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Evolution of sexually dimorphic flower production under sexual, fertility, and viability selection

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ABSTRACT

Question: How do different patterns of selection and constraint promote the evolution of sexually dimorphic flower production?

Mathematical methods: Simulation modelling. Key sets of assumptions are applied to individuals in the population and we determine whether alleles causing sexually dimorphic flower production can spread to fixation from a sexually monomorphic state.

Key assumptions: We model the evolution of sexually dimorphic flower production starting from a sexually monomorphic dioecious state (separate male and female plants that produce the same number of flowers) under different interactions of sexual, fecundity, and viability selection. Flower number and flower size trade off. Pollinator preferences are based on total flower production per plant.

Predictions: The relationship between flower size and pollen or ovule production critically determines whether sexual dimorphism can evolve from a monomorphic state. Fecundity selection can temper and sometimes reverse the evolution of sexual dimorphism predicted through sexual selection (i.e. pollinator preferences) alone.

Keywords: dioecious, flower number, life-history trade-off, model, sexual dimorphism.

INTRODUCTION

Although most flowering plants are hermaphroditic, gender specialization has repeatedly evolved across a number of angiosperm families. Nearly 6% of all known angiosperms are dioecious, with separate male and female individuals (Renner and Ricklefs, 1995). Dioecious species necessarily exhibit differences between the sexes with respect to primary sex characteristics (e.g. anthers and ovaries), and this separation of reproductive functions is thought to have evolved as a mechanism to promote outcrossing as well as a response to sex-differential allocation to reproduction (Charlesworth and Charlesworth, 1978; Thompson and Barrett, 1981; Charnov, 1982; Thompson and Brunet, 1990; Seger and Eckhart, 1996).

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Interestingly, many dioecious species are also sexually dimorphic for secondary sexual characteristics related to reproductive, physiological, and life-history traits (Geber *et al.*, 1999), and recent theoretical work has identified several primary mechanisms by which these differences might evolve. For example, disruptive selection on homologous characters in a dioecious plant species can easily lead to the evolution of sexual dimorphism (Lande, 1980). Competition within and between the sexes for some limiting ecological resource (Slatkin, 1980, 1984; Maynard Smith, 1991), as well as differential pollinator preferences (Kiester *et al.*, 1984) or post-pollination mate choice (Stephenson and Bertin, 1983; Kiester *et al.*, 1984; Maynard Smith, 1991; Snow, 1994; Andersson and Iwasa, 1996), may also drive the evolution of sexually dimorphic traits.

A morphological trait that is often sexually dimorphic is flower number (Darwin, 1877; Bell, 1985; Delph *et al.*, 1996; Eckhart, 1999), with males typically, but not always, producing more flowers than females (Table 1). Disruptive selection on flower number between the two sex functions in hermaphrodites may promote the evolution of gender dimorphism, and subsequently sexual dimorphism (Morgan, 1992, 1994); however, selection leading to the evolution of separate sexes need not also result in sexual dimorphism. Extant secondary sexual characteristics (e.g. flower number) that exhibit sexual dimorphism may be independent of traits that were historically under disruptive selection and initiated the evolution of gender dimorphism. As such, patterns of sexual dimorphism in flowering plants could be the result of evolution following the establishment of dioecy; traits that were initially monomorphic between the sexes may evolve to become dimorphic, especially if they are sex-limited in their expression. In this theoretical study, we identify a suite of interacting factors that promote the evolution of sexually dimorphic flower production following the establishment of dioecy, and discuss our results in light of empirical data.

We take the approach promoted by Arnold (1994) in which selection can take one of three forms: sexual, fecundity, or viability. Within this framework, differences in mating success will lead to differences in realized fecundity, which then lead to differences in fitness. In other words, the dependency of realized fecundity on mating success creates a path between the number of mates (or mating success) and realized fecundity. Simply stated, a plant's fecundity will depend in part on its ability to acquire mates (sexual selection). Realized fecundity will also depend, however, on such traits as how many resources the plant allocates to ovules or pollen, so fecundity selection entails components independent of sexual selection. The last type of selection, viability, is straightforward – how long a plant lives will directly affect its fitness.

In a population of dioecious plants in which flower production is sexually monomorphic, there may be sexual selection for dimorphic flower production if the sexes are under differential selection to be attractive to pollinators (Bateman, 1948; Kiester *et al.*, 1984). Clearly, how many pollinators visit a plant and either take away or deposit pollen will affect its mating success. Although this form of selection is usually attributed to selection for greater flower production in males (Bell, 1985), it can occur in either sex.

Fecundity selection can also select for sexual dimorphism in flower production. The relationship between pollen or ovule production per flower and flower size may directly impact fitness. Pollen and ovule production per flower may be canalized or some positive function of flower size (Stanton and Preston, 1988; Kudo and Toshihiko, 1998; Delph *et al.*, 2004a), and flower number/size trade-offs have been observed (Stanton *et al.*, 1991; Sato and Yahara, 1999; Worley *et al.*, 2000; Caruso, 2004; Delph *et al.*, 2004a). Therefore, ovule or pollen production per flower might be invariable or negatively correlated with flower production. If this genetic relationship

Table 1. Dioecious species reported to have sexually monomorphic or dimorphic flower production

M = F	
Baccharis halimifolia (Asteraceae)[18]	*Aristotelia fruticosa* (Elaeocarpaceae)[12]
Dendrocacalia crepidifolia (Asteraceae)[17]	*Coprosma lucida* (Rubiaceae)[12]
Lepidum sisymbroides (Brassicaceae)[12]	*Wikstroemia pulcherrinna* (Thymeliaceae)[12]
M > F	
Acer negundo (Aceraceae)[12]	*Silene latifolia* (Caryophyllaceae)[21]
Ceratiosicyos ecklonii (Achariaceae)[12]	*Sedum rosea* (Crassulaceae)[12]
Rhus typhina (Anacardiaceae)[20]	*Ecballium elaterium* (Cucurbitaceae)[9]
Aciphylla monroi (Apiaceae)[19]	*Carex picta* (Cyperaceae)[11]
Anisotome aromatica (Apiaceae)[19]	*Lindera benzoin* (Lauraceae)[8]
Anisotome deltoidea (Apiaceae)[19]	*Wurmbea dioica* (Liliaceae)[3]
Anisotome fillifolia (Apiaceae)[19]	*Chamaelirium lutum* (Liliaceae)[12]
Anisotome flexuosa (Apiaceae)[19]	*Asparagus acutifolius* (Liliaceae)[19]
Anisotome imbricata (Apiaceae)[19]	*Phoradendron flavescens* (Loranthaceae)[12]
Gingidia decipiens (Apiaceae)[19]	*Guarea rhopulocarpa* (Meliaceae)[6]
Gingidia enysii (Apiaceae)[19]	*Morus nigra* (Moraceae)[13]
Gingidia flabellate (Apiaceae)[19]	*Myristica inspida* (Myristicaceae)[2]
Gingidia montana (Apiaceae)[19]	*Compsoneura sprucei* (Myristicaceae)[5]
Gingidia trifoliolata (Apiaceae)[19]	*Nyssa sylvatica* (Nyssaceae)[12]
Lignocarpa carnosula (Apiaceae)[19]	*Clematis gentianoides* (Ranunculaceae)[13]
Lignocarpa diversifolia (Apiaceae)[19]	*Rhamnus alaternus* (Rhamnaceae)[14]
Scandia rosaefolia (Apiaceae)[19]	*Fragaria chilioensis* (Rosaceae)[15]
Ilex montana (Aquifoliaceae)[7]	*Oemelaria cerasiformis* (Rosaceae)[1]
Ilex opaca (Aquifoliaceae)[12]	*Galium nuttellii* (Rubiaceae)[12]
Aralia nudicaulis (Araliaceae)[4]	*Siparuna grandiflora* (Siparunaceae)[22]
Silene dioica (Caryophyllaceae)[10]	
F > M	
Antennaria parviflora (Asteraceae)[12]	*Rhodiola integrifolia* (Crassulaceae)[12]
Cirsium arvense (Asteraceae)[12]	*Tetracoccus dioicus* (Euphorbiaceae)[12]
Petasites japonicus (Asteraceae)[16]	

[1]Allen (1985); [2]Armstrong and Irvine (1989); [3]Barrett (1992); [4]Barrett and Helenurm (1981), Flanagan and Moser (1985); [5]Bullock (1982); [6]Bullock *et al.* (1983); [7]Cavigelli *et al.* (1986); [8]Cipollini and Whigham (1994); [9]Costich and Meagher (1992); [10]Cox (1981); [11]Delph *et al.* (1993); [12]L.F. Delph, L.F. Galloway and M.L. Stanton (unpublished data); [13]Godley (1976); [14]Guitián (1995); [15]Hancock and Bringhurst (1980); [16]Imazu and Fujishita (1961); [17]Kato and Nagamusu (1995); [18]Krischik and Denno (1990); [19]Lloyd and Webb (1977); [20]Lovett Doust and Lovett Doust (1988); [21]Meagher (1992); [22]Nicotra (1999).

(which we refer to below as gametophyte packaging) differs between the sexes, then sexual dimorphism might evolve.

Sexual or fecundity selection for increased flower production may create a higher cost of reproduction, such as reduced growth or longevity, and lead to viability selection (Antos and Allen, 1988; Bond and Maze, 1999). This follows from life-history theory, which predicts that trade-offs are generated between allocation to reproductive functions and survivorship (Williams, 1966). A relevant study is that of Bond and Maze (1999), who investigated the effect of intrasexual variation in flower production on survivorship of males, and found that males with elaborate floral displays died at a younger age than males with more limited flower production.

In this study, we model the evolution of sexual dimorphism in flower production in a demographic framework, utilizing a life-history/ecological simulation model that considers the fate of alleles that create differences between the sexes in their flower production. We explicitly incorporate genetic associations among flower number, pollen and ovule production, and survivorship, and assess how the interplay among sexual, fecundity, and viability selection on flower number affects the evolution of sexual dimorphism.

SIMULATION OVERVIEW

To investigate the evolution of sexually dimorphic flower production, we created a simulation of a dioecious plant population where the flower production of a plant was controlled by a single locus with two alleles (A_1 and A_2). In addition to determining the probability of pollinator visitation, we let flower production per plant influence pollen or ovule production per flower, and the probability of survivorship to the next growing season. The flower production of the entire population also affected the total number of pollinators that entered the population in a given time step. After defining our assumptions, we let the population evolve a level of sexually monomorphic flower production that was stable against invasion from all other sexually monomorphic strategies (ESM). Then we tested whether a strategy that caused some level of sexually dimorphic flower production could spread to fixation against the monomorphic strategy. By iterating this process over a variety of assumption combinations and comparing the results across simulations, we identify common conditions under which particular patterns of sexual dimorphism evolved and distinguish the influence of sexual, fecundity, and viability selection on the evolution of sexually dimorphic flower production. Our program was written in C and is freely available from the authors. All simulations were run on a 1.8-GHz dual-processor Macintosh G5 with 1 GB of memory.

Our simulations started with a small number of males and females that were homozygous for the A_1 allele, which conferred the same flower production in both sexes. When introduced, the A_2 allele was always recessive and sometimes conferred some level of sex-specific flower production. We defined flower production per plant as a fraction of a dummy variable called the 'physiological maximum flower production' (PM), which was meant to represent some species constraint. Assuming finite resources, a plant that made flowers at 100% PM would have many miniscule flowers that contain no pollen or ovules, and would not survive to the next time step. Similarly, a plant that made flowers at 1% PM would have few large flowers that may contain lots of pollen or ovules, and would have a high probability of surviving to the next time step. We assumed that males and females have the same physiological maximum. Allelic effects on flower production were defined as some proportion of the physiological maximum. This simplification made associations among traits relative, rather than absolute. For example, if there were a trade-off between flower production and survivorship, the relative magnitude of this trade-off would be the same no matter if the physiological maximum were set at 5000 flowers or 500 flowers.

We used the flower production of an individual to determine its pollen or ovule production per flower and its likelihood of surviving to the next time step. To do this, we assumed flower size was a linear decreasing function of flower number. When pollen or ovule production per flower was not canalized, we assumed that pollen or ovule production was a linear increasing or saturating function of flower size. As such, pollen or ovule production per plant was maximized near 100% PM under the former condition and at

intermediate levels of flower production under the latter two conditions. This yielded three ways in which ovule production per flower could be associated with whole-plant flower production (Fig. 1). For simplicity, we chose to model pollen production per flower as the probability that pollen was picked up by a pollinator (i.e. the more pollen per flower, the greater the chance that pollen is picked up), and modelled this relationship as canalized, as an increasing function of flower size, or as a saturating function of flower size (Fig. 1).

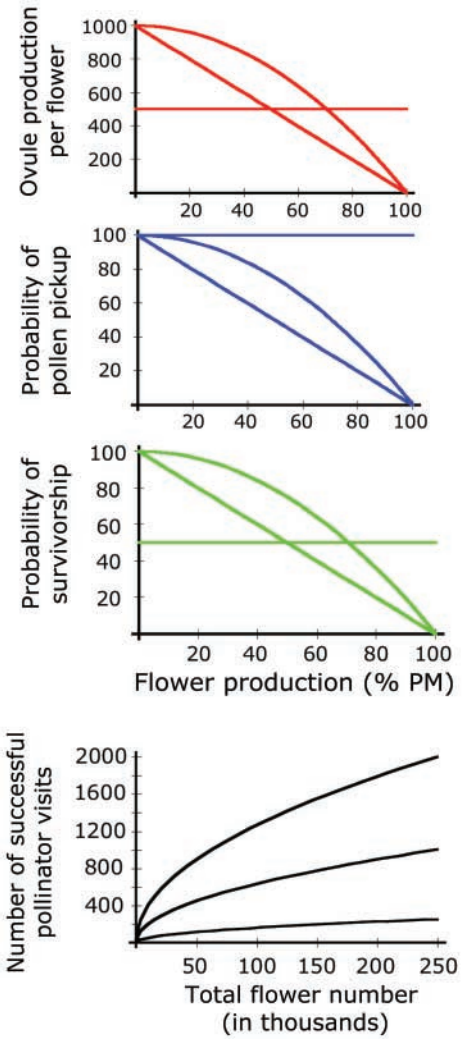

Fig. 1. Sets of assumption combinations used in our simulations. Ovule production per flower, the probability of pollen pickup, and the probability of survivorship all either traded-off with flower production in a linear or concave fashion or were fixed. The number of successful pollinator visits to the population increased with total flower number at three different levels: high, medium, and low. In combination with the assumptions concerning whether sexual selection for increased flower production occurred in both, one, or neither sex, our simulation evaluated a total of 324 different sets of assumptions.

To determine the total number of pollinator visits to the population, we randomly selected a plant and determined whether a pollinator visited that plant. We defined three ways in which the total number of pollinator visits to a population was associated with the flower production of the population (Fig. 1), and varied whether both, one, or neither sex experienced sexual selection for increased flower production relative to members of the same sex in the population. In cases where we assumed pollinator-mediated selection for increased flower production, we randomly selected a plant, selected a random number between 1 and 100, and determined whether it was less than the number of flowers that plant produced (e.g. selecting 50 would mean that a plant at 55% PM was visited, whereas a plant at 45% PM was not). When we assumed a sex did not experience selection for increased flower production, we randomly selected a plant as visited without the caveat of checking flower production. We kept a record of all plants visited by pollinators and when a visit to a female immediately followed one to a male, we defined that as a potential pollination event and assessed whether the pollinator had received pollen from that male. If so, all ovules produced by that female were fertilized by pollen from that male and the genetic identity of the seeds was determined according to standard Mendelian inheritance. Each seed had a 50% chance of being male or female. If the pollinator did not pick up pollen, no seeds were produced from that mating pair. This process was continued until either the total number of pollinator visits for the population was reached, or all females in the population were pollinated. As such, the fitness of a particular strategy was measured in terms of reproductive success (i.e. seed production of females and siring success of males).

Before moving to the next time step, we determined which adults survived and let a randomly selected subset of seeds enter the next time step as adults. Therefore, our population consisted of individuals in multiple age classes, and allowed for overlapping generations. We modelled the probability of survivorship as either fixed at 100%, or decreasing functions of flower production (Fig. 1), under the restriction that plants survived for a maximum of three time steps. The number of seeds randomly selected to enter the next time step was an inverse function of the total population size. As such, the population initially grew exponentially, and then smoothly reached an internally determined carrying capacity.

This simulation framework explicitly ties flower production to sexual, fecundity, and viability selection and allowed us to investigate how each affects the evolution of sexually dimorphic flower production alone and in combination. For example, by canalizing the relationship between flower production and pollen pickup, ovule production, and survivorship, we could assess the effects of pollinator-mediated selection for increased flower production on males, females, or both sexes. Similarly, by letting the probability of pollen pickup and ovule production covary with flower production in different ways, we were able to create a tension between sexual and fecundity selection (i.e. the likelihood of pollination continually increases with flower production, whereas the likelihood of pollen pickup and ovule production is maximized at an intermediate level of flower production). Given the probabilistic nature of our simulation, it is important to note that removing intrasexual selection for increased flower production on one or both sexes increases the amount of randomness (i.e. genetic drift) in our simulations.

METHODS

The assumptions described above yield 324 different sets of combinations (3 ovule * 3 pollen * 3 survivorship * 3 pollinator-visit number * 4 sexual selection) that were tested

individually in the following manner. To determine the evolutionarily stable level of sexually monomorphic flower production against alternative monomorphic strategies for a given set of assumptions (ESM), we first introduced an initial number of A_1A_1 males and females and let the allelic effect of the A_1 allele be 5% PM. Once this population reached carrying capacity, we introduced the recessive A_2 allele and let it cause a 5% increase in PM in both sexes when homozygous. We allowed the population to evolve until the A_2 allele spread to fixation or was eliminated. Then, we repeated this test 100 times to determine whether the A_2 allele spread to fixation most of the time. If so, we assigned that flower production level to the A_1 allele and set the genotype of all individuals in the population at A_1A_1. Then, we repeated this process in 5% PM increments (i.e. 5% vs. 10%, 10% vs. 15%, etc.) until the A_2 allele was consistently eliminated. Once the A_2 allele was consistently eliminated, we checked to ensure we reached a stable level of sexual monomorphism by letting the recessive allele cause a 5% PM reduction in flower production and verify that it was still eliminated from the population. For example, if a recessive allele causing 60% PM was eliminated when competing against 55% PM, we checked to ensure that a recessive allele causing 50% PM also failed to spread against 55% PM.

After determining the ESM for a given set of assumptions, we assessed whether an allele causing sexual dimorphism could spread against it. To do this we set the effect of the A_1 allele at the ESM, and allowed a population of A_1A_1 plants to reach carrying capacity. Then, we introduced a recessive A_2 allele that had *sex-specific* effects on flower production in units of 5% PM (e.g. males = 45% PM, females = 55% PM). We let the population evolve and determined whether the A_2 allele spread to fixation or was eliminated and then repeated this test 20 times to determine the frequency with which the allele spread. This procedure was repeated for all possible dimorphic effects of the A_2 allele. Because we tested allelic effects in units of 5% PM between 5% PM and 95% PM, a total of 342 different dimorphic effects of the A_2 allele were tested for each set of assumptions (19 male * 19 female – 19 monomorphic). This allowed us to determine whether a recessive allele that caused sexually dimorphic flower production could spread against an 'optimal' sexually monomorphic strategy (ESM). We show our results graphically and colour code the results based on the frequency with which a particular dimorphic strategy spread to fixation against the monomorphic strategy (Figs. 2B, E). By repeating this procedure for all possible combinations of assumptions, we identify relationships that consistently led to the evolution of sexual dimorphism.

The method described above assumes that a mutation causing sexually dimorphic flower production of any magnitude and direction may arise at the flower-production locus in the population. We also simulated a more realistic situation where we constrained the effects of the A_2 allele to a 5% PM deviation from the A_1 allele. Once the ESM was determined, we randomly assigned the effect of the A_2 allele on male and female flower production separately such that it was either equal to the sexually monomorphic state or +5% PM or −5% PM relative to the monomorphic state. Then, we tested this level of sexually dimorphic flower production against the monomorphic state 20 times. If the A_2 allele was eliminated an equal or majority of the time, we randomly selected a different set of values for the A_2 allele as described above. We repeated this process until either the A_2 allele spread to fixation a majority of the time, or all possible deviations from the sexually monomorphic state had been evaluated, in which case the simulation was ended. If the A_2 allele spread to fixation a majority of the time, we assigned that value of sexually dimorphic flower production to the A_1 allele and let all individuals be homozygous for A_1. Then, we randomly selected another

effect of the A_2 allele on male and female flower production as described above and continued the process until the population 'walked' from the initial monomorphic state to some stable level of sexual dimorphism, or the population had changed 100 times.

We repeated this process 100 times for each set of assumption combinations and show the results graphically (Figs. 2C, F). By comparing the results of this simulation to the first, we were able to confirm how particular sets of assumption combinations yielded different patterns of sexually dimorphic flower production. Additionally, by examining the plots we could determine the magnitude of sexually dimorphic flower production that was reached under each set of assumption combinations and assess how the population evolved to that state.

RESULTS

We ran a total of 324 simulations under different sets of assumption combinations (3 ovule * 3 pollen * 3 survivorship * 3 pollinator-visit number * 4 sexual selection) for each procedure described above. Rather than show the results of 648 simulation runs, we broadly describe how varying fecundity, sexual, and viability selection affected the evolution of sexual dimorphism, and emphasize the conditions under which M > F or F > M evolved from the monomorphic state. We present our results in figures that highlight particularly insightful comparisons between the evolutionary outcomes under alternative assumption sets. We found that both kinds of simulation runs (e.g. Fig. 2B vs. Fig. 2C) consistently yielded the same results, and therefore present simulation data from the first procedure (e.g. Fig. 2B) alone for simplicity. As seen in the figures, the evolution of sexually dimorphic flower production usually entailed an increase in flower production by one sex and a decrease in the other sex relative to the ESM.

Effects of fecundity selection (gametophyte packaging) with sexual selection on both sexes

We found that gametophyte-packaging constraints had a large influence on the direction in which sexual dimorphism evolved. Under conditions of sexual selection for increased flower production in both sexes, certain combinations of gametophyte-packaging assumptions *always* prevented alleles causing sexually dimorphic flower production from spreading, and others *always* resulted in the rapid fixation of alleles causing sexual dimorphism in one direction (M > F or F > M). Sexually monomorphic flower production was stable whenever ovule production and pollen pickup per flower were constrained in the same way – when either they were both canalized (i.e. do not vary with flower number), or both decreased in the same way with flower production (linear or concave). In contrast, when the relationship between gametophyte and flower production was canalized in one sex but not the other, alleles causing sexual dimorphism could spread to fixation. For example, when the probability of pollen pickup was canalized, but there was a linear or concave trade-off between flower production and ovule production per flower (Fig. 2A), alleles causing sexually dimorphic flower production could only spread if they caused males to make more flowers than females (Figs. 2B, C). Conversely, when ovule production per flower was canalized and there was a linear or concave trade-off between flower production and the probability of pollen pickup (Fig. 2D), alleles causing sexually dimorphic flower production could only spread if they caused females to make more flowers than males (Figs. 2E, F). In other

words, when the only way to have more pollen picked up or to make more ovules was to make more flowers (because pollen or ovule number per flower could not vary), sexual dimorphism evolved to overcome this constraint.

Interplay between fecundity and sexual selection

Not surprisingly, in the absence of fecundity selection, sexual selection for increased flower production in one sex but not the other affected the extent to which alleles causing sexual dimorphism could spread to fixation. When fecundity selection was the same for both sexes (i.e. the gametophyte-packaging assumptions were the same for both sexes), sexual selection drove the evolutionary outcome. There was a strong bias towards the evolution of M > F when there was only sexual selection for increased flower production in males, and a strong bias towards the evolution of F > M when there was only sexual selection on females (data not shown).

More interestingly, we found that sexual selection could sometimes exacerbate or reverse the evolution of sexually dimorphic flower production predicted under the effects of fecundity selection described above (Fig. 2). For example, under fecundity selection assumptions leading to the evolution of M > F (Fig. 2A), sexual selection for increased flower production in males but not females increased the parameter space under which alleles causing M > F could spread (Fig. 3A). Sexual selection allowed alleles causing more extreme M > F dimorphism to spread to fixation against the ESM. Similarly, under fecundity selection assumptions leading to the evolution of F > M (Fig. 2D), sexual selection for increased flower production in females but not males increased the parameter space under which alleles causing F > M could spread (Fig. 3B). Restricting sexual selection to one sex alone in our simulations also reduced the net strength of selection on flower number in the population. This caused an increase in the strength of genetic drift relative to selection because of the probabilistic nature with which mating occurred in our finite population. This relative increase in the strength of genetic drift allowed alleles causing sexual dimorphism opposite the direction predicted by sexual and fecundity selection to sometimes spread (e.g. sometimes a small amount of F > M could spread in Fig. 3A).

When sexual selection favoured the evolution of sexual dimorphism in a direction opposite that favoured under fecundity selection, we found that sexual dimorphism could evolve in both directions from the monomorphic state (Figs. 3C, D). In the absence of sexual selection for increased flower production, fecundity selection drove the evolution of sexual dimorphism (Figs. 3E, F). Because the relative strength of genetic drift was greater in the absence of sexual selection, the pattern of sexual dimorphism that evolved was not exclusively M > F or F > M. Instead, the kinds of alleles that could spread were strongly biased in the direction predicted under fecundity selection, but the parameter space was broader and sometimes sexual dimorphism opposite the predicted direction could spread (Figs. 3E and 3F compared with Figs. 2B and 2E, respectively).

Effects of pollinator density

Manipulating the overall number of successful pollinator visits to the population had little effect on the outcome of any simulation. The stable level of sexual monomorphism that evolved, the pattern of sexual dimorphism that emerged, and the parameter space under which sexual dimorphism could spread to fixation were always consistent. However, the

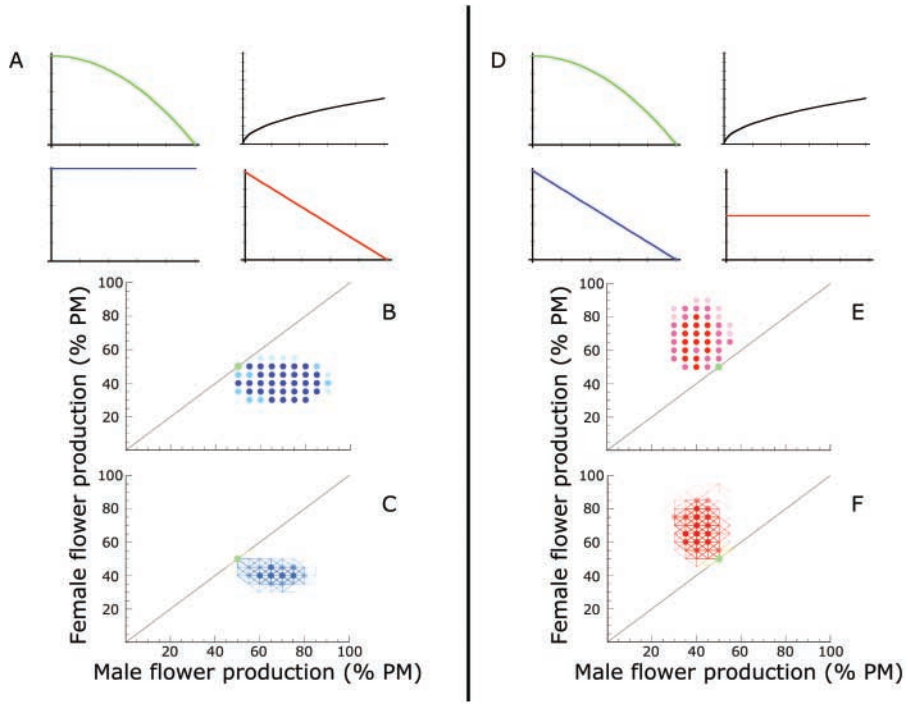
A
B
C
D
E
F
Female flower production (% PM)
Male flower production (% PM)
20
40
60
80
100

Fig. 2

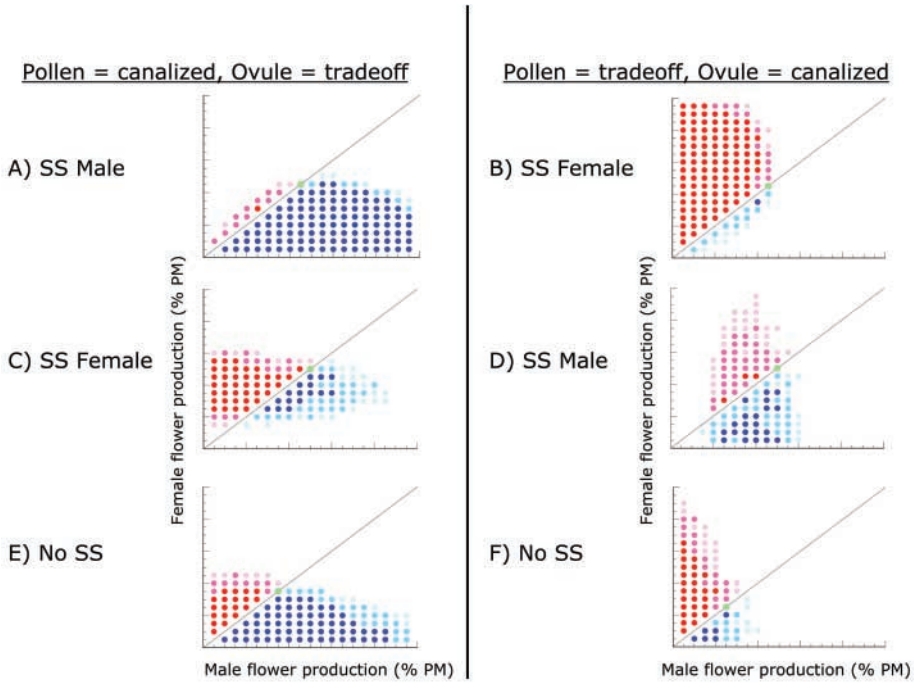
Pollen = canalized, Ovule = tradeoff
Pollen = tradeoff, Ovule = canalized
A) SS Male
B) SS Female
C) SS Female
D) SS Male
E) No SS
F) No SS
Female flower production (% PM)
Male flower production (% PM)

Fig. 3

frequency with which particular combinations of allelic effects spread to fixation increased with higher levels of pollinator visitation. For example, when both sexual selection and fecundity selection favoured the evolution of M > F or F > M (Figs. 2A, D), increasing the number of pollinator visits per time step increased the frequency with which alleles spread to fixation, but did not increase the kinds of alleles that could spread to fixation (Fig. 4, comparison among A, C, E and B, D, F).

Effects of viability selection trade-off

Manipulating the relationship between flower production and the probability of survivorship to the next season did not affect whether sexual dimorphism spread to fixation against the monomorphic strategy or the direction of sexual dimorphism that emerged. However, the sexually monomorphic level of flower production changed in a consistent fashion, as did the parameter space under which sexual dimorphism could spread. For example, when both fecundity and sexual selection favoured the evolution of M > F or F > M (Figs. 2A, D), increasing the trade-off between the probability of survival to the next time step and flower production caused the ESM to decrease (green circle) and restricted the parameter space under which the predicted direction of sexual dimorphism could evolve (Fig. 5, comparison among A, C, E and B, D, F).

Fig. 2. Example results under both kinds of simulations for two separate sets of assumption combinations (A and D), where both sexes experience sexual selection for increased flower production. The plots indicate whether alleles causing a certain level of male (x-axis) and female (y-axis) flower production could spread to fixation against the stable monomorphic strategy (green circle). Green denotes the 'optimal' sexually monomorphic strategy (ESM), blue denotes dimorphism such that males produce more flowers than females (M > F; below the diagonal), red denotes dimorphism such that females produce more flowers than males (F > M; above the diagonal), and the intensity of coloration denotes the frequency with which a particular dimorphic strategy spread to fixation in the 20 trials (0%, 50–74%, 75–94%, 95–100%; light → dark, respectively). Panels (C) and (F) show the results of our second kind of simulation (see text). The frequency with which particular endpoints were reached from the monomorphic strategy is indicated by the increased colour intensity of the circle. Each time the population moved to a different level of flower production, we denoted the path from which it came with a line and denoted the endpoint with a filled circle. For each of the 100 iterations, we overlaid lines and points such that the most common trajectories are darker than the more rare ones.

Fig. 3. The effect of varying sex-specific sexual selection. Plots are oriented as in Figs. 2B and E, and all other assumptions are equal to those presented in Figs. 2A and D. When sexual selection and fecundity selection are in the same direction (A and B), sexual dimorphism evolves in a predictable manner (i.e. in the direction of sexual selection and fecundity selection). When sexual selection and fecundity selection oppose one another (C and D), either direction of sexual dimorphism can evolve, although the likelihood that sexual dimorphism spreads to fixation is greater in the direction predicted by fecundity selection. In the absence of sexual selection for increased flower production (E and F), sexual dimorphism can still evolve; although there is greater drift in these simulations (see text), sexual dimorphism still evolves in the predicted direction most of the time.

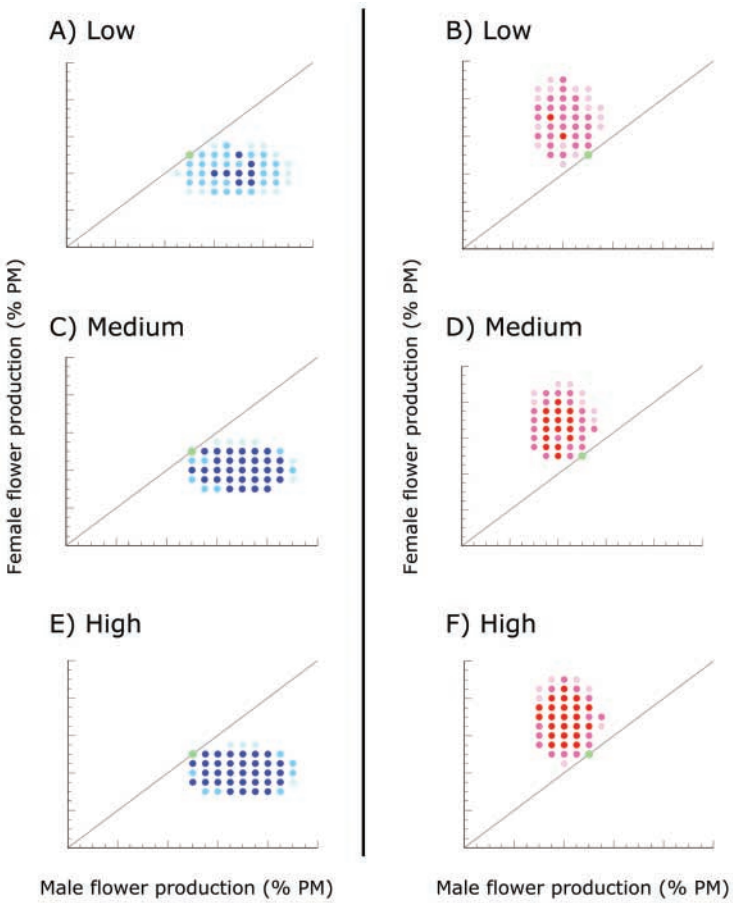
A) Low
B) Low
C) Medium
D) Medium
E) High
F) High
Female flower production (% PM)
Male flower production (% PM)

Fig. 4

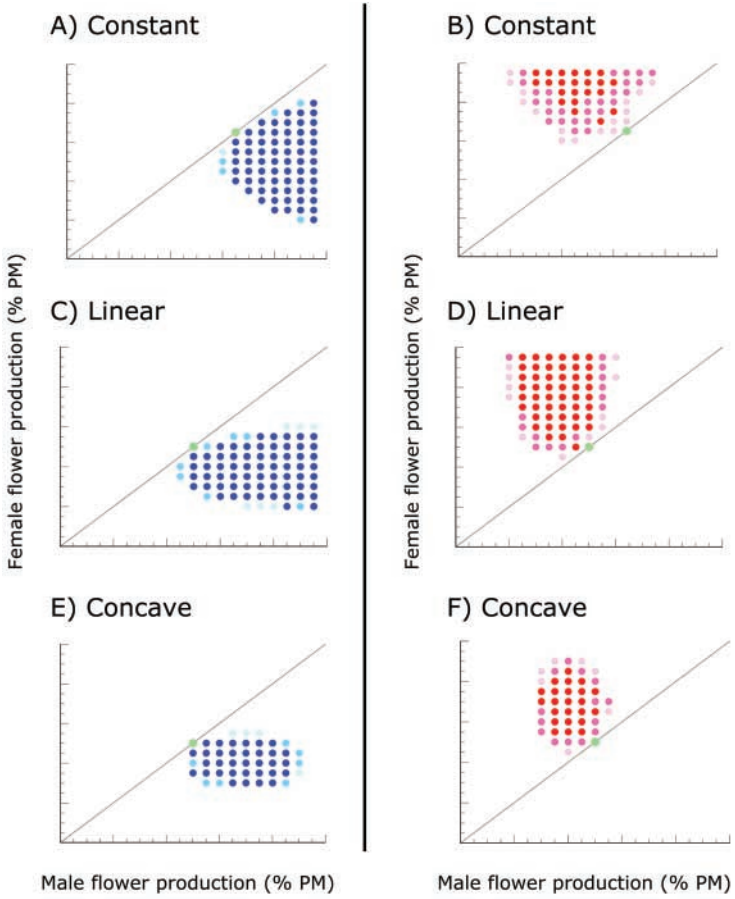
A) Constant
B) Constant
C) Linear
D) Linear
E) Concave
F) Concave
Female flower production (% PM)
Male flower production (% PM)

Fig. 5

DISCUSSION

We find that the interplay between fecundity and sexual selection critically determines whether alleles causing certain patterns of sexually dimorphic flower production can spread to fixation. Trade-offs between flower production and survivorship (viability selection) minimally influence the outcome; populations evolve in the same direction regardless of the assumptions made but the parameter space under which sexual dimorphism spreads is reduced as the trade-off intensifies. Manipulating pollinator density, which alters the intensity of sexual selection, also has minimal effects on the outcome; the direction and shape of the parameter space under which sexual dimorphism spreads stays the same but the frequency with which sexual dimorphism evolves increases as the relationship between flower number and pollinator visitation intensifies.

Sexual dimorphism did not evolve under all of our simulation conditions. Gametophyte-packaging assumptions followed one of three rules: canalized in both sexes, traded off with flower number in both sexes, or was canalized in one sex and traded off with flower number in the other sex. Importantly, when sexual selection occurs in both sexes, only the latter rule resulted in the evolution of sexual dimorphism in flower number, suggesting that when a sex is constrained in terms of how many gametophytes it can pack into a flower, sexual dimorphism evolves in a direction to alleviate this constraint by producing more flowers (Fig. 2). Floral traits, including flower number, are often under sexual selection via both sex functions in hermaphrodites (e.g. Devlin and Elstrand, 1990; Mitchell, 1994; Conner *et al.*, 1996a,b; Gómez, 2000; Maad, 2000; Delph and Ashman, 2006), and it is likely that flower number also influences attractiveness to pollinators in many dioecious species (e.g. Shykoff and Bucheli, 1995). If sexual selection for increased flower number is roughly equivalent between the sexes, our model predicts that sex-differential gametophyte-packaging strategies will determine the pattern of sexual dimorphism that ultimately evolves.

Our results also show that the interplay between fecundity and sexual selection impacts both the extent and direction of sexual dimorphism that evolves. When we restrict sexual selection to one sex, the direction in which sexual dimorphism evolves generally stays the same as when it occurs in both sexes as long as the sexual selection is on the sex with constrained gametophyte production per flower. However, because both fecundity and

Fig. 4. The effect of varying pollinator abundance. Plots are oriented as in Fig. 3, and all other assumptions used to generate panels (A, C, D) and (B, D, F) are as in Figs. 2A and D, respectively (see text). Increasing pollinator abundance does not affect the range of sexually dimorphic alleles that can spread against the monomorphic strategy, but does affect the frequency with which particular combinations spread to fixation because the strength of sexual selection is positively associated with the abundance of pollinators.

Fig. 5. The effect of varying viability selection. Plots are oriented as in Fig. 4, and all other assumptions used to generate panels (A, C, E) and (B, D, F) are as in Figs. 4E and F, respectively (see text). Varying the relationship between flower production and probability of survivorship affects the range of sexually dimorphic alleles that can spread against the monomorphic strategy, but not the frequency with which they do so. As the trade-off becomes more intense (e.g. A to C to E), monomorphic flower production decreases, as does the range of sexual dimorphism that can spread against it. This illustrates how viability selection on flower number can oppose fecundity selection and restrict the evolution of sexually dimorphic flower production.

sexual selection are driving one of the sexes in one direction and fecundity selection is driving the other sex in the opposite direction (whole plant gametophyte production does not increase linearly with increased flower production), the extent of sexual dimorphism that evolves can be greater. This process can be seen by comparing Fig. 2B with Fig. 3A: the parameter space over which M > F can invade is greater in Fig. 3A and nearly all alleles causing M > F can spread as long as females produce fewer flowers than the stable monomorphic strategy. This is even true for alleles that decrease flower production in males. In addition, alleles leading to small amounts of F > M can sometimes invade. This result could be due to fecundity selection on females (i.e. the advantage to females of producing larger and therefore fewer flowers is not counterbalanced by sexual selection for more flowers), and also increased genetic drift. In Fig. 3A, visitation to females is random with respect to flower number. As such, the net effect of sexual selection in Fig. 3A is weaker than in Fig. 2B. Therefore, small amounts of F > M relative to the monomorphic state might sometimes evolve because genetic drift outweighs net selection favouring M > F.

The direction in which sexual dimorphism evolves when sexual selection occurs in both sexes is wholly in one direction and is heavily biased in the same direction when sexual selection occurs only in the sex under the gametophyte-packaging constraint for reasons just discussed. What happens when sexual selection favours increased flower production in only one sex and the gametophyte-packaging constraint selects for increased flower production in the other sex? Under this scenario, sexual dimorphism can evolve in both directions (Figs. 3C, D). This occurs because one sex is under selection to produce more flowers in order to be more attractive to pollinators and the other sex is under selection to produce more flowers in order to produce more gametophytes. These results indicate that sexual selection is not required for the evolution of sexual dimorphism in flower number, and the likelihood that sexual dimorphism spreads to fixation is greater in the direction predicted by fecundity selection than by sexual selection. In Fig. 3D, for example, sexual dimorphism in the direction of M > F evolves readily. Even though males are not experiencing sexual selection to produce more flowers, they are experiencing fecundity selection for increased flower production because the only way to maximize whole plant pollen production is to produce more flowers. Moreover, fecundity selection alone, in the absence of sexual selection, can select for sexual dimorphism in flower number (Figs. 3E, F), and the extent of the sexual dimorphism that can spread reaches the heights seen when sexual selection is a factor (e.g. 5% PM for female flower production and 95% PM for male flower production; Fig. 3E). Hence, caution should be taken when assigning sexual selection as one factor for males making more flowers than females in any given species. If pollen production per flower increases with increasing flower size, then this conclusion would be supported by our model's results. However, if pollen production per flower is canalized, then sexual selection need not be responsible for the M > F flower production.

Experimental work has shown that the evolution of sexually dimorphic traits depends on the underlying genetic architecture (Delph *et al.*, 1996, 2004b; Ashman, 1999), and other theoretical work has suggested similar selective mechanisms by which sexually dimorphic flower production can evolve. Using an ESS framework, Sakai (1993) showed that evolutionarily stable floral display sizes would be affected by the optimal number of pollen or ovules produced per flower and the efficiency of pollination. Our simulation model recovers these results and illustrates how gametophyte-packaging constraints can drive the evolution of sexually dimorphic displays and sometimes counteract sexual selection. Vamosi and Otto (2004) showed that the degree to which sexual dimorphism evolves is dependent on pollinator

abundance, the relationship between floral display size and pollinator attraction, and trade-offs between display size and the dispersal of viable pollen. In addition, they showed that M > F should always evolve unless pollinators are rare and females are more efficient at converting resources into attractive floral displays than males (Vamosi and Otto, 2004). Our results show that increasing pollinator abundance affects the frequency with which particular patterns of sexual dimorphism evolve but not the extent of the dimorphism, and illustrate how differential gametophyte-packaging constraints can facilitate the evolution of display dimorphism. An important difference between our model and that of Vamosi and Otto (2004) is that our framework explicitly ties pollen and ovule production to flower production and the relative fitness of a particular strategy. In doing so, our results show that fecundity selection effectively bounds the parameter space under which sex-differential flower production can evolve.

Testing the theory

Our results illustrate the importance of fecundity selection in determining the trajectory with which sexually dimorphic flower production evolves, and also show how predictions made solely on the basis of sexual selection can sometimes be misguided. However, there are few data in the literature that can be used to test these predictions. Specifically, data on how variation in pollen and ovule production per flower varies with flower size are lacking for most dioecious plant species for which we have data on sex-specific flower production (Table 1), as are data on whether the strength of sexual selection for increased flower number differs between the sexes. We are aware of three species for which there are data that can be used to assess our model and discuss them below.

Silene latifolia (Caryophyllaceae) is a dioecious perennial species with a documented trade-off between flower size and number (Meagher, 1999; Delph *et al.*, 2004a), and pollinators of this species prefer plants with large floral displays (Shykoff and Bucheli, 1995). In terms of gametophyte-packaging constraints, ovule number increases with flower size, but pollen production per flower is canalized (Delph *et al.*, 2004a). This case matches the model assumptions used in either Fig. 2B or Fig. 3A, depending on whether there is equivalent sexual selection on males and females for increased flower number, and predicts that M > F sexual dimorphism should evolve. Indeed, males of *S. latifolia* produce as many as 16 times more flowers than females (Meagher, 1992; Laporte and Delph, 1996; Delph *et al.*, 2002, 2004a). Our model correctly predicted the direction of the dimorphism, and it also predicts that sexual selection should be much stronger in males than in females because it is under this case (Fig. 3A) that extreme sexual dimorphism evolves.

Antennaria parviflora and *Dendrocacalia crepidifolia* are both members of the Asteraceae, in which ovule production per flower is canalized at one. These two species have different patterns of sexual dimorphism: in *A. parviflora*, females produce more flowers than males (P. Bierzychudek, unpublished data), whereas in *D. crepidifolia* females and males produce the same number of flowers (Kato and Nagamasu, 1995). Our model shows how the canalization of ovule production per flower favours the evolution of F > M irrespective of sexual selection for increased flower production in either sex. Bierzychudek (1987) reported greater variation in visit duration to flowers on females than flowers on males, which may indicate a greater opportunity for sexual selection in females and would be in strong agreement with our model's predictions (Fig. 3B). Although we know of no other data on *D. crepidifolia*, our model predicts that pollen production per flower must also be canalized,

and predicts that the strength of sexual selection on flower production is equivalent between the sexes.

In conclusion, our model fuses together aspects of these previous studies and explicitly incorporates assumptions concerning allocation trade-offs, pollinator abundance, and the genetic integration of traits. The combined results of this study clearly illustrate how the way in which plants package their gametophytes may differentially tip the balance of sexual and fertility selection between the sexes, leading to the evolution of sexually dimorphic flower production from a sexually monomorphic state. Our model makes clear predictions concerning the evolution of sexually dimorphic flower production, and illustrates how the interplay between sexual, fecundity, and viability selection affect the pattern and degree of dimorphism that evolves. However, there are few empirical data with which to confirm or reject these predictions, especially with respect to how pollen and ovule number are associated with variation in flower size. We hope that subsequent research will provide the data necessary to critically evaluate our model.

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