitness reduction of selfed offspring relative to outcrossed offspring caused by a single locus, and all deleterious mutations in the genome

Table 2. A summary of the effects of sporophytic and gametophytic selfing on selection on a sex modifier. The strength of selection is for a modifier with the effect $\Delta\sigma = \pm 0.01$, and is estimated under the parameter values $s = 0.05, h = 0.2, U = 0.4$ for small-effect deleterious mutations, and $s = 0.8, h = 0.02, U = 0.05$ for large-effect mutations. The notations "+" and "−" mean whether selfing causes selection for or against the modifier, and "$+/-$" means both can happen, depending on the rate of sexual reproduction and selfing.

Types of selection	**Sporophytic selfing**	**Gametophytic selfing**
Indirect selection caused by deleterious mutations	Sexual reproduction: − Asexual reproduction: −	Sexual reproduction: + Asexual reproduction: −
Indirect selection caused by adaptive sweep	Sexual reproduction: − Asexual reproduction: +	Sexual reproduction: − Asexual reproduction: +
Direct selection caused by small-effect deleterious mutations	Sexual reproduction: +/− Asexual reproduction: +/− A higher selfing rate increases selection against sex	
Direct selection caused by large-effect mutations	Sexual reproduction: − Asexual reproduction: + A higher selfing rate increases selection against sex	
Selection caused by the transmission disadvantage and mate limitation	Selfing reduces the strength of selection against sexual reproduction.	
Strength of selection on a modifier that increases sexual reproduction	**Indirect selection:** small-effect mutations: $-10^{-2} \sim -10^{-1}$ large-effect mutations: $-10^{-3} \sim -10^{-2}$ **Direct selection:** small-effect mutations: $-10^{-3} \sim 10^{-5}$ large-effect mutations: $-10^{-3} \sim 0$ **Selection by the transmission disadvantage and mate limitation**: 10^{-3}	**Indirect selection:** small-effect mutations: $-10^{-4} \sim 10^{-3}$ large-effect mutations: $-10^{-4} \sim 10^{-4}$ **Direct selection:** small-effect mutations: $-10^{-4} \sim 10^{-5}$ large-effect mutations: $-10^{-3} \sim 0$ **Selection by the transmission disadvantage and mate limitation**: 10^{-3}

Figures

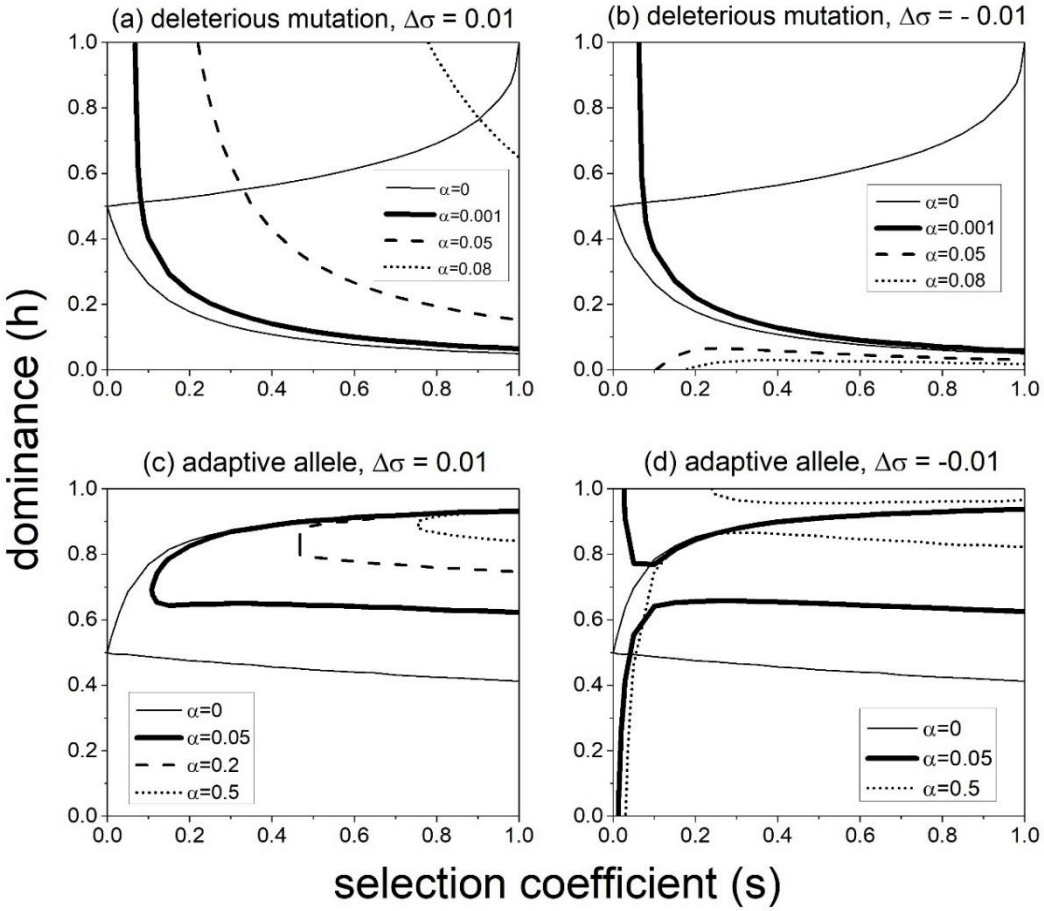

Figure 1. Panels (a) and (b) show the invasibility of a modifier of sex when allele a is deleterious. Panels (c) and (d) show whether the frequency of a modifier of sex increases or decreases when allele a is adaptive. The modifier increases sexual reproduction in panels (a) and (c), while decreases sexual reproduction in panels (b) and (d). In panel (a), invasion happens in the parameter space between the two solid lines for $\alpha = 0$, and for $\alpha > 0$, invasion happens in the parameter space above each line. In panel (b), for $\alpha = 0$, invasion happens in the parameter space above the upper line and below the lower line, and for $\alpha > 0$, invasion happens in the space below each line. In panel (c), for each value of α, the allele frequency of the modifier increases in the parameter space between the two lines. In panel (d), for each

value of α, the allele frequency of the modifier increases in the parameter space above the upper line and below the lower line. Other parameters used are $\sigma_0 = 0.1, h_\sigma = 0.5, r = 0.5, \mu = 10^{-5}$.

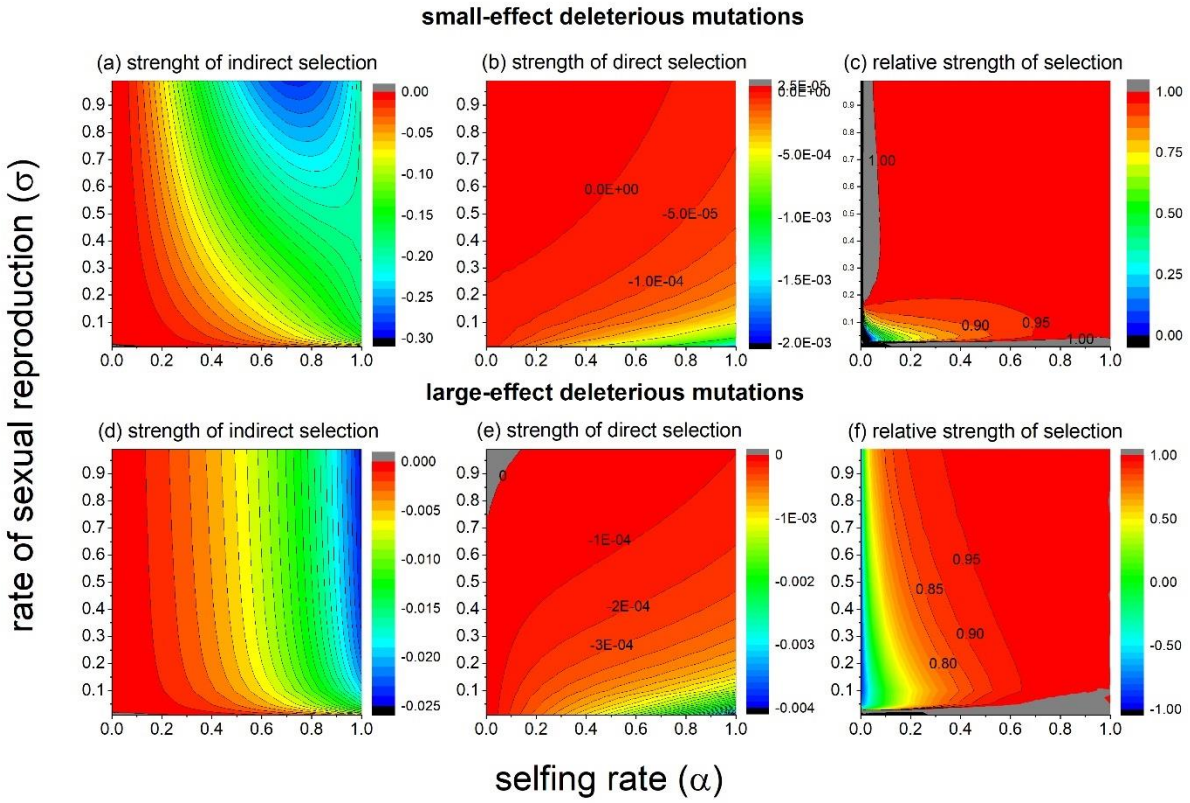

Figure 2. Strength of indirect and direct selection on a modifier of sex caused by small-effect (upper panels) and large-effect (bottom panels) deleterious mutations under sporophytic selfing. Panels (a), (b) and (d), (e) show the strength of selection on a modifier that increases sexual reproduction with $\Delta\sigma = 0.01$, $h_\sigma = 0.5$. Panels (c) and (f) show the relative strength of indirect selection against a modifier that increases asexual reproduction ($\Delta\sigma = -0.01$) compared to that for a modifier with $\Delta\sigma = 0.01$. In panel (f), the relative strength is negative when the selfing rate is small because asexual reproduction is favored while sexual reproduction is disfavored. Other parameters are $s = 0.05, h = 0.2, U = 0.4$ for small-effect deleterious mutations, and $s = 0.8, h = 0.02, U = 0.05$ for large-effect deleterious mutations. The value of the genomic mutation rate is chosen based on previous estimations (Mukai et al. 1972, Simmons and Crow 1977, Klekowski and Godfrey 1989).

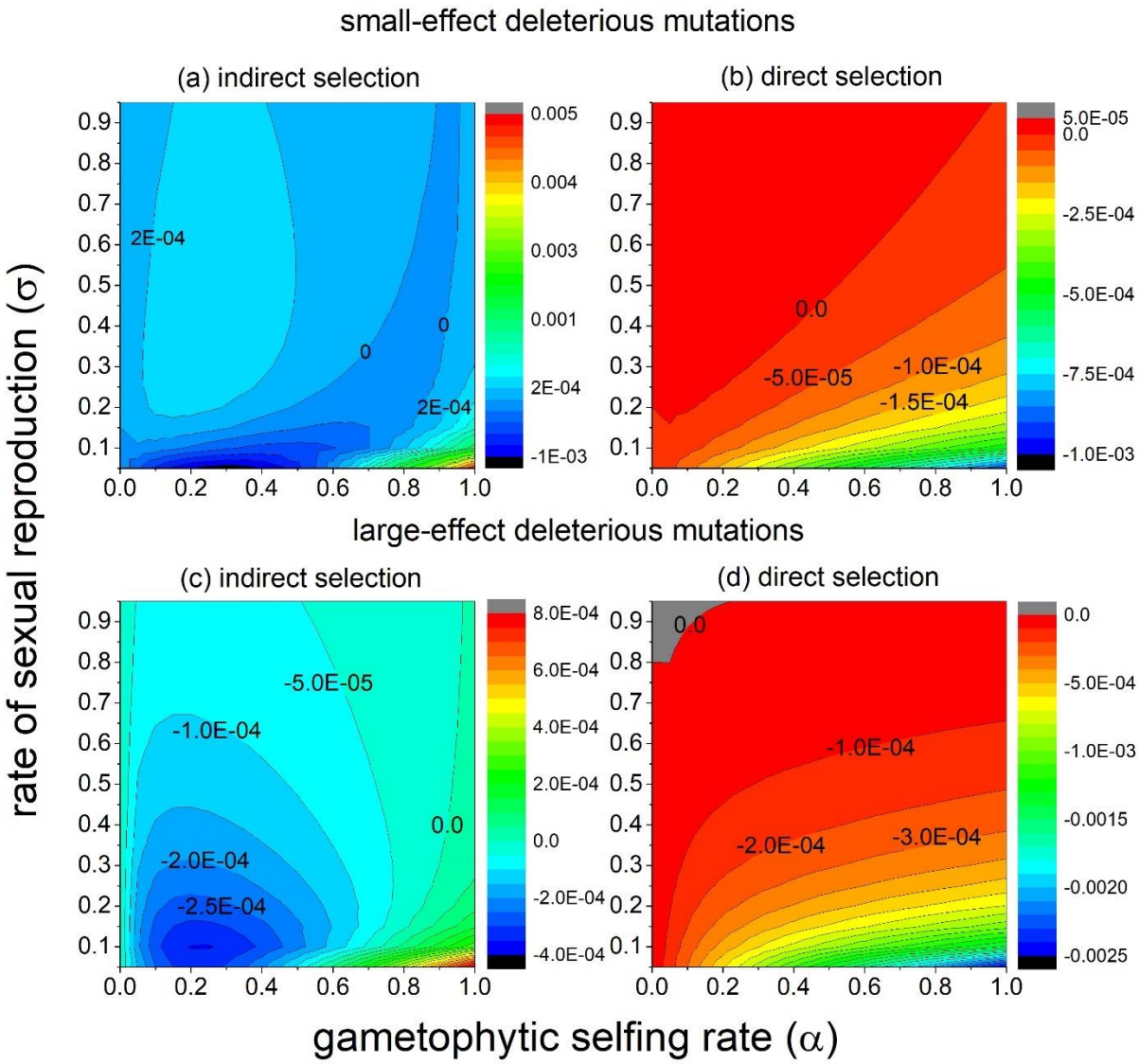

Figure 3. Strength of indirect and direct selection on a modifier of sex with $\Delta\sigma = 0.01$ caused by small-effect (upper panels) and large-effect (bottom panels) deleterious mutations under gametophytic selfing. Parameters used are the same as those in Fig. 2.

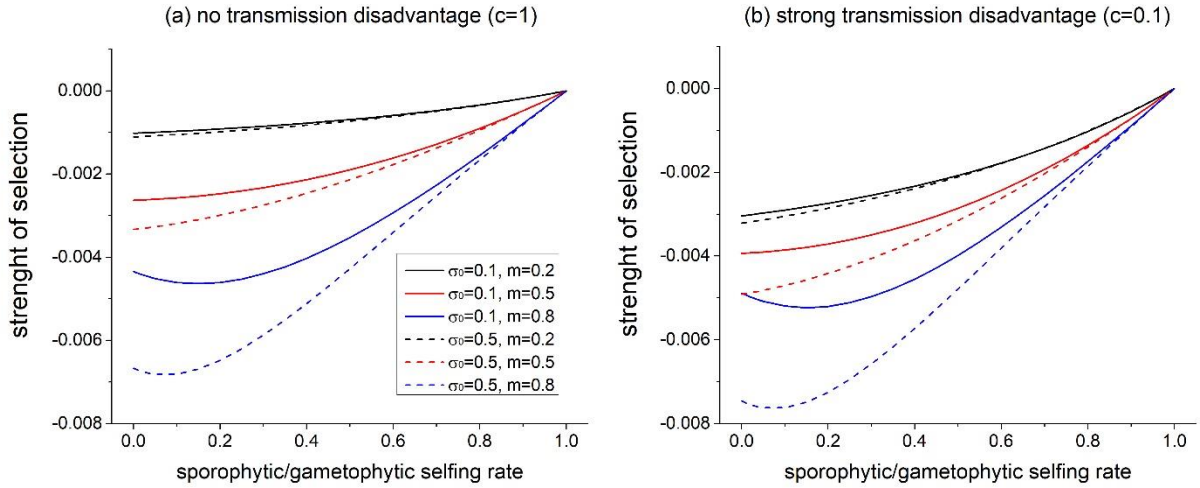

Figure 4. Effects of the selfing rate on the strength of selection against a modifier of sex ($\Delta\sigma = 0.01$, $h_\sigma = 0.5$) caused by the transmission disadvantage and mate limitation based on equation (12).

Appendix

Here I show how the rate of sexual reproduction and the selfing rate affect the relative fitness of asexually reproduced and selfed offspring under sporophytic and gametophytic selfing.

Sporophytic selfing

for the relative fitness of asexually reproduced offspring, we have

$$\frac{\partial d_a}{\partial \sigma} = \frac{F(1+F)h(1-2h)s}{\big((1+F-\sigma)hs + (F+h-hF)\sigma\big)^2}\mu, \tag{A1a}$$

$$\frac{\partial d_a}{\partial F} = \frac{h(1-2h)(s(1-\sigma)+\sigma)\sigma}{\big((1+F-\sigma)hs + (F+h-hF)\sigma\big)^2}\mu. \tag{A1b}$$

Since deleterious mutations are often recessive ($h < 0.5$), both equations are greater than 0. Therefore, the relative fitness of asexually reproduced offspring is lower when the rate of sexual reproduction and the selfing rate are higher. Similarly, for the relative fitness of selfed offspring, we have

$$\frac{\partial d_s}{\partial \sigma} = -\frac{F(1+F)(1-2h)^2 s}{2\big((1+F-\sigma)hs + (F+h-hF)\sigma\big)^2}\mu < 0, \tag{A2a}$$

$$\frac{\partial d_s}{\partial F} = -\frac{(1-2h)^2(s(1-\sigma)+\sigma)\sigma}{2\big((1+F-\sigma)hs + (F+h-hF)\sigma\big)^2}\mu < 0. \tag{A2b}$$

Therefore, the relative fitness of selfed offspring is higher when the rate of sexual reproduction and the selfing rate are higher.

If there are L fitness loci with identical selection and dominance coefficient, the genomic mutation rate is $U = 2\mu L$. The relative fitness reduction of asexually reproduced offspring σ_a, and that of selfed offspring δ_s, are respectively

$$\delta_a = 1 - \exp[-d_a L] = 1 - \exp\left[\frac{F(1-2h)\sigma}{(1+F-\sigma)hs + (F+h-hF)\sigma}\frac{U}{2}\right], \tag{A3a}$$

$$\delta_s = 1 - \exp[-d_s L] = 1 - \exp\left[-\frac{1}{2}\frac{(1-2h)\big(s(1+F-\sigma)+\sigma(1+F)\big)}{(1+F-\sigma)hs + (F+h-hF)\sigma}\frac{U}{2}\right]. \tag{A3b}$$

Gametophytic selfing

Under Gametophytic selfing, the relative fitness reduction caused by is

$$d_a = \frac{(1-2h)\beta\sigma}{hs + \sigma\beta + (1-s-\beta)h\sigma}\mu, \tag{A4a}$$

$$d_s = \frac{(1-2h)(s(1-\sigma)+\sigma)}{hs + \sigma\beta + (1-s-\beta)h\sigma}\mu. \tag{A4b}$$

The fitness reduction of selfed offspring relative to asexually reproduced offspring is

$$\frac{d_s}{d_a} = \frac{1}{\beta}\left(1 + \frac{1-\sigma}{\sigma}s\right) \geq 1. \tag{A5}$$

Therefore, selfed offspring have lower fitness than asexually reproduced offspring.

Taking the first order derivative of d_a and d_s for σ and β, and $h < 0.5$, we have

$$\frac{\partial d_a}{\partial \sigma} = \frac{\beta s h(1-2h)}{(\beta\sigma + h(s+\sigma-(s+\beta)\sigma))^2}\mu > 0, \tag{A6a}$$

$$\frac{\partial d_a}{\partial \beta} = \frac{h(1-2h)(s(1-\sigma)+\sigma)\sigma}{(\beta\sigma + h(s+\sigma-(s+\beta)\sigma))^2}\mu > 0, \tag{A6b}$$

and

$$\frac{\partial d_s}{\partial \sigma} = -\frac{(1-h)(1-2h)s\alpha}{\left(\beta\sigma + h(s+\sigma-(s+\beta)\sigma)\right)^2}\mu < 0, \tag{A7a}$$

$$\frac{\partial d_s}{\partial \beta} = -\frac{(1-h)(1-2h)(s(1-\sigma)+\sigma)\sigma}{\left(\beta\sigma + h(s+\sigma-(s+\alpha)\sigma)\right)^2}\mu < 0. \tag{A7b}$$

Therefore, as the rate of sexual reproduction and the gametophytic selfing rate increases, the relative fitness of asexually reproduced offspring decreases, while the relative fitness of selfed offspring increases. Also, it can be shown that

$$\frac{1-w_{sex}}{1-w_{asex}} = \begin{cases} 1, & \beta = 0 \\ 1 + s\left(\frac{1}{\sigma} - 1\right) \geq 1, & \beta > 0 \end{cases}. \tag{A8}$$

The relative fitness reduction of asexually reproduced and selfed offspring are respectively

$$\delta_a = 1 - \exp\left[-\frac{(1-2h)\beta\sigma}{hs + \sigma\beta + (1-s-\beta)h\sigma}\frac{U}{2}\right], \tag{A9a}$$

$$\delta_s = 1 - \exp\left[-\frac{(1-2h)(s(1-\sigma)+\sigma)}{hs + \sigma\beta + (1-s-\beta)h\sigma}\frac{U}{2}\right]. \tag{A9b}$$

Supplementary Figures

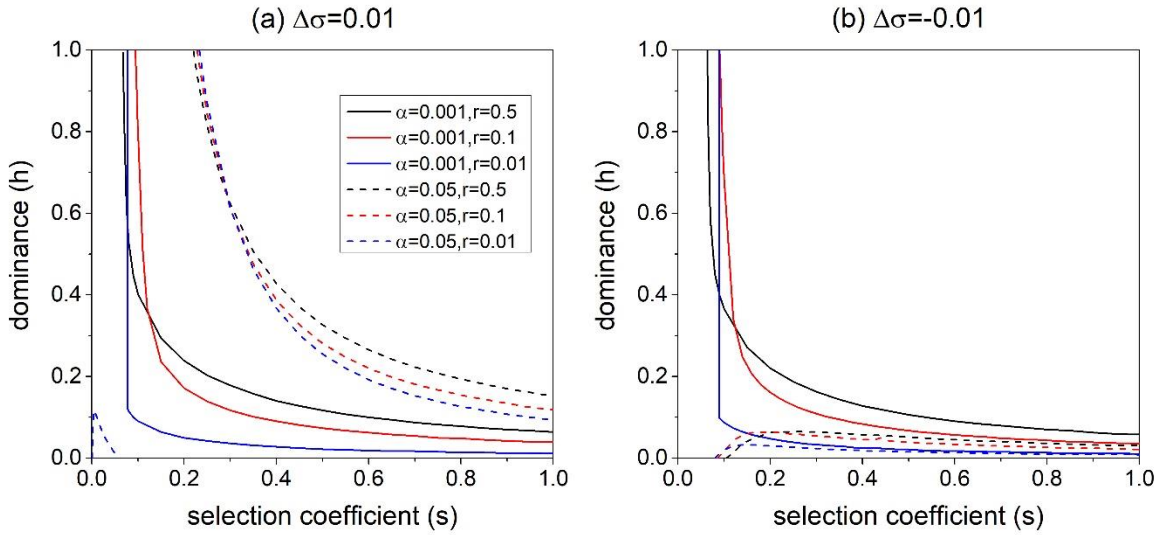

Figure S1. Effects of the recombination rate r on the evolution of sex when allele a at the fitness locus is deleterious. In panel (a), the modifier increases the rate of sexual reproduction, and will invade in the parameter space above each line, except for the blue dashed line at the bottom left corner, for which invasion happens below the line. In panel (b), the modifier increases the rate of asexual reproduction, and invasion happens in the parameter space below each line. Other parameters are $\sigma_0 = 0.1, h_\sigma = 0.5$.

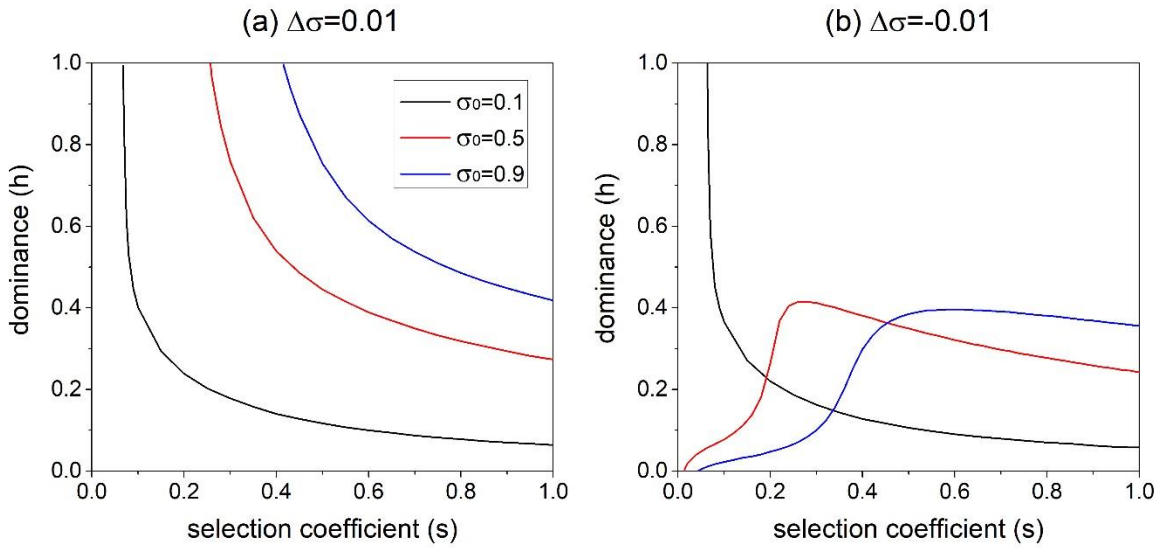

Figure S2. Effects of the background rate of sexual reproduction σ_0 on the evolution of sex when allele a at the fitness locus is deleterious and the sporophytic selfing rate is $\alpha = 0.001$. In panel (a), a modifier that promotes sexual reproduction invades in the parameter space above each line. In panel (b), a modifier that promotes asexual reproduction invades in the parameter space below each line. Actually, when the sporophytic selfing rate is $\alpha = 0.05$, the invasion of either sexual or asexual reproduction becomes impossible at $\sigma_0 = 0.5, 0.9$ (not shown in the figure). Other parameters are $h_\sigma = 0.5, r = 0.5$.

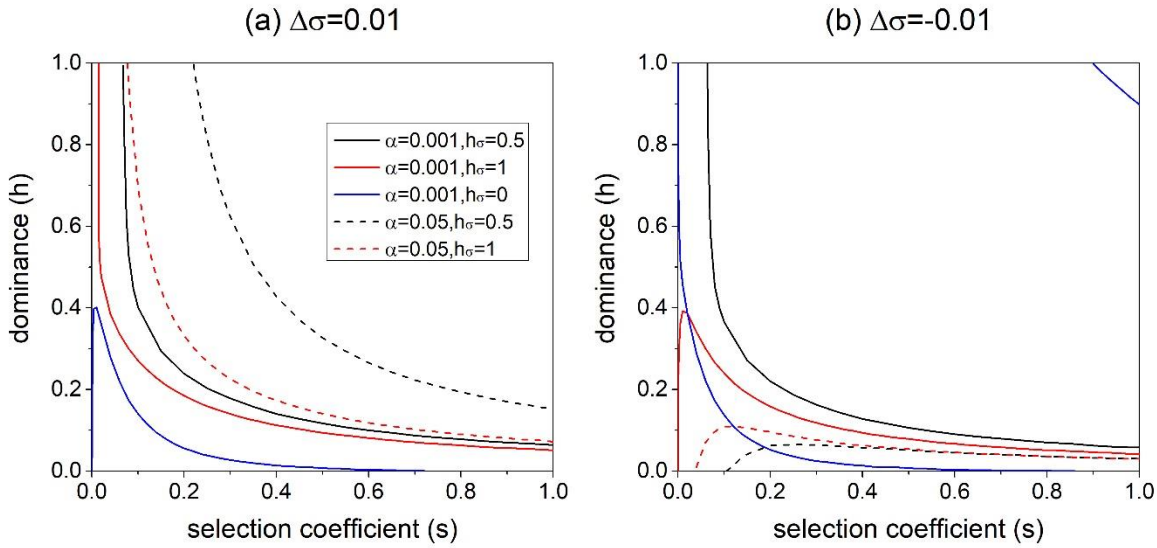

Figure S3. Effects of the dominance coefficient of a modifier (h_σ) on the evolution of sex when allele a is deleterious. In panel (a), a modifier that promotes sexual reproduction invades in the parameter space above each line except the blue line, for which invasion happens below the line. In panel (b), a modifier that promotes asexual reproduction invades in the parameter space below each line except the two blue solid lines, for which invasion happens between them. In both panels (a) and (b), invasion becomes impossible when $\alpha = 0.05, h_\sigma = 0$. Other parameters are $\sigma_0 = 0.1, r = 0.5$.